PATHOLOGY

of

BERI-BERI

"Scribendi recte sapere est principium et fons."

Horace.
MACROSCOPIC or POST-MORTEM APPEARANCES.

The condition of the body will vary according to the form and period of duration of the disease. In some cases the oedema is marked often accompanied by cyanosis, in others the body is pallid and emaciated. In acute cases there may be frothing at the mouth. Rigor Mortis comes on early and in cases where death has been sudden only lasts a short time.

On making the incision to open the abdomen one is struck:—

1). By the pale almost yellowish colour of the muscles
2). By the fluidity of the blood in the veins and its dark colour (saturated with Carbon dioxide).

On account of this condition of the blood hypostatic congestion occurs early.

3). In oedematous cases the presence of serous fluid in the cellular tissue.

Punctiform haemorrhages may be noticed over the visceral surface of the peritoneum, pericardium and pleurae, and effusion into these cavities is very common, especially into the pericardium. A certain degree of pericardial effusion is found in 95% of cases, but as a rule not enough to have caused death from mechanical means, but it may be regarded as one of the constant pathological signs of this disease.

ABDOMINAL VISCERA.

The Liver is often somewhat enlarged and congested, fatty degeneration may be present and it may have the typical nut-meg appearance.
Pathology——

The Gall Bladder contains fluid bile.

The Spleen is congested and frequently enlarged, this is probably of Malarial origin, for in Christmas Island a non-malarial locality there was no marked increase in the size of this organ.

The Stomach is as a rule dilated, its walls are oedematous and congested and at the tops of the folds haemorrhagic erosions may be seen. In acute cases a gastro-duodenitis is generally present, this point was particularly emphasized by Hamilton Wright, as being a constant pathological change in Beri-beri and the portal of entrance of the specific virus of the disease. But while great hyperaemia is present in the acute fulminating cases, it is not a conspicuous symptom of those in whom the disease has been of some standing, and the condition when found is probably due to the general venous congestion.

In the Intestines there is little, the follicles may be swelled and small haemorrhages may be found. Intestinal parasites are frequently seen, but have no etiological significance.

The Kidneys are as a rule quite healthy, and the capsule strips readily, puriform haemorrhages may be seen here as elsewhere and cloudy swelling may be present.

THORACIC VISCERA.

Lungs are usually pale, and frequently there is oedema and congestion at the bases. As a rule the lungs do not contain much air, but occasionally are markedly emphysematous. Here and there a patch of deep colour will point to an area of pulmonary apoplexy.
HEART. The pericardium constantly contains fluid varying in amount. The organ itself is always enlarged especially on the right side. The state of the myocardium will depend on whether the heart is hypertrophied or dilated. In the former cases the heart walls are reddish brown and firm, while in the latter they are pale and friable. Clots are often found on the right side passing into the pulmonary artery. In atrophic cases in which the death struggle has been protracted "chicken-fat" clots are often seen. The valves are in advanced cases are not "tight", especially so is this the case with the Tricuspid, but there is no disease of the endocardium. The coronary vessels are much dilated. The changes in the heart are degenerative and not inflammatory.

BLOOD VESSELS. Arterial lesions are inconstant, occasionally there is a disintegration of the elastic network of the aorta.

NERVOUS SYSTEM. To the naked eye there is little change to be made out in the nerves.

VOLUNTARY MUSCLES. Marked atrophy even in those that appear large from the swelling, this being due to the infiltration of serum in the inter-muscular cellular tissue. The muscles when atrophied are yellowish in colour, when oedematosous they present what has been termed a "marbled" appearance, yellow lines as a network throughout the redder flesh.
MICROSCOPIC or HISTOLOGICAL APPEARANCES.

HEART

The cardiac muscle fibres may be affected to all degrees, the right ventricle shows the changes best, and in the same section one may see all degrees of degeneration; alongside healthy fibres one finds some showing fatty degeneration; others in which transverse striation is lost and others again with marked granular degeneration and fragmentation. In very advanced cases all the normal elements may be lost, the fibres are frequently seen to be vacuolated and sometimes the interstitial tissue is increased.

Dirck reported, that in cases examined very shortly after death, acute changes in the myocardium were evidenced by the swelling of the muscle cells, they seem to be infiltrated by a homogenous vitreous mass in the form of bands, which blended insensibly with the striated protoplasm of the cardiac fibres. (see micro-photograph).

LUNGS

Unless congestion and oedema are present, these do not show any changes; there is nothing characteristic about the Spleen.

LIVER

Shows fatty degeneration and cloudy swelling, and there is congestion of the intra-lobular capillaries. Platt has described what he terms "the interstitial hepatitis of Beri-beri", which consists in an infiltration of the interlobular fibrous connective tissue with small round cells.
These show cloudy swelling, but little other change. Miura found glomerular nephritis, but this is so uncommon, that it cannot be considered the cause of the anasarca of the disease.

**MUSCLES**

In the case of dry atrophic muscles, the fibres will will be seen to be thin and to have lost their striation; this is one of the earliest changes. On cross section the characteristic polyhedral form will be seen to have become round or oval. Healthy and diseased fibres will be found to be lying side by side. Later there is a want of definition of the individual fibres and eventually they may resolve into a sort of granular mass, the sarcoplasm shrunken away from the sarcolemma.

In the case of dropsical muscles, the presence of the oedema enables one to dissect out the fibres with ease. These fibres are specially liable to develop colloid or serous degeneration, the sarcoplasm is swollen within the sarcolemma and under the microscope appears white, reflecting direct light more strongly than the normal fibre. The nuclei of the cells are increased, and there is decided proliferation of the interstitial tissue.

**NERVES.**

Here lies the principle changes in this disease, and to the nerve lesions can be traced all the symptoms, the paralysis, the cardiac symptoms, the fall of blood pressure.
NERVES (continued).

pressure, the diminished urine and all the other phenomena that go to make up the disease that is known as Beri-beri.

The histo-pathological appearances necessarily vary as greatly as the lesions produced. In one section every form of degeneration may be seen and the changes that occur are those of a typical parenchymatous neuritis; true Wallerian degeneration. The myelin sheath suffers first, the changes may be quite insignificant or very profound. On longitudinal section the sheath can be seen to have broken up into droplets or beads, finally it disappears. When the degeneration of the myelin sheath is advanced, the axis cylinder suffers, it first gets distorted, then irregular and eventually can only be seen in the neighbourhood of the Nodes of Ranvier; later it disappears entirely.

The nuclei of Schwann's Sheath undergo karyokinetic proliferation.

Thus anything may be found from a simple swelling of the medullary substance with thickening and displacement of the axis cylinder to complete destruction leaving only an empty sheath of Schwann, studded with newformed nuclei.

On transverse section in advanced cases some of the cells are seen to be still normal, others still retain their axis cylinders, others again contain homogenous masses composed of myelin detritus, whilst finally others are represented by mere empty spaces.

(see microphotographs)
The connective tissue also increases with multiple thickening of the nuclei and their is some thickening of the blood vessels.

In chronic cases there is sclerosis of the endoneurium and wrinkled bands stretch across the section. On account of these changes Scheube and Baclz conclude, that the process is not a simple degeneration, but that it is inflammatory: Yamagiva who did extensive work on this subject for Miura, upholds the original view of Pekelharing and Winkler, that the changes are those of simple degeneration.

If on cross section the nerve shows the presence of a number of fine fibres, it is said to be indubitable evidence of degeneration. The nearer one approaches the periphery the more advanced are the changes especially in the terminal twigs, as one gets up to the Spinal cord the changes are not so marked, so that it is difficult to be certain of any alteration in nerve Roots: the anterior Root is said by some never to be affected and the posterior only slightly.

It seems to me that clinically there are grounds for believing in the involvement of Posterior Roots in advanced cases, for acute girdle-like pains may be complained of and on examination the Cerebro-Spinal fluid shows decided increase in the Lymphocytes, which may be taken as a proof that a Radiculitis has occurred.

In the case of such important nerves, as the Vagi and Recurrent Laryngeals, advanced degeneration is
not seen. This is readily understood when one remembers how essential they are to the general economy. Hence in the transverse section of these nerves one seldom sees more than a few fibres filled with granular debris.

In order of sequence the Peroneal, Tibial and Saphena nerves are first affected.

Pathologists differ as to whether the Spinal Cord is affected at all. Pekelharing and Winkler held that it was not, Wright on the other hand found "hazy cells with swollen nuclei and slight chromatolysis in the ganglion cells of the Cord and Bulb."

Küsternann found acute degeneration of the ganglia at the base of the 4th Ventricle.

Sitta found degeneration of the Cauda Equina in 5 subjects, who had died of Beri-beri.

Lesions of the cord are probably rare: the following have been described:—

(1). Proliferation of the cells of the Ependyma and infiltration of nuclei the circumference.

(2). Atrophy or loss of Motor cells of the Anterior Cornua.

(3). Ascending degeneration of the Posterior Columns, in connection with atrophy of corresponding Sensory Roots.

(4). Vacuolation of Motor Cells of the Anterior Cornua.

(5). Accumulation of waxy bodies in the white sub-

stance of the cord.
It seems therefore to me from clinical reasons as well as from pathological findings, that the primary degeneration is in the nerves, but that it must not be limited too narrowly to the peripheral part alone. While the periphery is by far the most frequent part to be diseased, yet the "infection" may be more extensive, and then the posterior nerve roots and even the cord itself may be implicated.

The nervous theory is admitted now by everyone, and so need not do more than mention the view of Gliogner who considered, that the disease was not a polyneuritis but a polymyositis. (Muskelbruchkrankheit).

Such then in brief are the characteristics of the macroscopic and microscopic pathological changes met with in Beri-beri.
Sub-acute Beri-beri.

Section of Sciatic Nerve from advanced case (x250)

Consists almost entirely of vacuoles left by the degenerated nerves, here and there is a homogenous mass and a few black dots indicate an axis cylinder that has not altogether broken up. Thickening of the vessel walls with infiltration of nuclei.

Section of Optic Nerve. (x250)

Whole section consists of fibres in an advanced state of degeneration, the dark spots indicate those in which the condition is earlier. Vessels and interfascicular space thickened and infiltrated.
ACUTE BERI-BERI.

Longitudinal Section of Popliteal Nerve (×250).
Degeneration of Myelin Sheath.

(micro-photograph)

ACUTE BERI-BERI.

Longitudinal Section of Popliteal Nerve, (x250).
Shows fragellation of Myelin Sheath with characteristic bead formation.

(micro-photograph).
SUB-ACUTE "DRY" BERI-BERI
Longitudinal Section of Popliteal Nerve. (×550).
Marked degeneration of Myelin sheath,
(micro-photograph)

SUB-ACUTE BERI-BERI.
Transverse Section of Peroneal Nerve with Nerve Sheaths.
Considerable loss of nerve fibres, (A.A)=intact fibres
(B.B)= blood vessels, (C)=Nerve sheaths.
(Photograph from drawing after Pekelharing)
SUB-ACUTE BERI-BERI.

Transverse Section of Branch of Anterior Crural Nerve.
Most of the nerve fibres have been destroyed, leaving empty spaces surrounded by solid interstitial tissue. A few of the fibres are replaced by a swollen granular mass (A.A) and a few (coloured red) are still intact. (Photograph from drawing after Pekelharing)

SUB-ACUTE "WET" BERI-BERI.

Section of Myocardium. (x 500.)
Shows clearly the marked degeneration of the cardiac muscle fibres with fragmentation and segmentation. (micro-photograph)
DIAGNOSIS.

and

DIFFERENTIAL DIAGNOSIS.

"Non enim tam auctoritatis in disputando, quam rationis momenta querenda sunt."

Cicero.
The Diagnosis of Beri-beri does not rest upon any one symptom, but on a number of clinical signs. It is not as a rule difficult, but the rudimentary forms are apt to give the most trouble. It is a matter of importance, that the diagnosis be made early.

In the fully developed cases, whether atrophic or oedematous, the picture is too typical to be mistaken. From one's own observation in the Federated Malay States and elsewhere there is far too great a tendency to label almost everything as Beri-beri, cases which are suffering from Climatic Oedema, Malarial Neuritis, Anaemia, Ankylostomiasis and General Debility. This is especially the case in native managed hospitals.

I think then that the following can be regarded as the essential early signs of the disease, upon which diagnosis can be based.

1). Puffiness of the face.
2). Slight pretibial oedema.
3). Slight paraesthesia over the outer side of the leg.
4). Slight hyperaesthesia of the calves on firm pressure.
5). Easily accelerated cardiac action.
6). Slight epigastric fulness.

These signs are nearly always all present and taken together may be said to be pathognomonic of the disease.
DIFFERENTIAL DIAGNOSIS.

I think, that the following is a fairly comprehensive list of the possible diseases, which might give rise to any confusion in the diagnosis of Beri-beri. I think, that those considered first are by far the most important and apt to be the most misleading, while those towards the end should seldom give rise to any difficulty.

A. MALARIAL NEURITIS.

In Burma, as Fink has shown, and in many other places where Malaria is rife, a Peripheral Neuritis of undoubted Malarial origin occurs. In Burma this is known locally as "Htone Na" and shows itself in two forms "So" or wet and "Chauk" or dry. At first sight these suggest at once to the observer the two chief types of Beri-beri. In all the cases there is a strong antecedent Malarial history, and the spleens are enlarged.

Further there may be no fever at the time of the onset of paralytic symptoms and owing to the weakness of the extensor muscles, the foot-drop, wrist-drop, and "squatting" tests are all positive. There is anaesthesia varying in amount, loss of Knee jerks, hyperaesthesia of the calves of the legs and muscular wasting are all present.

On the other hand if there is any oedema it is always very slight, there are no gastric symptoms and no CARDIAC SYMPTOMS, nor is there any dyspnoea.
Diagnosis

In other words, the vagus nerve is not affected, this is of the greatest differential diagnostic importance, because of the similarity otherwise in the symptoms.

In Fink's cases the patients were, and had been for some time previously, eating freshly husked rice. As a further aid in the diagnosis, in some cases blood examination will prove of value, this is not of course of paramount importance, for in the malarial cases it may be negative, whilst in cases of Beri-beri, if the patient has Malaria as well, it may be positive.

Then lastly there is the all important therapeutic test, for the malarial cases as a rule clear up rapidly with the exhibition of quinine, the Beri-beri cases do not.

So important is the differential diagnosis between these two diseases and so often are they mistaken, that I have no hesitation in saying, that many of the cases diagnosed as Beri-beri may be attributed to previous attacks of Malaria.

The whole point of the matter lies in this, that in one form of neuritis the Vagus is affected in the other it is not. Therefore it is of importance to remember the course of this nerve, composed as it is of both Motor and Sensory fibres, and supplying the larynx, pharynx, lungs, oesophagus, stomach and heart.

One note of warning I should strike and that is the possibility, that in the gastric form of remittent malaria the end fibres of the vagus to the gastric mucosa may be irritated and so cause obstinate vomiting, but even...
the many other signs of Vagus affection so typical of Beri-beri will be wanting.

**E. EPIDEMIC DROPSY.**

There is marked oedema and frequently diarrhoea and vomiting. Nearly always there is a considerable initial pyrexia.

There is never any paralysis, the knee-jerks are present and there is no anaesthesia or paraesthesia of the skin and no muscular hyperaesthesia.

**C. ANKYLOSTOMIASIS.**

In this disease the onset is insidious, though it is sometimes ushered in with a swinging temperature. The anaemia is progressive and it is on account of the blood condition, that any similarity between this and Beri-beri exists. The appetite is perverted, there is no paralysis, anaesthesia or hyperaesthesia. As a rule the ova will be found in the stools, but I should mention, that in the latter stages of the disease parasites may not be found and yet symptoms remain, but the blood in these cases always shows a marked eosinophilia.

**D. LANDRY'S PARALYSIS.**

This disease can be very like an acute attack of Beri-beri in many of its symptoms. But there is no oedema, no wasting of the muscles and sensation is rarely lost. Complete paralysis occurs in a few hours and there is as well Fever, Splenic enlargement, Albuminuria and Skin eruptions.
E. ALCOHOLIC NEURITIS.

In this case there will be the previous alcoholic history, the tremulousness, the digestive disorders, and the albumen in the urine.

F. ARSENICAL NEURITIS.

The question of the analogy between Arsenic and Beri-beri has already been discussed when considering the etiology of the disease. Again there is the history to guide one, the marked diarrhoea, the abdominal pain, blood in the urine, discoloration of the skin and the absence of oedema.

G. LEAD POISONING.

Here one has the classical symptoms to guide one, the pains in the joints, the severe abdominal colic, the wrist-drop without any corresponding foot-drop, the blue line on the gums, and the absence of pain in the muscles.

H. LOCOMOTOR ATAXIA.

The girdle pains of this disease are very characteristic, there is probably a very definite history to aid one, the course of the symptoms, the anaesthesia of the soles of the feet and the presence of the Argyll-Robertson pupil will all make the diagnosis easy.

I. BRIGHT'S DISEASE.

There is really not much similarity between these two diseases, a mistake might arise in a very oedematous case, but in Bright's there is no loss of sensation, no hyperaesthesia, no loss of knee jerks and there is albumen in the urine.
Diagnosis——

J. PELLAGRA.

History of eating maize, (I am not contending that this is the cause of this disease), but apart from the history there are very definite differences: the knee-jerks are increased, marked tremors are present and often tetany, there is distinct inco-ordination, and there is no tenderness of the muscles and the other cardinal symptoms of Beri-beri, are also absent.

K. ERGOTISM.

Again the history will be of help, the consumption of rye; as regards the symptoms besides the many negative points, there is the tingling of the skin and the typical gangrene.

L. LATHYRISM.

In this case there is a history of eating pulse, then again the knee-jerks are increased, and negatively there is no tenderness, no inco-ordination, no paralysis, no anaesthesia.

M. MYELITIS.

The rapid onset is characteristic, bladder troubles are prominent with incontinence of urine, there is no pain or tenderness in the muscles.

N. SPASTIC PARAPLEGIA.

Here the picture is very different, there is the slow onset, the characteristic stiffness, the great increase of the knee-jerks, and the absence of tenderness, of oedema, and of muscular atrophy.
O. PROGRESSIVE MUSCULAR ATROPHY. (ARAN-DUCHENNE)

There is really not very much similarity between these two diseases. The Progressive Muscular Atrophy takes years to form, there is an absence of hyperaesthesia in the muscles and no anaesthesia.

P. SYRINGOMYELIA.

The onset is very slow, the knee-jerks are increased, there may be scoliosis and there is the characteristic thermo-anaesthesia.

Q. STRACHAN'S DISEASE.

In the acute form of Beri-beri the symptoms are somewhat similar to those of Strachan's disease, but in the latter there is no oedema, no paraesthesia, no acute cardiac crisis, and the paresis passes on to atrophy.

R. LEPROSY.

The disease is similar in that there are areas of anaesthesia, but there is little other similarity, the slow progress is different, the skin and trophic changes, the perforations, the bone reabsorption. (In very rare cases bone reabsorption has been seen in Beri-beri?).

S. CARDIAC DISEASE.

There may be oedema, but it is of the feet, there will be murmurs over the cardiac areas, albumen in the urine and no nervous symptoms, no paralysis, no anaesthesia.
T. PERNICIOUS ANAEMIA.

As already stated, in advanced cases of Beri-beri the blood findings may be very like those of Pernicious Anaemia, but in the latter disease there are none of the other symptoms of Beri-beri, no paralysis, no anaesthesia, no true muscular hyperaesthesia.

The only other possibilities, that one can think of need only be mentioned, namely Trichina and possibly Spinal Meningitis.

MALINGERING.

Apart from recognized diseases, I think that the possibility of malingering should be held in mind. In areas where the disease prevails, it is no very uncommon thing to find coolies attempting to get off work on the plea of Beri-beri, they know many of the cardinal symptoms and induce cardiac irregularities by the use of drugs. Such cases can be most suggestive of Beri-beri, especially if examined hurriedly, but a careful inquiry into the nervous system, with examination of the knee jerks and the alterations of sensation will overcome any difficulty so long as the possibility is held in mind.

Briefly then, these are the possible diseases, which might be mistaken for Beri-beri in one or other of its phases. I have not gone into this matter in any detail, but I think the points mentioned are sufficient to show the main distinctions, and before passing on to deal with the treatment one would like to briefly discuss the question of Ship Beri-beri.
NOTES ON SHIP BERI-BERI.

I have not included either this or Asylum Beri-beri amongst the differential list owing to the controversy, that still exists on the subject. The opinion of medical men differ very much, as to whether Ship Beri-beri is to be regarded as true Beri-beri or not.

Le Dantec in a paper published in 1905 referred to an outbreak as Nautical Beri-beri, drawing a distinction between it and the ordinary form. Bullimore from cases seen at Falmouth also did not consider the disease to be true Beri-beri, from his observations he further held, that it was neither infectious nor due to rice, but to general digestive disturbances caused by long sea voyages on a poor dietary.

In cases seen by Nocht at Hamburg the morbidity was as high as 62.5% and the mortality 15%.

A commission was appointed in Norway to go thoroughly into this matter, and as a result of their investigations they came to the conclusion, that two forms existed.

1). Of vegetable origin caused by the eating of faulty rice.

2). Of animal origin caused by the eating of bad stale meat and fish.

As a result certain prophylactic measures were advocated including Government inspection of food and the limitation of certain necessary articles, thus compelling more frequent re-provisioning.
Manson and Holst considered Ship Beri-beri to be a form of Scurvy. It occurs practically only on sailing vessels engaged in long voyages during which fresh food is unavailable. Symptoms typical of Scurvy and common to Ship Beri-beri are haemorrhages, loosened teeth, pain when the muscles are grasped, loss of reflexes and even anaesthesia. Holst considers that it is due to eating food deprived of its antiscorbutic properties by boiling and drying.

It is fairly generally recognized that the taking of Lime Juice prevents true Scurvy, but it does not always prevent Ship Beri-beri, as was shown by the Norwegian Beri-beri committee.

Professor Torup's theory is that scurvy is a chronic poisoning due to the decomposition of food. Dried foods allowed to get wet are apt to generate Scurvy, dogs are very susceptible to it as was seen in Scott's South Pole expedition, for the dogs, that were fed on dried fish allowed to get wet always developed the disease; so also Manson himself developed it from this cause. In the same way on board ship food from damaged or rusty tins is far more liable to cause it. Norwegian ships are especially prone to it as they purchase timned food condemned by the London inspectors and have no regulations regarding the taking of Lime Juice as in the British service.

Italian ships are on the other hand very free from it and this is undoubtedly explained by the fact, that their dietary is very largely supplemented by onions and wine.
In the Journal of the American Medical Association (May 30, 1908) a case was reported of "Beri-beri" occurring without a definite rice factor in a Dutch ship, which came into San Francisco with the crew suffering from this disease. The point stress was laid on was that there was no rice on board either as cargo or as food. The conclusion, that was arrived at was, "that other vegetables besides rice may be factors in the production of Beri-beri". This is not to be denied and one has already attempted to show, that it may be true of Sago.

But this is the point, that I would lay most emphasis on, the sole diet of the crew in this vessel consisted of ship's bread, salt meat and dried beans, which were stated to be mouldy. Thus the probability here, as in all these cases of Beri-beri on ships, is very strong, that the disease in question had a very large element of Scurvy in it.

Epidemics of so-called Ship Beri-beri are often due to Tropical Oedema from lowered blood pressure. A case of this came before my notice at Christmas Island. A cargo vessel came to load phosphate, and the captain sent a message to say, that a number of the crew were suffering from Beri-beri. The crew were principally Australians, the food on board was not very first class and there was a great lack of fresh vegetables. The men on examination displayed oedema, which in some cases was very considerable, and most of them had refused to work.
Besides the oedema there was some pain on pressure over the muscles, there was however no anaesthesia and no cardiac affection, the urine was free from albumen. The Blood Pressure was lowered in every case, in some to below 100 mm. of Hg: There may have been a certain scorbutic element present, but they were not suffering from Beri-beri.

Warm weather produces vaso-motor dilatation from diminished tone of the vaso-constrictors and thus tends to the development of oedema in the dependant parts. It has been clearly shown, that the blood vessels of the skin and those of the abdominal viscera frequently act in direct antagonism to one another (Janeway).

Thus the dilatation of the peripheral vessels is not always accompanied by a fall in the blood pressure, but when balanced does not occur, there is a fall in the blood pressure with congestion of the viscera and transudation of fluid. This is so frequently seen in the Oedema of the Tropics.

In most of the notable sieges of history a condition resembling Ship Beri-beri has occurred amongst the besieged. Dechambre has shown how a disease resembling Beri-beri was rife during the siege of Paris, whilst Baclz reported the same in the cases of Port Arthur.

It seems therefore, that so-called Ship's Beri-beri may frequently be but Tropical Oedema, whilst on the other hand the cases which are seen on vessels arriving in temperate climates, whilst they may have a Beri-beri
element present, are largely complicated by Scorbутus.

Consider for a moment the surroundings, the damp almost hermetically sealed, badly ventilated fo'castle full of perspiring Lascars and at once one has a combination of many of the predisposing causes, and add to this a dietary deficient in Phosphorus, (whatever form that dietary may take) and with the further addition of a food deficient in anti-scorbutic properties.

Is there any wonder, that mixed symptoms arise? Some of which point to Beri-beri and others to Scurvy, for in addition to the oedema, hyperaesthesia, and para-aesthesia of Beri-beri, there are many of the typical evidences of Scurvy the spongy gums, the loosened teeth, and the subcutaneous haemorrhages.

On the other hand the reviewing of a large number of reports of so-called Ship Beri-beri brings out this important point, that in a great number of cases the symptoms do not correspond to the typical picture of Beri-beri at all. While oedema is a prominent symptom it is unaccompanied by atrophy of the muscles or any evidence even of paresis. In fact, most of the symptoms in these cases too point to a pseudo-scurvy, the additional feature being caused either by a form of chronic ptomaine poisoning or else deficiency of phosphorus.

In every case a decided inferiority of the food supply can be proved.

One must not be supposed to infer from this, that true Beri-beri never occurs at sea for it undoubtedly does.
But in these cases there is a definite rice factor and the symptoms are those of the disease that has been described throughout this paper (and it is not by any means common to find authentic cases) the disease described as Ship-Beri-beri is not the true disease.

NOTES ON ASYLUM BERI-BERI.

Reports of an outbreak of a disease analogous to Beri-beri occurring in different asylums for the insane, have been published from time to time.

While the disease resembles true Beri-beri in many particulars, yet the picture presented is not that of the disease as seen in the Tropics.

From reading the accounts of such, one is struck briefly by the following points.

The oedema was not the same for it is hard and brawny in consistence, and does not yield on pressure.

Paralytic symptoms only showed themselves in one third of the cases. There was no atrophy of the gastrocnemii. The bladder and rectum were early affected and often seriously so. The skin of the hands and face became brown suggesting Pellagra, and there were patches of Erythema and early trophic troubles, bed-sores, purpura and the like.

Further at the post-mortem examinations, besides the nerve degeneration, there were distinct changes in the cord, the large motor cells in the Anterior Cornua were swelled and were in a state of chromatolysis, the protoplasm was vacuolated and the nuclei eccentric.
Finally motile organisms were isolated from the Liver, Spleen, and Cerebro-spinal Fluid, which resembled Proteus Vulgaris.

How far this organism was the cause of the disease I am not in a position to state, but from the symptoms displayed the disease was not true Beri-beri. There were many things, that pointed to the possibility that the Beri-beri element was present, the over-crowding, the deficient hygiene, the faulty dietary. But here as in the case of Ship Beri-beri something else was undoubtedly superimposed either of a Scorbatic or Infective nature.
PROGNOSIS AND EVOLUTION

of

BERI-BERI.

"Vir sapit qui pausa loquitur".

SENeca.
EVOLUTION AND PROGNOSIS.

The commonest complications of Beri-beri are Tuberculosis, Dysentery and Malaria.
Although most cases recover generally there is a lot of subsequent weakness and anaemia, with an absence of the knee-jerk often for a long period after apparent "cure" has taken place.

The prognosis is as a whole good, but it varies widely in different epidemics, and owing to the fact, that acute cardiac symptoms may supervene at any period, it must always be extremely guarded.

Amongst the unfavorable symptoms, which must be noted are:— Vomiting, marked dilatation of the heart, and irregularity of the pulse.

The mortality returns vary exceedingly.
In Sumatra at one time amongst the Chinese coolies it was as high as 60 or 70 %, and in Hongkong, taking the average over a number of years, it reaches the figure of 49.5 % in Males and 35.4 % in Females.
On the other hand amongst the troops in the Dutch Indies it was only from 2 to 6 %, and according to Scheule in Japan it was 3.5 %.
In the Russo-Japanese War it was 2 % and at Christmas Island the average over a period of 9 years was 9.5 %, (the highest being 33.7 % in 1901 and the lowest 1.2 % in 1909).

The duration of illness also varies, some cases are very prolonged, others developing acute symptoms die suddenly.
The average in the Tung Wah Hospital, Hongkong, is 47 days, on Christmas Island it was 43 and under a form of treatment to be described it fell to 34 days.

Relapses are frequent and points, that call for extra care are:

1. Non-return of the knee-jerk.
2. A persistent paresis.
3. Fatigue on the slightest extra exertion.
4. Localized areas of hyperaesthesia.
5. Slight pre-tibial oedema.
6. Loss or feebleness of the sexual appetite.
7. Acceleration of the pulse on the slightest exertion, a violent apex beat with a feeble radial impulse.

N.B. This is of the greatest importance and so too is the next point.

8. A persistent diminution in the amount of urine excreted, almost oliguria.

In a large percentage of cases the ultimate usefulness of the coolie is not impaired, a certain percentage on account of cardiac trouble are only fit for light work, whilst some, who on account of a protracted attack, have developed deformities talipes etc, are not much use to the employer at all, although even in these good results may be obtained by tenotomy of the Tendo Achilles or with fixation of the ankle joint.
PROPHYLAXIS.

OF

BERI-BERI.

"Principis obsta, sero medicina paratur
Cum mala per longas convaluere moras."

OVID.
After all that has been written, there can be little doubt, that the form of rice in daily use is the one paramount all-important point in connection with the prophylaxis of this disease. It is no exageration to say, that if "chred" rice be employed the disease can be prevented from occuring. All that is required is a rice, that is not highly polished, which therefore retains a great deal of its pericarp, and so yields a generous supply of Phosphorus.

I have endeavoured by the citation of a number of cases from a variety of sources, to prove the incon-trovertability of this statement.

Sooner or later the big rice mills of Rangoon, Sian and elsewhere will realize that, though a pure white rice is still demanded by the European market, yet they must produce cheaply for the contractors' coolie a rice, which though ot may have a dirty appearance when cooked, retains the all-important property so essential for health.

Large sea ports would do well to prohibit the importation of a rice, which falls short of the specified requirements, by this means alone can the disease, which annually forms such a large percentage of hospital admissions, and in Hongkong such a large mortality, be exterminated.

When one considers the crippling of work in big estates and in mines, which Beri-beri causes by its devastation of the Chinese labour, one wonders how long employers will continue to use a rice, which is
so toxic, when so easily they could at least give the other form a trial.

Coupled with the use of "cured" rice one would advocate a liberal and varied diet. I append a few lists to show the different articles of food in Christmas Island, with their respective Calorific Values and Energy producing properties.

None of these articles will cause much additional expense, the coolies will be fed better than ever before in their lives, and one will have the great satisfaction of watching the under-developed man, who arrives, becoming month by month a strong, sturdy and efficient coolie.

This is not the picture of a fancied Utopia, for on Christmas Island I used to take regular chest measurements of the coolies as they arrived, and it was surprising to see the change, that occurred. There was no doubt, that the power to do work was more than doubled, and in a very short time the original extra cost was repaid ten-fold.

I have already mentioned, that in Christmas Island there were certain difficulties in the introduction of the sole use of "cured" rice, personally I do not think it would have taken a great effort to overcome these difficulties, but even with the partial addition of "cured" rice to the daily dietary the number of cases of the disease fell progressively each year.

There is one point, which I have omitted to touch on and that is, that some employers have found
that the use of a rice, which retains practically all its pericarp, is apt to set up a rather irritable form of diarrhoea. It is not necessary to use such an irritating form, all that is required, is a grain, which is not highly polished and still retains 4% of P₂O₅, such a rice never sets up diarrhoea.

Apart from the dietary the general hygiene must be kept up to the standard.

The coolie houses should be well built, raised from the ground and thoroughly dry. The roof should be prolonged out to protect from the sun and the rain and keep the verandah dry. The houses should be so built, that with all windows closed there is still an abundant circulation of fresh air and should if possible face the prevailing wind.

I append a photograph of one of the latest type of coolie houses now being built on Christmas Island, and all these points have been attended to. As will be seen it is built on slopping concrete, which drains into an open gutter, which in turn passes directly into the sea.

The houses should be regularly washed out with some form of disinfecting fluid, this precaution will certainly do no harm and will often prove most beneficial. At Christmas Island the method employed was to start at one end of the coolie lines and do 3 houses thoroughly each day, until all were completed and then to start from the beginning again.
Efficient drainage must be provided and proper methods taken for the destruction and removal of night-soil. In Christmas Island the coolie lines, which were built at the top of the hill some distance from the sea, were provided with a most efficient home-made incinerator, which was cheap and worked admirably.

A good and abundant water supply should be provided.

Thus by careful attention to the form of dietary in use, coupled with ordinary hygienic precautions, the prophylaxis of the disease will be assured.

In the case of Ships the same points regarding food, sanitation and disinfection held good.

I append a few photographs to show the different forms of rice. So much has been said throughout this paper about rice, that it seemed to be of interest to show as clearly as a photograph can the naked-eye differences. In the case of the Indian rice the branny dark coloured pericarp can be seen and compared with the white look of the Rangoon or Siam rice.
Photograph of most recent type of Coolie House as erected at Christmas Island.

This may be taken as almost a model for this type of building.

Built on concrete, sloped so as to drain readily into a gutter in front of the building and so into the sea. Raised well above the ground and so easily kept clean. Thoroughly ventilated having 3 windows, and an air space of 2 feet at the bottom all round and 3 feet at the top, so that with all the windows closed there was still an abundance of pure air in circulation.

Covered with corrugated iron, which proved much more efficient in stormy weather than the original "attap" roofing and extended so as to keep the verandah dry as well. The extra heat from this form of roofing imaterial as houses not occupied during the day, but could be easily met with by covering the surface with a layer of attaps or a composition of tar, sand and white-wash.
Photographs of two forms of Rice.

(1). Sample of finely polished Siam rice, natural size. This is the rice that is so largely eaten by Chinese coolies. It is cheap and not of the first quality, as seen from the large number of fragmented grains.

Phosphorus percentage = 0.210  Cost = 6d for 10 lbs.

(2). Sample of Indian Rice, slightly magnified. This grain is not nearly so finely polished and so retains a great deal of the pericarp, the red colour of which can be seen in the print. It is the grain used by Indian coolies.

Phosphorus percentage = 0.469  Cost = 7½d for 10 lbs.
Photographs of two forms of Rice.

(1). Sample of ordinary third grade Rangoon rice, finely polished. A cheap variety largely purchased by coolies. Natural size.
Phosphorus percentage = 2.43 Cost = 5½ d for 10 lbs

(2). Sample of Chinese rice from Shanghai, this is also a polished rice, but a certain amount of the finer pericarp remains adherent. It is largely used by the Chinese for making cakes with. Natural size.
Phosphorus percentage = 2.61 Cost = 8 d for 10 lbs
(1). Sample of ordinary "padi", prior to milling, this is used by the natives of Malaya and China in the villages, being husked and winnowed by primitive methods. 1st 4th to 10th.

(2). Sample of "Kadiang Idju" or the "Java Pea". This legumen is mixed with a form of sugar known as "Gula Aren" and boiled to a gruel so as to represent a rice diet. It is given largely in Java, and when used Beri-beri does not occur.

(Botanical name = Phaseolus radiatus).
Photographs of two forms of Rice "Dust".

(1) This is the pericarp of the rice removed by the coarser form of milling, a lot of the fine pericarp remains adherent to the grain. There are some small fragments of rice amongst the sample. In Singapore used to feed pigs, in Rangoon made into cattle cakes for exportation.

Phosphorus percentage = 4.1.

(2) Similar to the above, but taken from a very finely polished rice, on which no pericarp was left at all.

Phosphorus percentage = 4.5.
---Dietetic Tables No 1---

Table to show the **Amount of Food-stuffs** (with daily variations) supplied per man per day.

<table>
<thead>
<tr>
<th></th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sam Rice</td>
<td>28 Sam Rice</td>
<td>28 Sam Rice</td>
<td>28 Sam Rice</td>
<td>28 Sam Rice</td>
<td>28 Sam Rice</td>
<td>28 Sam Rice</td>
</tr>
<tr>
<td>Indian Rice</td>
<td>2 Indian Rice</td>
<td>2 Indian Rice</td>
<td>2 Indian Rice</td>
<td>2 Indian Rice</td>
<td>2 Indian Rice</td>
<td>2 Indian Rice</td>
</tr>
<tr>
<td>Prawns</td>
<td>1/2 Prawns</td>
<td>1/2 Prawns</td>
<td>1/2 Prawns</td>
<td>1/2 Prawns</td>
<td>1/2 Prawns</td>
<td>1/2 Prawns</td>
</tr>
<tr>
<td>Lent</td>
<td>1/2 Lent</td>
<td>1/2 Lent</td>
<td>1/2 Lent</td>
<td>1/2 Lent</td>
<td>1/2 Lent</td>
<td>1/2 Lent</td>
</tr>
<tr>
<td>Onions</td>
<td>1/2 Onions</td>
<td>1/2 Onions</td>
<td>1/2 Onions</td>
<td>1/2 Onions</td>
<td>1/2 Onions</td>
<td>1/2 Onions</td>
</tr>
<tr>
<td>Pork</td>
<td>1/2 Pork</td>
<td>1/2 Pork</td>
<td>1/2 Pork</td>
<td>1/2 Pork</td>
<td>1/2 Pork</td>
<td>1/2 Pork</td>
</tr>
<tr>
<td>Vegetables</td>
<td>1 Vegetable</td>
<td>1 Vegetable</td>
<td>1 Vegetable</td>
<td>1 Vegetable</td>
<td>1 Vegetable</td>
<td>1 Vegetable</td>
</tr>
<tr>
<td>Cabbage</td>
<td>1 Cabbage</td>
<td>1 Cabbage</td>
<td>1 Cabbage</td>
<td>1 Cabbage</td>
<td>1 Cabbage</td>
<td>1 Cabbage</td>
</tr>
<tr>
<td>Tomatoes</td>
<td>1 Tomato</td>
<td>1 Tomato</td>
<td>1 Tomato</td>
<td>1 Tomato</td>
<td>1 Tomato</td>
<td>1 Tomato</td>
</tr>
<tr>
<td>Beans</td>
<td>1/2 Bean</td>
<td>1/2 Bean</td>
<td>1/2 Bean</td>
<td>1/2 Bean</td>
<td>1/2 Bean</td>
<td>1/2 Bean</td>
</tr>
<tr>
<td>Macaroni</td>
<td>1/2 Macaroni</td>
<td>1/2 Macaroni</td>
<td>1/2 Macaroni</td>
<td>1/2 Macaroni</td>
<td>1/2 Macaroni</td>
<td>1/2 Macaroni</td>
</tr>
<tr>
<td>Now Cake</td>
<td>1/2 Now Cake</td>
<td>1/2 Now Cake</td>
<td>1/2 Now Cake</td>
<td>1/2 Now Cake</td>
<td>1/2 Now Cake</td>
<td>1/2 Now Cake</td>
</tr>
</tbody>
</table>

---

**NB**

Under this heading comes Fruit (Papaya, Banana, Pumelo) eaten freely, both as a fruit and as a vegetable and Goods bought from Kongoi such as Mill, Flour, Peanuts, Raisins, Oranges, "Julie Malaccio" etc.
Table to show the approximate Analysis of Food-stuffs in daily use, with their Energy Value per ounce in Calories, and the Calorific Value to be obtained from the amount supplied per man per day.

<table>
<thead>
<tr>
<th>Food Stuff</th>
<th>% of Water</th>
<th>% of Protein</th>
<th>% of Fat</th>
<th>% of Carbohydrate</th>
<th>Energy Value per ounce in Calories</th>
<th>Quantity required per man per day</th>
<th>Daily Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>32.7%</td>
<td>5.0%</td>
<td>0.1%</td>
<td>41.7%</td>
<td>38.94</td>
<td>30.03</td>
<td>294.0</td>
</tr>
<tr>
<td>Pork</td>
<td>60.0%</td>
<td>12.3%</td>
<td>26.2%</td>
<td>0.1%</td>
<td>81.61</td>
<td>402.03</td>
<td>367</td>
</tr>
<tr>
<td>Lard</td>
<td>10.0%</td>
<td>89.5%</td>
<td>0.5%</td>
<td>0.5%</td>
<td>240.00</td>
<td>103.03</td>
<td>312</td>
</tr>
<tr>
<td>Salt Fish</td>
<td>16.5%</td>
<td>62.5%</td>
<td>0.7%</td>
<td>11.4%</td>
<td>99.42</td>
<td>230.03</td>
<td>228</td>
</tr>
<tr>
<td>Vermicelli</td>
<td>10.0%</td>
<td>12.5%</td>
<td>0.8%</td>
<td>75.5%</td>
<td>96.04</td>
<td>103.03</td>
<td>96.0</td>
</tr>
<tr>
<td>Bean Stick</td>
<td>10.5%</td>
<td>12.5%</td>
<td>0.5%</td>
<td>71.5%</td>
<td>96.04</td>
<td>103.03</td>
<td>72.0</td>
</tr>
<tr>
<td>Dried Peas</td>
<td>13.0%</td>
<td>21.0%</td>
<td>18.5%</td>
<td>26.2%</td>
<td>312.04</td>
<td>103.03</td>
<td>68.0</td>
</tr>
<tr>
<td>Fresh Vegetables</td>
<td>92.8%</td>
<td>1.0%</td>
<td>0.3%</td>
<td>3.6%</td>
<td>6.05</td>
<td>103.03</td>
<td>64.0</td>
</tr>
<tr>
<td>Macaroni</td>
<td>12.0%</td>
<td>10.8%</td>
<td>0.6%</td>
<td>75.7%</td>
<td>100.04</td>
<td>103.03</td>
<td>50.0</td>
</tr>
<tr>
<td>Dried Beans</td>
<td>13.0%</td>
<td>28.0%</td>
<td>17.5%</td>
<td>33.7%</td>
<td>67.48</td>
<td>103.03</td>
<td>50.0</td>
</tr>
<tr>
<td>Sweet Potatoes</td>
<td>72.9%</td>
<td>1.6%</td>
<td>0.5%</td>
<td>22.5%</td>
<td>28.71</td>
<td>233.8</td>
<td>66.0</td>
</tr>
<tr>
<td>Yams</td>
<td>79.6%</td>
<td>2.2%</td>
<td>0.5%</td>
<td>15.8%</td>
<td>21.71</td>
<td>233.8</td>
<td>49.0</td>
</tr>
<tr>
<td>Onions</td>
<td>89.0%</td>
<td>1.6%</td>
<td>0.3%</td>
<td>6.3%</td>
<td>9.54</td>
<td>1123</td>
<td>16.0</td>
</tr>
<tr>
<td>Prawns</td>
<td>25.0%</td>
<td>2.0%</td>
<td>0.4%</td>
<td>0.6%</td>
<td>6.20</td>
<td>1123</td>
<td>9.0</td>
</tr>
<tr>
<td>Salt Vegetables</td>
<td>75.5%</td>
<td>3.6%</td>
<td>21.0%</td>
<td>3.9%</td>
<td>233.8</td>
<td>233.8</td>
<td>8.0</td>
</tr>
</tbody>
</table>

NB. The above are not all supplied on the same, the daily rations are shown on Table 0.
Table to show the Total Potential Energy in Calories to be obtained from the Daily Food Supply with the relative percentages of Proteids, Fats and Carbo-hydrates.

<table>
<thead>
<tr>
<th>Day of Week</th>
<th>Food Stuff</th>
<th>Calories Value Daily Supply</th>
<th>Percentage of Protein</th>
<th>Percentage of Fat</th>
<th>Percentage of Carbo-hydrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mondays</td>
<td>Rice</td>
<td>2940</td>
<td>5.0</td>
<td>0.1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>Pork</td>
<td>367</td>
<td>12.3</td>
<td>25.2</td>
<td>—</td>
</tr>
<tr>
<td>Sundays</td>
<td>Love</td>
<td>312</td>
<td>—</td>
<td>8.9</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Sweet Potatoes</td>
<td>26.0</td>
<td>1.6</td>
<td>0.5</td>
<td>22.9</td>
</tr>
<tr>
<td>Tuesdays</td>
<td>Macaroni</td>
<td>50</td>
<td>10.8</td>
<td>0.6</td>
<td>75.7</td>
</tr>
<tr>
<td></td>
<td>Fresh Vegetables</td>
<td>29</td>
<td>1.0</td>
<td>0.3</td>
<td>3.6</td>
</tr>
<tr>
<td>Thursdays</td>
<td>Jam</td>
<td>49</td>
<td>2.2</td>
<td>0.5</td>
<td>15.8</td>
</tr>
<tr>
<td></td>
<td>Onions</td>
<td>16</td>
<td>1.6</td>
<td>0.3</td>
<td>6.3</td>
</tr>
<tr>
<td></td>
<td>Peas</td>
<td>8</td>
<td>4.0</td>
<td>0.4</td>
<td>—</td>
</tr>
</tbody>
</table>

Total Potential Energy = 3866 of which:
- Proteids supplied 11%
- Fat 36%
- Carbo-hydrate 52%

---

<table>
<thead>
<tr>
<th>Day of Week</th>
<th>Food Stuff</th>
<th>Calories Value Daily Supply</th>
<th>Percentage of Protein</th>
<th>Percentage of Fat</th>
<th>Percentage of Carbo-hydrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mondays</td>
<td>Rice</td>
<td>2940</td>
<td>5.0</td>
<td>0.1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>Love</td>
<td>312</td>
<td>—</td>
<td>8.9</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Salt Pork</td>
<td>228</td>
<td>62.5</td>
<td>0.7</td>
<td>—</td>
</tr>
<tr>
<td>Wednesdays</td>
<td>Vermicelli</td>
<td>29</td>
<td>12.5</td>
<td>0.8</td>
<td>75.5</td>
</tr>
<tr>
<td></td>
<td>Sausage</td>
<td>72</td>
<td>12.5</td>
<td>0.5</td>
<td>71.5</td>
</tr>
<tr>
<td>Fridays</td>
<td>Dried Beans</td>
<td>68</td>
<td>25.0</td>
<td>1.7</td>
<td>53.4</td>
</tr>
<tr>
<td></td>
<td>Onions</td>
<td>16</td>
<td>1.6</td>
<td>0.3</td>
<td>6.3</td>
</tr>
<tr>
<td>Saturdays</td>
<td>Peas</td>
<td>9</td>
<td>2.1</td>
<td>0.4</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Salt Vegetables</td>
<td>8</td>
<td>—</td>
<td>—</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Total Potential Energy = 3749 of which:
- Proteids supplied 25%
- Fat 20%
- Carbo-hydrate 55%
Hospital Dietary

Daily Supply per coolie with times of meals.

N.B. Hospital coolies were thus provided with an abundant and varied diet, considerably more than that of the ordinary working coolie and possessing an even higher Calorific Value.

<table>
<thead>
<tr>
<th>Time</th>
<th>Dietetic List</th>
<th>Food Stuff.</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.45 a.m.</td>
<td>Milk, Broken Hown, Sugar</td>
<td>Milk</td>
<td>12 oz.</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>Pulot (boiled)</td>
<td>Pulot</td>
<td>10 oz.</td>
</tr>
<tr>
<td>11 a.m.</td>
<td>Green Peas (boiled), Sugar &amp; Water</td>
<td>Peas</td>
<td>4 oz.</td>
</tr>
<tr>
<td>2 p.m.</td>
<td>Tea (no solids, no milk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 p.m.</td>
<td>Monday</td>
<td>Pork</td>
<td>4 oz.</td>
</tr>
<tr>
<td></td>
<td>Tuesday</td>
<td>Potatoes</td>
<td>3 oz.</td>
</tr>
<tr>
<td></td>
<td>Thursday</td>
<td>Brown Rice</td>
<td>1 oz.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Preserves</td>
<td>1 oz.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rice</td>
<td>1 oz.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vermicelli</td>
<td>1 oz.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>White Beans</td>
<td>1 oz.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Beans</td>
<td>1 oz.</td>
</tr>
<tr>
<td>6 p.m.</td>
<td>Milk (no solids)</td>
<td>Milk</td>
<td>12 oz.</td>
</tr>
</tbody>
</table>

 Extras:
(1) Pigeon Broth  ... frequently ...
(2) Fresh Fruit  (Banana, Papaya, Pomelo)  ... daily ...
(3) Fresh Vegetables  (Long Bean, Chinese Cabbage, Radish, Vegetable marrow, Bok choy, etc.)  ... frequently ...
(4) Juiced Pineapple  ... once a week ...
TREATMENT
OF
BERI-BERI.

"Nam, quoniam variant animi, variamus et artes;
Mille mali species, mille saluitis erunt".

OVID.
TREATMENT OF BERI-BERI.

From what has been said it will have been gathered, that if the prophylactic precautions indicated are taken, the necessity for treatment will steadily become less. However in view of the great prevalence of the disease at the present day it may be as well to make a few statements upon the subject, with special reference to certain modes of treatment, which have been adopted with success and not mentioned in the literature on the subject.

A. HYGIENIC.

The lines already mentioned should be followed out. If possible the patient should have a change of dwelling, if not of locality. I do not think the importance of this is as great as was formerly insisted upon, but a change of air and surroundings must have a certain beneficial effect. In Christmas Island good results frequently followed the removal of a patient from Loading Point up to the hill hospital at the top of Phosphate Hill (altitude about 1000 feet), which faced the prevailing wind.

The Beri-beri patient should be placed in a well ventilated and thoroughly dry ward, if possible one kept entirely for this disease. This I mention more for the protection of the case of Beri-beri, than for the other patients. The disease is not infectious, no coolie ever acquired Beri-beri in hospital at Christmas Island, but on the other hand a man suffering from
Beri-beri is far more liable to develop some other complication if exposed to it, most notably dysentery and tuberculosis.

Care in selecting the site for a hospital should be taken to see, that the drainage is efficient and that the water supply is pure and abundant.

The patient should be out in the sunshine as much as possible and avoid all forms of dampness.

In mild cases and during convalescence he should be encouraged to take gentle exercise, provided no cardiac condition prohibits it.

B. DIETETIC.

The daily diet should be carefully regulated. Many coolies demand rice and they should be given nothing but the "cured" form.

The Java Pea (PHASEOLUS RADIATUS) forms an excellent substitute and when cooked with Gula Aren (a form of sugar) can be made to resemble a sort of Rice mash, which is most palatable.

Care should be taken to see, that the stomach is never overloaded, only a moderate amount of food being taken at each meal. The principle meal should be in the middle of the day.

In the Tung Wah hospital Hongkong, patients may choose, which ward they prefer in one "cured" rice is eaten and in the other "uncured". In 1910 the mortality in the former ward was 19% in the latter it was 41%. The appended chart shows the progressive fall in the
number of cases of Beri-beri on Christmas Island, the result of improved hygiene and of gradually increasing the percentage of "cured" rice in the food.

The list following is the dietetic scale of hospital rations in the Government Hospitals of the Straits Settlements.
<table>
<thead>
<tr>
<th>Year</th>
<th>Admissions</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1901</td>
<td>680</td>
<td>229</td>
</tr>
<tr>
<td>1902</td>
<td>693</td>
<td>95</td>
</tr>
<tr>
<td>1903</td>
<td>591</td>
<td>60</td>
</tr>
<tr>
<td>1904</td>
<td>973</td>
<td>105</td>
</tr>
<tr>
<td>1905</td>
<td>544</td>
<td>12</td>
</tr>
<tr>
<td>1906</td>
<td>700</td>
<td>17</td>
</tr>
<tr>
<td>1907</td>
<td>269</td>
<td>5</td>
</tr>
<tr>
<td>1908</td>
<td>201</td>
<td>7</td>
</tr>
<tr>
<td>1909</td>
<td>149</td>
<td>2</td>
</tr>
</tbody>
</table>
### DIET FOR CHINESE.

- Rice: 1½ lbs daily.
- Fresh Fish: 3 oz 5 times a week.
- Salt Fish: 3 oz 2 times a week.
- Fresh Beef or Pork: 4 oz daily.
- Vegetables: 8 oz daily.
- Salt: ½ oz daily.
- Onions: 1 oz daily.
- Garlic: 1 oz daily.
- Lard: 1 oz daily.

### DIET FOR MALAYS.

- Rice: 1½ lbs daily.
- Fresh Fish: 3 oz 5 times a week.
- Salt Fish: 3 oz 2 times a week.
- Fresh Beef or Goat: 4 oz daily.
- Vegetables: 8 oz daily.
- Dhall: ½ oz daily.
- Salt: ½ oz daily.
- Curry Stuff: 1 oz daily.
- Coconut Oil: ½ oz daily.

### DIET FOR BENGALIS.

- Flour, Karak: 1 lb daily.
- Ghee: ½ lb daily.
- Vegetables: 6 oz daily.
- Dhall: 1 oz daily.
- Salt: ½ oz daily.
- Curry Stuff: 1 oz daily.

### DIET FOR SIHKHS.

- Flour, Karak: 1½ lbs daily.
- Ghee: 1 lb daily.
- Milk: ½ pint daily.
- Sugar: 2 oz daily.
- Kachhun Idju: 2 oz daily.
- Vegetables: 1 lb daily.
- Curry Stuff: 1 oz daily.
C. MEDICINAL.

This is almost entirely symptomatic, but the following general lines of treatment should be adhered to, as being of the utmost importance.

(1). The bowels should be kept freely open. For all time the saline purgatives have been held in honour in this respect especially Magnesia. Occasionally Castor Oil or Jalap may have to be resorted to and care must of course be taken not to overdo it so as to increase the weakness especially in oedematous cases.

(2). Massage at least twice a day with some simple liniment. I cannot lay sufficient emphasis on this. I have seen most splendid results by thus keeping up the tone of the muscles, and it is a precaution which is all too apt to be neglected, in places where a large amount of coolie labour is employed. It is so easy to do and many a Chinese coolie will be found to be a born masseur.

(3). The administration of Tonics during convalescence. During the acute stages when there is much hyperesthesia of the muscles Strychnine is contra-indicated, but in the later stages it is of the greatest value. The combination, that I found to be far the most satisfactory was Strychnine and Formic Acid (25%). The results, that one obtained were occasionally most striking, and others to whom I have recommended a trial of this formula, have expressed their great gratification at the effects produced.
The patients felt that they were improving, which is half the battle in the treatment of any disease, and especially such a depressing complaint as Beri-beri. They expressed a real sense of well-being.

Taking the results taken over 12 months, it was found, that the average period in hospital under this form of treatment was reduced from 47 to 34 days.

Cases in which it was found, that there was oxaluria present were given Calcium Formate in the hopes, that the Calcium would combine and form the insoluble Calcium Oxalate and liberate the Formic Acid. I think from what I saw, that the choice of drug was good, but I have not seen a sufficient number in which oxaluria was present to be able to judge the real therapeutic value of this line of procedure.

FORMIC ACID.

The question of the therapeutic value of Formic Acid is still quite undecided. Many glowing accounts of its use in certain diseases have been published and, from the good results obtained by Croom in Diphtheria, a trial of it in this disease seemed to be indicated.

It was originally held, that Formic Acid and the Formates, when properly given, had a powerful stimulating effect upon the voluntary muscular system increasing its vigour.

Clément of Lyons in a paper read before the Paris Academy pointed out, that the use of Formic Acid caused a feeling of increased strength, vigour and activity. He referred to its action "toni-musculaire" and asserted
and asserted that, using a Mosse Ergograph, an increased power of work could be demonstrated, even as much as 85 kilogrammeters. Further he stated, that the eagerness for work became greater, sleep more refreshing, movements more brisk and causes, which lead to exhaustion of mind and body, felt less. Later Huchard after 2 years treatment and experiment confirmed these views.

More recent work has shown, that the Formates do not increase cardiac tonicity, but they do increase the contractility of the heart. It is only right to mention, that the work of Goodall and Mitchell (1909) disposed of many of these claims of Formic Acid and they summed up with the conclusion, that in the doses stated Formic Acid had no demonstrable effect on the circulatory or muscular systems.

Apart from any experimental evidence whatever and judging simply by the clinical results obtained, I should certainly be inclined to the view, that the therapeutic power in certain cases is anything but small, it was frequently found to be of very exceptional value, and I should be sorry now to be compelled to do without it in the treatment of Beri-beri.

In acute cases it was found of value to administer the rice "polishings" made up with Aqua Chloroformi as a medicine. Coolies who refused to take the polishings in their food could always be got to take them in this form. By this means it was often prescribed throughout the whole course of the disease.
Occasionally one tried Sanatogen, but the price of this preparation is too prohibitive to enable it to be given to coolie patients, otherwise it should prove of value in this disease, for, I believe, it consists of Phosphoric Acid and pure Casein, the phosphorus being entirely in the organic form, thus supplying this essential element for the physiological well-being, which is so deficient in Beri-beri.

Such then are the general lines of treatment in this disease, for the rest it is purely symptomatic, emergencies being dealt with as they arise.

We will then pass on to consider the special treatment of the different prominent symptoms of the disease.

Edema.

So great in amount may this be, that the most energetic treatment is often called for. In the ordinary cases a simple diuretic is all that is necessary: of those in common use I have found Potassium Citrate to be decidedly the most useful, especially so on account of its non-irritating action on the kidneys, while any action it may have is at the expense of the edema. This was used as a routine in combination with Infusion of Digitalis.

In these cases spontaneous diuresis was the first and surest sign of convalescence. Tapping had on some occasions to be resorted to, and one case in which the urinary secretion had fallen to an extremely low rallied rapidly after being tapped on several consecutive days.
I am not convinced of the value of vapour baths; they cause a lot of discomfort in the Tropics and aggravate the vascular weakness. Though by this means a lot of water can be got rid of, yet the amount of Urea lost is small, so the kidney is left with a more concentrated mixture to deal with; this of course can be partly met with by reducing the amount of Protein in the food.

In dropsical cases it was customary to insist on a chloride-free dietart, which according to the views at present held ought to have had some beneficial effect. Pilocarpine was occasionally resorted to and this drug in combination with Sodium Salicylate was highly spoken of by Baclz.

GASTRO-INTESTINAL SYMPTOMS.

DYSPEPSIA.

This is often met with in early cases and can usually be dealt with by dietetic means plus the exhibition of such drugs as Rhubarb, Bismuth and Soda.

DIARRHOEA.

This in my experience is not met with so frequently, but it may become a prominent and even urgent symptom at any stage of the disease. It can usually be controlled by a careful attention to the diet accompanied by initial purgation and subsequent use of astringents and sedatives. I found, that in such cases, if the symptoms were irresporsive to this line of treatment, the administration of Milk curdled by the Lactic Acid organisms often proved of the greatest therapeutic value and would always employ this method in obstinate cases (see special case).
MICCOUGH.

This is often a prominent and ominous symptom in advanced cases. In order to combat it when it arises Cocain is often the best drug to use, and this can be considerably aided by the application of mustard over the epigastrium. By experience one found that the best way of applying this is to rub the dry powder into some cotton wool and then apply it without moisture to the skin, and cover it with oil-silk. The moisture of the body frees the sulpho-cyanate of allyl into action, and the result is that one gets a good reaction without any discomfort. Traction of the tongue is recommended and may be resorted to.

VOMITING.

In advanced cases one learnt to look upon this as a particularly dangerous symptom, in fact persistent vomiting at any stage is a thoroughly bad omen. The drugs recommended for Hiccough are all useful, especially Cocain, but sometimes the whole medical armamentarium may be tried and prove equally inefficient. The sucking of small quantities of Ice may allay the symptoms and gastric lavage is sometimes the only means of coping with the difficulty.

PARALYSIS OF THE DIAPHRAGM.

By the time that this symptom has occurred, I do not think, that the patient has very much chance. It is said, that the best results have been obtained
by electricity. One pole is applied to the epigastrium, the other to the root of the neck over the Phrenic Nerve. The application is made for a period of 10 minutes several times a day.

**CARDIAC SYMPTOMS.**

In mild cases the cardiac condition may not require any special treatment. Digitalis, if reliable preparation used, is always of the greatest when palpitation or any cardiac weakness shows itself.

In the Acute Fulminating Form a number of drugs have been advocated by different clinicians, a certain sign that none of signal value.

When the cardiac distress was very great, I have seen temporary relief from the inhalation of Amyl Nitrite, and Nitroglycerine (gr. 1/100th) every ¼ hour was recommended warmly by Manson. I have tried this method, in fact the dresser was instructed to have it always in readiness, and it proved of the greatest value in one case, in which the patient eventually made what appeared to be an impossible recovery. In other cases it has failed signally to produce the slightest reaction.

Strychnine provided that the muscular hyperaesthesia is not too acute is one of the sheet anchors and sub-cutaneous injections of Camphor will sometimes prove most beneficial.

The question of **VENESCTION** has to be considered. It is not by any means always advisable, especially in anaemic cases with a low blood pressure. In others
where there is no contra-indication the drawing off
of 10 to 20 ounces of blood is often most advantageous,
thus giving the engorged right heart a chance.

In cases where contra-indications exist good results
can be obtained by practising venesection upon one arm,
and transfusing at the same time into the other.

Or an excellent method in atrophic cases is to apply
a bandage of elastic to one limb in such a way as to
obliterate the venous return, but not to interfere with
the arterial supply, by this means the limb swells up
and quite a large quantity of blood can be cut off from
the system, this gives the heart a chance to rally, and
when matters have somewhat improved, the blood supply
can be gradually returned to the general system by
putting on the bandage at lower levels.

In cases where there is considerable pericardial
effusion, to such extent, that the cardiac action is
embarassed, paracentesis must be employed, as giving the
patient the only possible means of recovery.

In two cases I tried ASPIRATION of the Right
Ventricle, and in one of these the patient, who was
comatose at the time temporarily recovered consciousness.

RESPIRATORY SYMPTOMS.

The dyspnœa that is so often present is
usually cardiac in origin. Of whatever origin striking
use can be got by the method of dry-cupping.
The inhalation of wet Oxygen has been recommended, but
I have had no opportunity of judging its therapeutic
value.
In cases where the dyspnoea is a marked symptom, a hypodermic injection of Morphine in combination with Digitaline should be tried, also the pleurae should be frequently examined to ascertain if any effusion has occurred, which is such a constant cause of urgent dyspnoea, and if present to deal with it by tapping if necessary.

**MUSCULAR SYSTEM**

When the pain is very acute Strychnine is contra-indicated, but some relief can be obtained by the applications of hot fomentations and it may be necessary to give Morphia hypodermically. I have already laid stress on the value of massage to preserve the muscular tone. If Talipes Equinus occurs Tenotomy of the Tendo Achilles may have to be resorted to, or Fixation of the ankle joint, in order to give the patient a useful limb.

During convalescence Electricity should be of value; with the battery at my disposal I could form no adequate judgement of the full possibilities of this line of treatment, but imagine it might in some cases be almost indispensable. If muscular excitability still exists to some extent, one would recommend the Faradic Current otherwise the Galvanic.
CONVALESCENCE.

This has already been touched upon, but I would just sum up the remarks again. Massage and Electricity, graduated exercises with cold packs and douches recommended, and the ordinary tonics Strychnine, Iron and the like administered.

A drug much used in Brazil during convalescence and most highly spoken of, is the Fluid Extract of Marapuana (Livicosma ovata), given in doses of 10 to 20 drops a day.

Any other symptoms that arise such as Bedsores, Ulcerations, Convulsions (rare) and Laryngitis must be treated by ordinary means.

Other drugs, that have not been mentioned and may prove useful are Urotropine, in cases where some bladder trouble has arisen and Nuclein. This later drug I have tried hypodermically and a leukocytosis certainly resulted, but no marked alteration in the symptoms were noticed.

Such then are the general lines of treatment, that one would recommend, though but briefly referred to. Most of the drugs mentioned have at one time or another proved of conspicuous value, only unfortunately when put to the test on another occasion to prove singularly inefficient.

There is thus no specific remedy for the disease, and some cases will demand all the therapeutic resources available and call forth all the physicians ingenuity in their treatment.
It is really remarkable how on many occasions, when all seems hopeless, one may strike a remedy, that seems to act upon that particular patient like a charm and he makes a steady though always a slow recovery. Whether it is the drug, that one has administered that has really helped him round the corner or the VIS NATURAE, the saving effort of nature to retain the species, is not always a matter easy to decide, but it is always comforting to the physician to take the credit to himself, to wipe off a little from the list of those who have slipt through in spite of everything.
HISTORIES and NOTES on SPECIAL CASES
of
BERI-BERI.

I have added these notes on special cases of this disease, as I think, that they may be of some interest and value. They are copied from one's own Case book and there is in consequence a deal of repetition, but as some of the cases dealt with exhibited symptoms, which are of great rarity in Beri-beri, I do not think that they are altogether out of place.
Atrophic Beri-beri with double Post-beri-beric Optic Atrophy and Paralysis of the Diaphragm.

AGE 27
OCCUPATION COOLIE
NATIONALITY CHINESE—CANTONETSE.
ARRIVED C.I. FEB. 1908.

History.

On May 23rd 1908 he was admitted to hospital suffering from a mild attack of Beri-beri. He complained of numbness of the legs and fingers with weakness on exertion accompanied by some palpitation.

On examination there was comparative anaesthesia to all sensory stimuli down the anterior of the tibiae and over the tips of the fingers, hyperaesthesia of the calf muscles and complete loss of knee jerks. The heart's action was accelerated, but the valves were closed.

There was no oedema, no alteration in the Skin reflexes, and the Arm jerks were still present, but he was unable to perform the "squatting" test.

Placed under suitable treatment he improved steadily and was discharged apparently quite well on July 10 1908.

The knee jerks were present though slight and he could do the "squatting" test, while the heart was regular and well balanced. He returned to light work for 4 weeks, during which time he was under supervision and then went back to the quarries.

On October 22nd 1908 the patient was re-admitted on this occasion his only complaint was increasing loss of vision. With oblique illumination both lenses appeared clear.
Special Cases——

The pupils reacted readily to light and accommodation. There was no conjunctivitis and the only abnormality was an Internal Pterygium of the right eye, which did not interfere with the line of vision.

Ophthalmoscopic examination at this stage proved negative, a note was made, that the discs seemed to be rather pale, but this was attributed to his general condition, for he was distinctly anaemic at the time. On testing his vision he said, that he could not discern even large objects at a distance of 10 feet, and could not read any of the test letters, but could accurately count fingers at 18 inches from the Left eye and 12 from the Right.

As regards other evidences of nervous disorder none were present, the knee jerks were active and the heart was sound.

He was admitted to hospital and placed on tonic treatment, at first it was thought, that there was a large element of malingering about him, for at night when no one was about he was able to play Chinese cards with the other patients, but soon it became apparent, that he was steadily losing his sight.

Ophthalmoscopic Examination was frequently made and a slowly progressing double optic atrophy was seen to be occurring. This was a simple degenerative change, at no time was there any apparent neuritis preceding the atrophic change, no hyperaemia of the discs and no loss of definition of their margins.

On Dec 20 1908 the eye condition was as follows:-

1. The fundus was anaemic, vessels both arteries and
Special Cases——

veins were much diminished in size.
2. The disc was a pearly white colour with sharp clearly defined margins.
3. Vision was lost and the loss had apparently been concentric, he could still recognize a bright light when flashed on the retina.
An attempt to show the appearance of the right fundus is appended.
The general health remained perfectly good, his appetite was excellent and he seemed quite happy.

On January of 1909 arrangements were made to send him home, as he especially desired it, these took some time to complete as a lot of correspondance was necessary.

On March 29 1909 when everything was finally settled he developed a second attack of Beri-beri. It is interesting to note, that though in hospital only "cured" rice was eaten, yet the patient developed the disease, but on inquiry this interesting point came to light, that as there was nothing apparently wrong with the patient save his blindness, he had been allowed to go down to the coolie lines as much as he liked and had been practically eating nothing but "uncured" rice, for some time previously.
It may be considered, that the progressive degeneration of the Optic nerves, was an evidence of existing Beri-beri, but I am rather inclined to look upon it as a post-beriberic atrophy, for during all these months in hospital, when he had been under careful observation, he had none of the other manifestations of the disease. His knee jerks were active, locomotion was unimpaired,
the "squatting" test could be readily performed and the cardiac condition was highly satisfactory.

The onset of active symptoms of the disease seems to me to point to a fresh infection.

This attack, which was not preceded by any rise in temperature, nor was there any subsequently, was ushered in with Gastro-intestinal symptoms, and was at first diagnosed as being probably dysenteric.

On March 29th then he vomited 3 times and his bowels moved 16 times, the stools contained a trace of mucous but no blood. He received Castor Oil followed by Morphia, Chloroform and Bismuth. On the 30th he had 14 motions and on the 31st 19, after that the stools became less frequent, lost the trace of mucous and on April 4th had returned to a normal well-formed motion. The faeces were examined for the ova of Ankylostoma duodenale, but with negative results.

Two days later on April 6th he first complained of numbness of the hands and legs, and a feeling of oppression in the pit of the stomach with weakness of the limbs.

EXAMINATION.

Definite anaesthesia amounting to considerable blunting of sensibility and delay in perception to all stimuli. Loss of perception to Tactile and Thermal stimuli over the anterior and external surface of the legs, as far up as the knee (not of the feet) and of the finger tips and thenar eminences, all of which were almost symmetrical on both sides (fig 1). There was marked hyperaesthesia of the calf muscles
which felt flabby and toneless. The knee jerks were just perceptible, but the arm jerks were quite active. There was no alteration in the superficial reflexes, the gait was of the "stepping" type, he walked on a broad base, and raised the feet high in order to clear the ground. He was quite unable to perform the "squatting" test and testing the push of the foot showed the weakness of the extensors. He had no cardiac subjective symptoms and during repose the heart's action was regular and steady, only on exertion was there some cardiac irritability present, after attempting to walk a few steps the pulse rate would rise from 75 to 125. There was no oedema and no dyspnoea present.

The Eye. Pupils equal, moderately dilated, inactive to light. Sight by this time had completely gone.

Ophthalmoscopic Examination.

The fundus was paler and the vessels even more constricted and along them a white line could be seen to run on either side far out from the disc. The disc itself was sharply defined, slightly cupped and of bluish white colour. (There was no further alteration in this condition).

The Cerebro-spinal Fluid was clear, SP GR 1001, under no pressure, microscopically an occasional epithelial cell, no lymphocytosis.

The treatment at this time was on general lines, tonics including Formic Acid with absolute rest, massage, and a generous easily digested diet.
April 22, 1909

The patient continued to get steadily worse and by April 22nd the anaesthesia had spread to the level of the umbilicus and over the anterior surface of the forearms (Fig 2), this loss of sensibility to stimuli was complete over the legs from mid-thigh downwards and much impaired over the rest of the affected area.

The muscular hyperaesthesia was much more marked, no longer being limited to the calf muscles, while at night he suffered from cramps in the legs sharp and excruciating, causing him to cry out loudly, and requiring injections of Morphia to give relief. All the deep reflexes were absent, the superficial were present. He was unable to stand alone and foot-drop was apparent, while increasing weakness of the forearm muscles could be demonstrated. The heart was normal in size and position and the sounds remained closed, but its action was increased in rate, the rhythm being almost embryonic from the shortening of the diastolic pause. There was no oedema. Occasionally there were attacks of dyspnoea and a cough had developed, but no physical signs of any pulmonary disease.

The Blood, which had been examined frequently, was becoming progressively more anaemic (vide chart) and the Urine, which had hitherto been quite clear, became alkaline, pale, thick, foetid and had a deposit of phosphates.

Thus, with the more extensive involvement of the nervous system, the downward progress of the disease went on, presenting a clinical picture of striking interest.
On April 28th information regarding cutaneous sensibility could not be obtained, but there was general paresis of the limbs and marked muscular atrophy; the cramps though not so frequent were still severe and his whole body seemed to be extremely sensitive. Wrist-drop was very apparent and the feet had assumed the position of equino-varus. He had to be constantly shifted on his bed, speech was reduced to a hoarse whisper and coughing became more and more difficult owing to the paralysis of the laryngeal muscles and those of forced expiration. The heart was little altered in size, but the apex beat was displaced outwards into the axilla. The action was rapid and the first Mitral sound was replaced by a soft blowing murmur propagated out into the axilla as far as the Post-scapular line, the second sound was short and sharp. In the pulmonary area a systolic murmur was also audible. The pulse was still regular, but the Maximum Systolic Pressure was steadily falling. Slight oedema of the ankles present, but no hepatic enlargement or tenderness. The urine had rapidly cleared under the administration of Urotropine, but now retention set in and catheterization had to be employed. The blood remained much as before, but there was a steady increase of the coagulation period (vide blood chart). There was loss of control over the rectal sphincters and incontinence of faeces.

The Cerebro-spinal fluid was clear, watery, SP GR 1007, trace of albumen, and microscopically epithelial cells with an occasional lymphocyte.
On April 29th the last scene was ushered in with paralysis of the diaphragm, the costal arch became unduly widened, while the epigastrium was drawn backwards and upwards with each inspiration, all over this area there was marked tympanites and the apex beat was displaced outwards, (an attempt to show the condition present will be seen in the diagram).

There were frequent attacks of dyspnoea and complete aphonia. The superficial reflexes were lost. The patient lay stretched out and motionless, his sightless eyes "gazing" upwards, but consciousness was still present and nourishment could still be administered. The apex beat was still further out and the left side of the chest became dull, Blood Pressure continued to fall. The pulse at the wrist became impalpable, while auscultation over the heart revealed its tumultuous action with bruits in all areas.

On May 2nd a condition of coma came on, the extremities became perfectly cold and slightly livid, respiration shallow and ineffectual, while the cardiac action suddenly became slower and death silently intervened.

BLOOD CONDITION dealt with on separate chart, coagulation period advanced from 3 to 9 minutes.

URINE CONDITION
Dec 27 Acid, clear, amber, no abnormality.
Mar 29 - - - - Indican in abundance.
Ap 10 - - - - less.
Ap 22 Alk, turbid, pale, Indican trace, Phosphates abundant, pus.
Ap 28 Acid, clear, amber, slight urate deposit, no pus.
May 1 (catheter) 15 oz in 24 hrs, ac, 1017, clear, no abnormality, trace of Indican, Urea 36.25 g a in 24 hrs, Chlorides 59 g a, Micros: no organisms, "hyaline bodies."

May 2 (P.M. spec) Urea 4.00 g a to oz, Chlorides 40 g a.
NOTES on the CASE.

A full history of this case seemed to be of considerable interest, as it illustrates in many ways the salient points of Beri-beri, and at the same time possesses many symptoms not commonly seen in the disease.

The question of Treatment has been almost entirely disregarded in the description of the case, as it had little bearing on the matter, but throughout it was symptomatic. Before the second attack of Beri-beri occurred the patient was kept in the open air, given a generous dietary coupled with such tonics as Arsenic, Iron and Cod Liver Oil and under this his blood condition and general health improved. After Beri-beri symptoms developed treatment was directed to keeping up the tone of the muscles and avoiding deformities, by daily massage and the administration of Formic Acid, combined with general tonics. The cramplike pains were treated with Lead and Opium dressings and Potassium Bromide internally, and finally injections of Morphine. Later the failing heart required energetic and frequent stimulation, Strychine, Ether and Nitro-glycerine, Venesection also in combination with Adrenaline, proved of value.

In anaemic cases in which the venous blood pressure was high, it was not always deemed advisable to employ venesection, but a good substitute was found to be obtainable by applying an elastic bandage round the limbs, tight enough to obliterate the vein but not the main artery. By this means a large amount of
blood is, so to speak, cut off from the general circulation and the venous pressure falls and the heart obtains relief for the time. The blood is only allowed to return by degrees, namely by altering the position of the ligature, and thus a too sudden over-loading of the right side of the heart is avoided.

To return to the case in hand, it was pre-eminently Beri-beri of the True Atrophic Type, the clinical picture was complete, as shown by the ever-increasing anaesthesia of the skin, the stage of the acute cramp like pains, the flabby wasted muscles, the daily loss of flesh, the progressive loss of muscular power, with the Foot- and Wrist-drop. The retention of consciousness also was characteristic, retained indeed, till with incontinence of faeces, paralysis of bladder, diaphragm and larynx, attacks of dyspnoea and falling blood pressure, death closed the scene.

To the following points I would draw special attention.

The Progressive Anaemia.

As shown by blood examinations taken over a considerable period. It was held at one time by some, that Beri-beri might be caused by anaemia, but it is now certain, that the anaemia is a sequel of the Beri-beri, the toxin of which disease has apparently a very constant deliterious effect on the corpuscular elements of the blood. During the quiescent stage, when there were no apparent active symptoms of the disease, considerable improvement in the blood condition did occur, but hand in hand with the appearance
of acute symptoms there was progressive anaemia, which
did not lend itself to treatment at all. There was de-
tcrease in R.B.C. - Hh - and in the W.B.C. this last is
of interest for as a rule there is a certain amount of
leucocytosis; finally the blood film almost resembled
Pernicious Anaemia and the Color Index was 1, and besides
the alterations in shape, size and staining power a few
nomoblasts were also seen.

THE DOUBLE OPTIC ATROPHY.

This symptom is of great interest on account of
its rarity, for beyond the pneumogastric the cranial nerves
seldom become affected in Beri-beri. In Allbutt's
System of Medicine, Manson in discussing this disease
saya "the cranial nerves as a group and the higher
nerve centres being exempt" and "neither, unless very
exceptionally, is there any implication of the centres
or nerves of sight". Searching through the literature on
the subject I can find no mention of the Optic nerves
having been implicated.

On the other hand there is little reasonable
doubt, but that the complication was a sequel to the
first attack of Beri-beri, the onset of atrophy being
slow and progressive, a pure degeneration of the nerve,
to the naked eye unaccompanied by any inflammatory pro-
cess. The nerve supply of the extrinsic and intrinsic
muscles of the eye was unaffected.

Other points worthy of note, in the review of the
case on account of their comparative infrequency, are:
Special Cases

The complete anaesthesia of the legs to all sensory stimuli during the later stages of the disease. This occurs most often in atrophic cases such as this, but is not a common symptom and appears to me to be a probable sign, that a Radiculitis had occurred, similar to the degenerative process in the nerves. The Sensory Roots having become affected, especially as the specimen of Cerebro-spinal Fluid removed in the post-mortem room contained a distinct lymphocytosis, as compared with that examined during life before the advanced symptoms referred to had made their appearance.

The terminal loss of superficial reflexes is also of interest. The paralysis of the diaphragm with the characteristic tympanites and falling in of the abdominal wall on inspiration, is another point of note.

Also the slowing of the pulse just before death and the sudden final development of cardiac bruits in all areas, due possibly to cramp of the heart muscles themselves, for there was no post-mortem evidence of any valvular disease.

The manner in which structure after structure supplied by the Vagus became affected, formed a clinical picture of wonderful interest and completeness.

APPENDED ARE:

1). Post-mortem Notes on the Case.
2). Two Charts to show the increasing change of Cutaneous Sensibility.
3). Blood Pressure and Pulse Chart.
4). Diagram of the Fundus Oculi.
5). Table of Blood Counts.
6). Graphic Chart of Blood Counts.
7). Diagram to show Paralysis of the Diaphragm.
8). Micro-photograph of Transverse Section of Left Vagus Nerve.
9). Micro-photograph of Transverse Section of Right Optic Nerve.
POST-MORTEM NOTES on the CASE of LOH SUN.

May 2nd. Necropsy at 2 pm, 4 hours after death, weather hot and dry, marked emaciation of body. Rigor mortis complete. Pupils equal and medium. All superficial veins contain fluid blood, abdominal muscles pale.

ABDOMEN. Liver small, firm, anterior border sharp, bile runs freely, Kidneys healthy capsule strips freely. Spleen small, firm, cuts with some resistance. Stomach no congestion of mucosa, but marked pallor and friability with a few areas of necrotic absorption. Duodenum first part slightly reddened. Rest of intestines normal, no worms.

THORAX. Lungs and pleurae healthy, Pericardium contains slight excess of clear fluid, is non-adherent. Heart right side much dilated, full of dark, semi-fluid, very friable post-mortem clot, muscle wall of all chambers atrophied, pale, flabby and attenuated. Coronary vessels normal, cardiac valves healthy but aortic and mitral incompetent.

MUSCLES. Of lower limbs especially pale, wasted and flabby, no oedema of muscle or subcutaneous tissue. Cerebro-spinal Fluid removed post-mortem was clear and under no pressure, Sp Gr 1008, trace of albumen, no coagulation on boiling. Microscopically after being centrifulized, presence of a few cell elements epithelial and polymorpho-nuclear leucocytes, only a few lymphocytes (2 to a field).
Lob Skin  Benl Hai

Blue colour denoted impaired Sensibility
Red colour denotes complete lesion

April 22 1909
NOTES.

Blood Pressure readings registered during the last 10 days of life, taken by Martin's modification of the Riva-Rocci Sphygmomanometer. Chart shows the steady fall of the Blood Pressure and the rather rare terminal fall in the Pulse Rate. The final Blood Pressure reading was only approximate. All readings taken from the right arm with the body in the prone position.
Primary Optic Atrophy (Case of Lok Loy)

**Fundus.** Pallor towards the disc.
Vessels both veins and arteries diminished in size and showing characteristic white line.

**Disc.** Pearly white, slightly cupped, edges well-defined, lamina cribrosa well seen.
**Blood Counts**

**Name:** Loh Siew  
**Disease:** Beri Beri

<table>
<thead>
<tr>
<th>Date</th>
<th>Date</th>
<th>Red Corpuscles per c. mm.</th>
<th>Red Corpuscles per cent of normal</th>
<th>Haemoglobin per cent of normal &amp; normal</th>
<th>Colour Index</th>
<th>Leucocytes per c. mm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sep 28</td>
<td>1908</td>
<td>3,700,000</td>
<td>74%</td>
<td>74%</td>
<td>1,000</td>
<td>1,400</td>
</tr>
<tr>
<td>Dec 27</td>
<td>1909</td>
<td>4,400,000</td>
<td>88%</td>
<td>85%</td>
<td>0.965</td>
<td>7,200</td>
</tr>
<tr>
<td>1909</td>
<td>4-6.15</td>
<td>3,200,000</td>
<td>64%</td>
<td>70%</td>
<td>1.093</td>
<td>8,400</td>
</tr>
<tr>
<td>Jan 21</td>
<td>1909</td>
<td>2,900,000</td>
<td>58%</td>
<td>58%</td>
<td>1,000</td>
<td>6,400</td>
</tr>
<tr>
<td>Feb 23</td>
<td>1909</td>
<td>2,500,000</td>
<td>52%</td>
<td>52%</td>
<td>1,040</td>
<td>6,200</td>
</tr>
<tr>
<td>Mar 14</td>
<td>1909</td>
<td>2,500,000</td>
<td>50%</td>
<td>50%</td>
<td>1,000</td>
<td>6,300</td>
</tr>
</tbody>
</table>
### Special Cases

**LOH Sün.**

Graphic Chart to show the result of Blood Examinations.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>8,400</td>
<td>5,000,000</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8,200</td>
<td>4,750,000</td>
<td>95</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8,000</td>
<td>4,500,000</td>
<td>90</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,800</td>
<td>4,250,000</td>
<td>85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,600</td>
<td>4,000,000</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,400</td>
<td>3,750,000</td>
<td>75</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,200</td>
<td>3,500,000</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,000</td>
<td>3,250,000</td>
<td>65</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,800</td>
<td>3,000,000</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,600</td>
<td>2,750,000</td>
<td>55</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,400</td>
<td>2,500,000</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,200</td>
<td>2,250,000</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,000</td>
<td>2,000,000</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Temp.: 86°**

### Alkalinity

- **W.B.C.**
- **R.B.C.**
- **Hb**

### Temperature: 86°

**Alkalinity:** $\frac{N}{40}$ $\frac{N}{50}$

### NOTES.

Changes in the blood vessels were progressive, in the last Film (May 1st) the appearance was almost that of Pernicious Anaemia. There was marked pallor of the R.B.C. with alterations in size (macrocytes and microcytes) in shape (poikilocytosis) and in colour (polychromatophilia). Vacuolation was also present and 2 Normoblasts were seen.

- **Differential Leucocyte Count**
  - Polymorphs --- 68%
  - Lymphocytes --- 18%
  - Mononuclears --- 14%
LOH SÜN. Paralysis of the Diaphragm.

Loh Sun
April 30, 09

Area of Hepatic Decubitus.

Area of Sympomites.

Afferent Beat.
Transverse Section of Left Vagus Nerve (× 260)

Two bundles of nerve fibres separated by band of Endoneurium.

In the bundle nearest to the observer degeneration is advanced, and there are many large empty spaces with here and there a black dot indicating the remains of an Axis Cylinder.

In the bundle farthest off degeneration has also occurred, but is not so advanced, a few healthy fibres still exist.

The various degrees can be well seen in the original section, but the micro-photograph fails to bring them out very clearly.
Transverse Section of Right Optic Nerve. (×250)

Advanced degeneration, most of the nerve fibres have lost their axis cylinders, here and there throughout the section a dark spot indicates the remains of one less degenerated than the rest. The remainder of the section is a mass of vacuoles some containing degenerated fibres as a homogenous mass.

The endoneurium is sclerosed and wrinkled bands run across the section.
Case of Beri-beri with marked Hyperaesthesia and Laryngeal Paralysis.

Name: LOK LOY
Age: 24
Occupation: Coolie
Nationality: Chinese--Kheh.
Arrived: Feb 1909

HISTORY.

April 10 1909. Admitted to hospital with mild attack of beri-beri, improved rapidly and went out May 23 to do light work, as the heart still somewhat excitable. For the next 6 weeks he did not come near hospital. July 1st he came complaining of fever and headache, his temperature was 102 F and he was admitted. The knee jerks were present and there was no complaint of numbness.

Three days later however he complained of pain in the skin of the legs, the knee jerks were much diminished, and there was a trace of oedema over the tibiae, the calf muscles were not hyperaesthetic as far as one could make out.

July 9th considerable alteration in the condition, there was paraesthesia amounting to anaesthesia, symmetrical on both sides and rising to two inches above the knee, above this there was a zone of acute hyperaesthesia, the knee jerks were lost, but the arm jerks were present. There was no increase of the oedema.

The cardiac condition on admission was satisfactory, all the sounds were closed and regular. 120 mm
Max: Syst: Press: = 120 mm of Hg
Pulse Tracing--no change.
July 12th the zone of hyperaesthesia had spread up to the Manubrium Sterni, the slightest pinch or touch with a pin causing acute pain, the numbness passed up to the mid-thigh. There were no cramps. The arm jerks were lost and there was marked weakness of the extensors. The heart was considerably more rapid and excitable in action, though still regular, over the pulmonary area the 1st sound was replaced by a blowing murmur propagated down wards. Some enlargement of the Right Heart. \( \frac{2/3}{3} \).

This condition became progressively worse, the weakness of the muscles more marked and the cardiac condition more and more unstable, until dyspnoea set in.

July 19th has been getting steadily worse at 12 noon today he was breathing rapidly and with great difficulty. Resp = 42, Pulse = 135, Temp normal. Shoulders moving spasmodically, face white, pasty and puffy. Voice inaudible, heart irregular, very rapid with wild precordial pulsation. Skin of Abdomen, Thorax and Arms acutely hyperaesthetic. Right side of heart much dilated, venous pulsation in the neck. \( \frac{21/23}{3} \).

Other symptoms were not marked, the tongue remained clean, the bowels regular, slight oedema about the legs and face. Urine no abnormality except for reduction of the Chlorides (Amount 23oz). No Indican present. Blood examination shows slight anaemia no leucocytosis, but an ever increasing period of coagulation, having gone from 3½ to 7 min. in 3 days. Patient was bled 30 oz and transfused 25 oz NaCl Sol; followed by Nitro-glycerine gr 1/100th every 4 hour. At first after the Nitro-glycerine, there was a great
difference in the pulse tracing and he lay quietly instead of trying to toss about, but the improvement was only evanescent.

July 21 patient died, remaining conscious to almost the last.

NOTES on the CASE.

The points of interest in this case are the acute hyperaesthesia of the skin, the slightest touch causing agony and the terminal paralysis of the laryngeal nerves and muscles. This may have been due to pressure on the Right Recurrent Laryngeal nerve by the dilated right heart, but it seemed at the time to be more likely due to a toxic degeneration of the nerves themselves, and subsequent histological examination of the nerves proved that they were in a state of degeneration. The initial temperature was also of interest, it is said to occur in Beri-beri, but in my experience is very seldom seen. Lastly note the retention of consciousness right up to the closing scene.

Appended are:

1. Notes on the Post-mortem examination.
2. Temperature Chart showing initial rise.
4. A progressive series of Pulse Tracings, which are rather interesting as showing the increase in Rate passing on to Irregularity.
Post-mortem Notes.

At 9.30 am 12 hours after death, weather cool and damp (T 94). Rigor Mortis complete, no hypostatic congestion, body well clothed, pupils equal and medium, veins contain fluid blood.

Adhesions between Pericardium and Diaphragm, and Left Pleura and Diaphragm. No pericardial effusion. Heart considerably dilated. Rt Auricle and Ventricle contain antemortem clot extending ½ in into Pulmonary artery, Pulmonary orifice incompetent, Aortic competent. In section Rt Vent: wall thinned, not friable, left Vent wall some slight fatty infiltration, no endocarditis. Coronary vessels and Aorta healthy.

Lungs pale, fully congested, in section passive congestion on both sides.

Abdomen. Liver and Spleen enlarged, firm, congested. Gall Bladder contains fluid bile. Stomach dilated contains large quantity of inodorous fluid deeply coloured with bile, muc: membrane pale, except close to pylorus where there are several petecial submucous haemorrhages. No erosion of muc: membrane, Duodenum no congestion. Muscles pale and flabby, especially of calves. No oedema of glottis or of the subcutaneous tissue. A few enlarged glands in the groin.

Inoculation Experiment.

The serum obtained from bleeding the patient was injected into a guinea-pig (10cc). Result animal seemed unwell for about a week, but developed no paralysis or even weakness of the limbs, and quite recovered.
Temperature Chart.

<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>111.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>110.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>109.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>108.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>107.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>106.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>105.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>104.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>103.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>102.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>101.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>100.°</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. Pulse.</td>
<td>70</td>
<td>72</td>
<td>65</td>
<td>75</td>
<td>82</td>
<td>75</td>
<td>72</td>
<td>75</td>
</tr>
<tr>
<td>M. Respn.</td>
<td>24</td>
<td>24</td>
<td>23</td>
<td>26</td>
<td>26</td>
<td>24</td>
<td>25</td>
<td>24</td>
</tr>
<tr>
<td>R. O.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amount of Urine</td>
<td>145</td>
<td>20</td>
<td>154</td>
<td>22</td>
<td>17</td>
<td>20</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Sp. Gr.</td>
<td>1.015</td>
<td>1.011</td>
<td>1.015</td>
<td>1.005</td>
<td>1.010</td>
<td>1.012</td>
<td>1.010</td>
<td>1.009</td>
</tr>
<tr>
<td>Albumen</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
</tbody>
</table>
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

NOTES.

Atrophic case, developed acute symptoms on July 10th, and from that date onwards showed a steady fall of Blood Pressure with increasing Pulse Rate ending in Death. Just prior to death the patient developed acute swelling of the Submaxillary and Parotid Glands.
A series of Pulse Tracings taken at two-day intervals in a case of Beri-Beri that developed acute symptoms. The pulse rate became increasingly rapid and the Blood Pressure fell to a low point just before any marked irregularity of the pulse appeared. In acute Beri-Beri, the cardiac action may be seriously impaired before any marked irregularity of the pulse occurs. The pulse rate became increasingly rapid and the Blood Pressure fell to a low point just before any marked irregularity of the pulse appeared. In acute Beri-Beri, the cardiac action may be seriously impaired before any marked irregularity of the pulse occurs.
Special Cases—
LOK LOY

Acute Beri-beri.

Section terminal branch Right Recurrent Laryngeal Nerve. (×250)
Fibres varying in their state of degeneration.
To the left some almost normal and a large number of intact Axis Cylinders. In the centre the fibres are replaced by homogenous masses. To the right a band of epineurium and a collection of Red Blood Corpuscles.

Section of Phrenic Nerve. (×250)
This section shows nerve degeneration in all its stages. To the right some fibres have been cut longitudinally and the twisting of the Sheath of Schwann and an attempt at beading of the medullary substance can just be made out. Further over the fibres are cut transversely and some are still healthy, others retain their Axis Cylinders, and others are quite empty.
Chronic Beri-beri with Trophic Skin Lesion.

Name Lim Leh.
Age 33
Occupation Coolie
Nationality Kheh—Chinese.
Date of Arrival August 1906.

Disease Beri-beri.

History.

On July 19 1907 patient was admitted to hospital with all the symptoms of Beri-beri, there was nothing special to note about his case and after 7 weeks treatment in hospital, he was discharged on Sept 8th in good health. The Knee Jerks were present, but owing to increased excitability of the heart's action with a tendency to palpitation on exertion, he was put on light work.

On January 2 1908 he was readmitted with Acute Beri-beri, having every appearance of a rapid fatal termination from Cardiac failure. He complained of palpitation, shortness of breath, pain over the heart, with great weakness, numbness and tingling of the legs. Locomotion was just possible with aid and the gait was of the "high-steppage equine" variety.

On examination,

Patient lies prone, face pale, puffy and not cyanotic, but very anxious. Respiration rapid, Pulse greatly accelerated, but without irregularity in rate or rhythm, apex beat diffuse, heaving, tumultuous.

At this time the patient was too ill to give accurate replies to any questions, but there was comparative anaesthesia of both legs below the mid-thigh.
and of the hands, with marked hyperaesthesia of the calves and oedema over the tibias and round the ankles. Knee Jerk and Achilles Jerk were absent, Arm Jerks present, no Babinski's sign, no clonus. There was pulsation in the veins of the neck and epigastrum and the right side of the heart was enlarged. On auscultation the sounds were closed, but the cardiac action was very rapid, with marked shortening of diastole giving rise to a tic-tac embryonic rhythm. The Mitral 1st was weak and muffled, the 2nd Pulmonary short and accentuated. There were no gastro-intestinal symptoms, and no vomiting, but the stomach was dilated and highly tympanitic, and the bowels constipated.

The patient was kept at absolute rest and the bowels freely opened. Cardiac stimulants were administered and after a period of anxiety his cardiac condition improved, but there was little change in his other symptoms. He gradually developed into the slow chronic type of Beri-beri with constant numbness of the legs and arms, loss of knee-jerks, flabbiness of the muscles, slight oedema and an irritable heart.

On July 15, 1908 his condition was as follows:--
Numbness and deadening of sensibility over the crest of the tibias, the posterior surface of the calves, the wrists and fingers and the back inferior to a line joining the lower angles of the scapulae, higher on the Left side than on the Right.

The interesting point of the case was that over those areas where the patient complained of numbness, a skin
eruption had broken out, apparently of truly trophic origin and not found where sensation was normal. It was especially marked over the calves and lower part of the back (see the accompanying photograph). In type it was acneform, but the spots were purpuric in appearance, being dark purple and not coming "to a head". There was slight hyperaesthesia of the calf muscles, absolute loss of all the Deep Reflexes, but activity of the Superficial. The blood showed progressive anaemia, with a definite leucocytosis but no eosinophilia. The Urine was normal in amount, but the Chlorides were markedly diminished. The patient was still unable to walk, but the weakness was principally of the flexors of the foot and there was no Foot or Wrist Drop. The heart was very easily thrown out of balance, the slightest exertion causing the pulse to race away. There was still cardiac enlargement to the right, but now no extracardial pulsation and the apex beat was limited to the 4th and 5th spaces. A soft blowing Mitral systolic murmur was present, propagated out to the axilla, the Pulmonary second was reduplicated.

On January 2, 1909 just a year after admission the progress made was very slow though quite definite. The patient now had only slight numbness of the legs, no oedema and the skin rash had disappeared except over the calves. He could walk with the aid of a stick, the gait being now of the ataxic type as seen in the photograph on page 118. The heart was not enlarged, and all sounds were closed and only after considerable
exertion was there much acceleration of the heart's action.

On March 28, 1909, he left for China. He was able to get about quite readily with the aid of a stick, he had no numbness and only complained of weakness after exertion. The knee jerks were still lost, and the heart was regular and he felt well and could take his food heartily.

The rash had quite disappeared.

**Blood Counts.**

<table>
<thead>
<tr>
<th>Date</th>
<th>RBC</th>
<th>Hb</th>
<th>WBC</th>
<th>Film</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 19, 1907</td>
<td>4,400,000</td>
<td>90%</td>
<td>3,300</td>
<td>no change.</td>
</tr>
<tr>
<td>Jan 2, 1908</td>
<td>4,100,000</td>
<td>85%</td>
<td>3,700</td>
<td>no change.</td>
</tr>
<tr>
<td>July 15, 1908</td>
<td>3,200,000</td>
<td>72%</td>
<td>9,300</td>
<td>pallor of RBC, etc</td>
</tr>
<tr>
<td>Jan 20, 1909</td>
<td>3,700,000</td>
<td>79%</td>
<td>11,000</td>
<td>lymphocytosis.</td>
</tr>
<tr>
<td>Mar 28, 1909</td>
<td>4,100,000</td>
<td>85%</td>
<td>10,200</td>
<td>slight increase in lymphocytes.</td>
</tr>
</tbody>
</table>

**NOTES ON CASE.**

The chief interest in this case is the skin eruption, such trophic lesions are very rare in Beri-beri. In distribution it corresponded so closely with the areas of anaesthesia, that there could be little doubt that this was the cause. Another interesting point was the acute cardiac condition, which seemed to portend the gravest results, being largely caused by the pressure of a very dilated stomach on the heart. This should always be remembered in Beri-beri, as frequently with relief of the symptoms the cardiac condition rapidly subsides.
Skin Eruptions in Beri-beri are rare. This case is of interest, in that a well-marked Purpuric Rash developed over the areas of anaesthesia. The anaesthesia spread upwards very extensively, affecting even the back and the rash followed its course with great accuracy.

In the photograph an attempt has been made to show the rash over the calves of the legs and the back, where like the anaesthesia it reached a higher level on the Left side than on the Right.
Lim Lab
July 15th, 1908

Areas of Partial Anesthesia.

N. trig.
N. supracristal.
N. cut. medialis.
N. cut. medius.
N. cut. lat.
N. infra.
N. palm.
N. median.
N. trunci.
N. cut. lat.
N. cut. post.
R. cut. lat.
N. peron.
N. saph.
N. peron. super.
N. saph.
N. peron. prof.

N. occip. maj.
N. occip. min.
N. Auric magn.
N. supracristal.
R. Dors. N. spin.
N. asill.
N. cut. medialis.
N. intercost.
N. cut. medio.
N. cut. clunium.
N. sacro-tub.
N. cut. post.
N. obli.
N. cut. int.
N. obliter.
R. cut. post.
N. peron.
N. cut. lat.
N. peron.
N. saph.
N. medialis.
N. plant. lat.
N. plant. med.
Case of Beri-beri with Gastro-intestinal Symptoms.

Name: HONG MING
Age: 35
Occupation: Coolie
Nationality: Chinese--Hylan.
Arrived: Aug 1907.
No previous Beri-beri.

History.
Patient was admitted to hospital on October 6, 1909, complaining of vomiting and gastric pain, with numbness of the legs and hands, weakness and loss of energy, but no history of breathlessness or cardiac pain.

Examination.
There was definite impairment, over the anterior and internal surfaces of both legs and around the ankles, to sensory stimuli. In a like manner, but to a less degree, were affected the tips of the fingers and the wrists. Over these areas there was also paraesthesia in the form of numbness and tingling. The calf muscles were hyperaesthetic, the Knee Jerks were lost, and there was slight oedema over the crests of the tibiae and around the ankles. There was no fever. The heart was not enlarged and the valves were closed in all areas, there was accentuation of the Mitral 1st sound, and the whole action though regular was markedly accelerated on the slightest exertion. The gait was typical of a Peripheral neuritis. Blood examination proved negative.

The Gastro-intestinal symptoms were the most urgent, the patient had spasms of abdominal pain accompanied by vomiting. The vomit was slightly bilious but principally undigested milk curd.
Diarrhoea was present with frequent evacuations, dark in colour and extremely offensive, but containing neither blood nor mucous, and was accompanied by considerable tenesmus.

The spasmodic nature of the abdominal pain suggested intestinal obstruction, but there were no abdominal patterns. At times the sharp and shooting like the Girdle Pains of Locomotor Ataxia.

The Urine was slightly clouded, dark amber in colour, Sp Gr = 1023, Acid, with little diminution in the daily excretion (42 oz). There was reduction of the total amount of Urea (gr 4 to the oz or 190 gr in the 24 hrs) and of Chlorides, as estimated by Mohr's Method, (namely 75% or 148 grs in the 24 hrs).

Lastly there was a large quantity of Indican giving an intensely deep purple blue colour.

The Cerebro-spinal Fluid clear, watery, Sp Gr = 1006, under no pressure; Film stained with Leishman negative, no lymphocytosis.

The treatment adopted was the administration in 1 gr doses per hour for 5 hours, followed by Mag. Sulph: next morning. Hot Antiphlogistine was applied as a poultice to the abdomen, and a mixture containing Bismuth and Tr Morph: et Chloroform: was given the following day every 4 hours. The result of this free purgation was satisfactory, but the stools maintained their colour and offensiveness and so B-Naphthol was administered and the vomiting ceased, but the pain continued.
Special Cases

On October 12 there was still persistent and offensive diarrhoea, alkaline in reaction, with continuance of the abdominal pain. Indican in the urine was abundant.

The patient was then put upon milk curdled by Bacillus Bulgaricus, to obtain this Lactobacilline was used in powder form. The reaction of the curdled milk was tested and the bacillus demonstrated before use, each administration was accompanied by a spoonful of Malt Syrup.

The result was most striking, the amount of Indican in the urine decreased progressively, and with the diminution the abdominal symptoms disappeared, the pain ceased entirely 2 days after and within 6 days the motions became lighter in colour, more formed, less frequent and almost inodorous. The patient was on the full diet given to Beri-beri cases on the 8th day after starting the Lactobacilline, by which date there was only a faint trace of Indican in the urine (Oct 20).

The rest of the history is of little importance, the patient was placed on Tonic treatment including Formic Acid and energetic massage.

On Nov 2nd, he was able to do light work and on the 12th he was discharged and returned to full work at the quarries, the cardiac action being steady and the knee jerks present.

NOTES on the CASE.

It is not rare to find an attack of Beri-beri ushered in with diarrhoea, when this is present however it is usually of short duration and is readily amenable
to treatment, occasionally blood and mucous are present suggestive of Dysentery. As a rule however there are no abdominal symptoms in the initial stage, this case therefore seemed worthy of record, for these symptoms called attention by their very severity. The shoot-
ing girdle-like pains pointed to some active infection of the Sensory Roots, but the Cerebro-spinal Fluid did not show any attempt at lymphocytosis.

Bülz stated, that Indican is abnormally abundant in Beri-beri, but this has not been ones experience, there is always a certain amount present, but it is only in excess in those cases, which have gastro-intestinal symptoms, and seldom is it in such abundance as in the present case.

The presence of Indican in the Urine can be taken as a very fair guide to the extent of the putrefactive changes occurring in the bowel. Unfortunately there is no ready way, that one knows of to accurately estimate the precise quantity, and the colour reaction alone was taken as the guide. An attempt has been made in the accompanying plate to show the successive changes, which occurred so rapidly under the administration of Lactobacilline.

The test used was as follows:—To 10 c.c, of Urine add 10c.c of Pure Hydrochloric Acid and shake the mixture thoroughly. Add a few drops of Hydrogen Peroxide and mix. Allow to stand till the maximum colour develops, then add 2c.c of Pure Chloroform, again shake well and allow to settle, the amount of Indican is judged by the depth of the blue colour of the precipitate.
Metchnikoff's comparatively recent work on Lactic Acid organisms has led certain manufacturing chemists to place upon the market in the form of tablets or powders, what purport to be dried cultures of the true Bacillus Bulgaricus. The form used in this case was that known as Lactobacciline, which is considered a reliable production—a pure culture in association with certain paralactic organisms. In order to test its efficacy a quantity of condensed milk, diluted and boiled, was inoculated with the ferment and incubated at 37°C. After 24 hours a firm clot had formed, this was abundance of time for the production of an adequately soured milk, and for the predominance of the Lactic Acid organism. The curd, strongly acid, was examined microscopically and Bacillus Bulgaricus was found in abundance as well as a diplococcus and a yeast. The curd was then administered to the patient. The change in the consistency of the feces was most marked, and after 7 days the Lactic Acid organism was regained from the stools and cultured on Glucose Agar.

As the etiological factor in Beri-beri is considered by many to be of intestinal origin, it is at least rational to hold, that the less abundant the toxic intestinal flora are, the better are the chances of the patient's recovery, and the prompt reaction in this case to the Lactic Acid organism has led one to use this line of treatment in all cases of Beri-beri, in which Gastro-intestinal symptoms are at all conspicuous.
INDICAN TEST.

Case of Acute Beri-beri with marked Gastro-intestinal symptoms.

Six Indican Tests at intervals of two days, while under the administration of Milk curdled by the Bacillus Bulgaricus. Progressive diminution of Indican corresponding absolutely with alleviation of the Gastro-Intestinal symptoms.

Eventual Recovery complete.
CASE OF CHU CHOY.

AGE 32
OCCUPATION Coolie
NATIONALITY Chinese—Hheh.
ARRIVED January 1909
ADMITTED March 1909.

The notes of this case are only given in brief.

HISTORY.

He felt quite fit since his arrival on the Island three months ago, until today, when he noticed that his legs were feeling numb, and so came to the out-patient department for treatment.

EXAMINATION.

On admission paraesthesia over the anterior and external surfaces of both legs below the knee, the ankles are not affected.
Calves slightly hyperaesthetic. Knee-jerks absent.
Slight pre-tibial oedema none of the perineum or scrotum. He is unable to perform the "squatting" test, and walks on rather a broad base with a somewhat thumping gait, there is no foot-drop.
The heart sounds are regular and closed, but easily accelerated. There is cardiac pain, but no dyspnoea.

PROGRESS.

This patient was similar to any other case of Bari-beri with the exception that the oedema, which was at first so slight, became daily more and more marked, until on the 10th day after his admission, he was an enormous size, swollen out with general anasarca. All forms of treatment were adopted to try and reduce
the dropsy and at last tapping was resorted to both of the legs and abdomen. This was done by means, either of a simple incision or by the use of Southey's Tubes.

The urine had progressively diminished and had fallen as low as about six ounces a day. It was therefore feared, that at any time he might develop Uraemia.

The heart continued to act efficiently and his appetite was very fair. The Blood Pressure remained low.

After being frequently tapped and kept on a rigid chloride-free diet, the amount of urine gradually began to increase.

He was taking by the mouth a mixture containing Potassium Citrate, Caffeine and Infusion Digitalis.

The kidneys having recommenced their function the general anasarca became less and less and he made a complete recovery, being discharged on the 32nd day.

With the absorption of the oedema the atrophic condition of the muscles became apparent.

One point of considerable interest was that, during the free purgation, which was resorted to when the dropsy began markedly to decrease, he developed for a few days gastro-intestinal symptoms, passing frequent motions containing blood and mucus. There was no rise of temperature and no indication, that the condition was dysenteric and it seemed to me at the time, that the probable cause was the too rapid excretion of toxic
Special Cases——

substances by the intestinal mucous membrane.
This case therefore seems to me to be of some interest,
the comparison between the analysis of the drossical
fluid and that of the urine, especially as regards
the total solids they contain, bears out what one
found in other cases and may be taken as a sample
of what one found in the analysis of these cases.

Appended is the actual analysis, with all the figures
given in full, such was made on each occasion, that
the patient was tapped.

Also appended a Graphic Chart to show the actual
amount of Urine passed each day, with clinical notes
margined to show the change in the Maximum Systolic
Pressure, and the days on which tapping took place,
and when the gastro-intestinal symptoms became
manifest.
CASE OF CHU CHOI.

ANALYSIS OF DROPSICAL FLUID AND URINE.

Droptical Fluid.

| Weight of dish | 46.8410 |
| Weight of dish & fluid | 66.9138 |

Fluid evaporated on water bath.

| Weight of dish & residue | 46.8723 |
| Weight of dish | 46.8400 |

Residue = 1.14 % SOLIDS.

Residue ignited over bunsen.

| Weight of dish & residue | 46.7900 |
| Weight of dish | 46.8400 |

Residue = 0.74 % mineral salts.

Residue treated with HCL Ag solution filtered and tested for sulphates. Glazed surface of porcelain dish was badly etched and dish after experiment weighed 46.0650, a loss of weight of 0.0073.

TOTAL SOLIDS = 1.14 %
SALTS = 0.74 %
SULPHATES = 0.17 %
ALBUMEN = trace.

DROPSICAL FLUID ANALYSIS complete.

APPEARANCE Pale, clear.
SPECIFIC GRAVITY 1003.5
SOLIDS 1.14 %
ALBUMEN faint trace.
MINERAL SALTS
SOLUBLE 0.72 %
INSOLUBLE 0.02 %
TOTAL 0.74 %, of which 0.17 % were as Sulphates.

Urine estimated by same process.

TOTAL SOLIDS 5.32 %
ALBUMEN absent.
Clinical Notes on the margin, show the changes in Blood Pressure, and the Treatment adopted.
EXPERIMENTS.
EXPERIMENTS.

Most of the experiments carried out during the research into the subject of Beri-beri, proved negative. It may however be as well to indicate their nature in a brief summary.

(1).
Experiments to test the Arsenic in the hair of Chinese coolies, with a view to disproving the Arsenic Theory.

The nature of these has already been stated and it is not necessary to enter into details again. See page 16.

(2).
Inoculation experiments with animals.

The results of these were as a whole negative. The few positive ones probably arose from faulty technique: they have already been discussed in detail upon page 27 et seq.

The following were also undertaken:—

A. The blood serum from a patient with an acute attack of Beri-beri, upon whom venepuncture had been performed, was injected sub-peritoneally into a guinea-pig with all the usual precautions. No change occurred, the animal remained brisk and no paresis of the limbs showed itself.

B. Another guinea-pig was injected with the Cerebro-spinal fluid from an acute case, in which a cytological examination of the fluid showed the presence
of lymphocytes. Taking this as an indication, that the nerve roots were probably affected, it was thought, that this might prove more toxic than the samples tried up to that time, but again the results were absolutely negative.

C. Fowls similarly inoculated with blood serum an Cerebro-spinal fluid from an advanced case of the disease, showed no change whatsoever.

(3). Feeding Experiments.

A. A number of Fowls were put on mouldy rice, the pickings from the bags, often containing a number of weevils. Though on this diet for many months they continued healthy and laid eggs regularly. The reason of this was at first obscure, but later it was seen, that along with the fragments of rice, they were getting a lot of the rice polishings sticking to the bags. Further the amount of weevils eaten may have to some extent added a fresh factor to their dietary, and possibly, though I mention this with reservation, the fine phosphate dust, which settled from the works upon the enclosed area where they were kept, may have had some beneficial effect.

B. A few fowls were then taken and kept in separate pens, and not allowed to run about the enclosed area. They were fed on pure"white"rice, and after a period varying from a few weeks to several, changes occurred:—the birds went off their food, their combs drooped and became anaemic and finally they showed weak-
weakness of the legs and wings and did not lay any eggs. This was the most positive result obtained, and one next intended to have divided them up into two parties: one to continue to eat "white" rice, until an opportunity to examine their nerves histologically occurred, and the other party to be put upon "red" rice or "white" rice plus polishings, in order to watch if any improvement in their condition took place.

Unfortunately at this point a signal disaster occurred for one morning all were found dead and some were missing as well. At first it was thought, that they had succumbed rapidly to the disease, but it was seen apparent that they had been killed by rats.

Want of time prevented one carrying out this experiment on a second occasion.

This was most unfortunate, for in the light of Fraser's experiments, it is very probable that a decided improvement would have occurred amongst those, who were eating the "red" rice.

C. Fowls fed on rice with which was mixed oxalic acid for many weeks showed no change at all, except for two, who without being definitely paralysed were weak about the legs, but the condition in these two did not seem to alter at all for the worse.

The experiment was abandoned, as the birds were required for another series, probably it was not continued over a long enough period, but it did not seem, that much value was going to be derived from a mere repetition of Trentlein's experiments.
D.

The next and final experiment to be recorded is, I think, of considerable interest.

A monkey, *Macacus nemestrinus* referred to on page 50, fed on fruit and toxic rice, began to develop paresis of the lower limbs. I should mention, that a short time previously the animal had received a subcutaneous injection, consisting of an emulsion made from the nerves of a case, that had died of acute cardiac beriberi. I do not think, that this had any effect on the animal at all, for some fowls done at the same time were also uninfluenced, but it is unfortunate, that this was done as it tends to complicate the experiment.

At first it was noticed, that he was off his feed, then that he seemed less active. Gradually symptoms became more marked, he was unable to hold on with his feet and could not grip one's finger with his toes, but his hands remained quite useful.

By degrees the paralysis increased in amount and spread up the limbs, until the whole of both lower limbs were involved: the feet were cold and clammy and slight oedema was present. The animal now scarcely moved, but when he did so, dragged himself about with his hands, while his legs trailed uselessly behind him. He seemed very unhappy and scarcely ate anything.

The symptoms which at first were slow, now became rapid and he died with great dyspnoea and general muscular weakness, there was no rise of temperature and vomiting only occurred on the last day.
It was most unfortunate that symptoms developed so quickly, as it was then too late to note any beneficial changes from the alteration to a diet of "red" rice.

I am not prepared to say that this was a case of Beri-beri in a monkey, but the symptoms resembled that disease most faithfully. I believe that at the Research Institute at Kuala Lumpur, they have not been able to produce in a monkey symptoms analogous to Beri-beri.

At the autopsy a small amount of fluid was found in the pericardium, the cardiac muscles were flabby, the voluntary muscles were atrophic and almost yellow in colour especially those of the legs. Microscopic examination of the cardiac branches of the Vagi, and of the terminal twigs of the Sciatic, showed typical nerve degeneration in all its stages.

I have unfortunately not as yet been able to prepare sufficiently good micro-photographs of the slides in my possession, but will publish the case in the near future with complete details.

The breaking up of the myelin substance and the eventual disappearance of both the myelin substance and axis cylinder, leaving only the empty sheaths of Schwann, is well seen. The changes in the muscle fibres is also typical, the loss of striation and the presence of fragmentation is well marked. In the cardiac muscles these changes are not so advanced, but there is distinct fatty degeneration.

Before closing the account of this interesting case
case, one further point should be mentioned. When the monkey was first observed to be growing weak, it was attacked by a small dog, being unable to escape up its own pole, it turned on the dog and bit it deeply over the hind quarters, the native dresser hearing the noise, rushed out and tried to soothe the monkey, who in its fright sunk its teeth into the man's right wrist.

Four weeks later the dog developed paralysis of the hind limbs, at first it had difficulty in turning quickly and later could not rise at all even to drag itself to its food. After 4 weeks more the symptoms, which had never spread to the fore legs at all, gradually passed off and the animal made a complete recovery.

I thought at first, that the dog was developing a form of Landry's paralysis, which I believe is not uncommon in animals, who have received an unclean bite.

I am unable however to offer any explanation of the symptoms, although they were definite enough.

The native dresser did not take proper care of his wound, and I saw it two days later for the first time, when it was already septic and required to be freely incised. The wound healed slowly and the final touch to the picture was when, 7 days after the dog had developed its paresis, I was sent for at night, the message being that the dresser was very ill.

On arrival I found the man with face purple and bloated, teeth tightly clenched and his whole body in a state of
opisthotonos.

The spasm passed off and was followed by a series of violent clonic abdominal spasms, then another tonic one; the temperature was 103° F.

I thought he had developed hydrophobia and was presented with the following:— the man inside, the monkey on the verandah and the dog under the house.

The patient responded to Bromides and Chloral in large doses and though he had several attacks at longer intervals, they were gradually less severe; and during convalescence he was sent away for a change, to return quite fit.

His attack I attribute to hysteria with an element of alcoholism behind it, and I need hardly say that it was not true hydrophobia.

But at one o'clock in the morning when I first saw him by the uncertain flickering light of a hurricane lamp, with symptoms coming on five weeks after he had been bitten, and a high temperature, I thought that there was no reasonable doubt as to the diagnosis.

Especially as up to that time I had thought him to be a teetotaler and anything but an imaginative man. In this case"the man and dog both recovered from the bite, the ape it was that died."

Such then were the few experiments, that were attempted and I hope at a later date to have an opportunity of proving or disproving and of amplifying the conclusions to which one has arrived.
FINIS.

"Valeat quantum valere potest".
REFERENCES.
<table>
<thead>
<tr>
<th>Author</th>
<th>Title</th>
<th>Pages/Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>BAELZ</td>
<td>Handbuch der Tropenkrankheiten Vol 2 page 172-174 1905.</td>
<td></td>
</tr>
<tr>
<td>BENTLEY</td>
<td>Beri-beri &quot;Kakke gleanings from Japan&quot; Edin:</td>
<td></td>
</tr>
<tr>
<td>BRADDON</td>
<td>The Cause and Prevention of Beri-beri. 1907.</td>
<td></td>
</tr>
<tr>
<td>BROOKE</td>
<td>Tropical Medicine. page 132--143 1903.</td>
<td></td>
</tr>
<tr>
<td>DANGERFIELD</td>
<td>Le Béribéri</td>
<td>Paris 1905.</td>
</tr>
<tr>
<td>DANIELS</td>
<td>Laboratory Studies in Tropical Medicine 2nd Edit: London 1907.</td>
<td></td>
</tr>
<tr>
<td>GIBSON</td>
<td>Beri-beri</td>
<td>London 1904.</td>
</tr>
<tr>
<td>JEANSELME</td>
<td>Le Béribéri.</td>
<td>Paris 1908.</td>
</tr>
<tr>
<td>MANSON</td>
<td>Tropical Diseases. pages 356--383. 1907.</td>
<td></td>
</tr>
<tr>
<td>MIURA</td>
<td>Handbuch der Tropenkrankheiten vol 2 1905.</td>
<td></td>
</tr>
<tr>
<td>PEKELHARING &amp; WINKLER</td>
<td>Beri-beri</td>
<td>1904.</td>
</tr>
<tr>
<td>SCHAUBE</td>
<td>Die Krankheiten der Warmen Länder. page 252-254. 1900.</td>
<td></td>
</tr>
<tr>
<td>WINKLER</td>
<td>Pekelharing and Winkler. Beriberi 1904.</td>
<td></td>
</tr>
</tbody>
</table>
REFERENCE TO LITERATURE.

<table>
<thead>
<tr>
<th>Author</th>
<th>Title</th>
<th>Source</th>
<th>Page(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANDEL van</td>
<td>&quot;A contribution to the etiology and treatment of Beri-beri&quot;</td>
<td>Journ of Trop Med &amp; Hyg: No5 vol 12</td>
<td>63, 64</td>
</tr>
<tr>
<td>BERTRAND</td>
<td></td>
<td>Ann de l'Inst Past: t. 16 No3 (Aug 1903)</td>
<td></td>
</tr>
<tr>
<td>BIDDOES</td>
<td></td>
<td>Meeting of Soc Trop Med &amp; Hyg: B.M.J. May 29 1909</td>
<td>1802</td>
</tr>
<tr>
<td>BOURGUINGNON</td>
<td>&quot;Prophylaxie du Béribéri&quot;</td>
<td>Congress international d’Hygiène. Brux 1905</td>
<td></td>
</tr>
<tr>
<td>BUCHANAN</td>
<td></td>
<td>Lancet Aug 27 1898 vol 2</td>
<td>577</td>
</tr>
<tr>
<td>BULLMORE</td>
<td></td>
<td>Lancet Sept 23 1902</td>
<td></td>
</tr>
<tr>
<td>CLÉMENT</td>
<td></td>
<td>Comptes rendus de l'acad des Sci: No 18 1905</td>
<td></td>
</tr>
<tr>
<td>DANTEC le</td>
<td>1) Precis de path exotique.</td>
<td>Paris 1906</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2) Cong: colonial de Paris.</td>
<td>1906</td>
<td></td>
</tr>
<tr>
<td>DAUBLER</td>
<td>Archiv f Schiff's u Trop: Hyg:</td>
<td>page 375 1897</td>
<td></td>
</tr>
<tr>
<td>DÉCHAMBRE</td>
<td>D'apres les Arch: de Méd Nav: T 15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DIEREN van</td>
<td>&quot;Beriberi één rijstvergiftignng.&quot;</td>
<td>Amsterdam 1897</td>
<td></td>
</tr>
<tr>
<td>DURHAM</td>
<td>&quot;Notes on Beri-beri in the Malay Peninsular and on Christmas Island&quot;</td>
<td>Journ of Hyg Vol 14 1904 No 1 P 112--158</td>
<td></td>
</tr>
<tr>
<td>DYKES</td>
<td></td>
<td>Indian Medical Gazette. June 1904</td>
<td></td>
</tr>
<tr>
<td>EIJKMAN</td>
<td>&quot;Polyneuritis big Hoenders nieuwe bijdrage tot de etiologie der Ziekte&quot;</td>
<td>Batavia 1896</td>
<td></td>
</tr>
<tr>
<td>ELLIS</td>
<td>&quot;The etiology of Beri-beri&quot;</td>
<td>B.M.J. 1903 vol 2</td>
<td>1288</td>
</tr>
<tr>
<td>FEIBIG</td>
<td>&quot;Path: und Therap: der Beri-beri Krankheit&quot;</td>
<td>Geneskad Tijd: 1899</td>
<td></td>
</tr>
</tbody>
</table>

Portions relating to etiology translated for me from the original Dutch.
<table>
<thead>
<tr>
<th>Author</th>
<th>Title</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAGEN</td>
<td>&quot;Du béribéri à la Nouvelle Calédonie et quelques observations tendant à prouver son caractère contagieux&quot;</td>
<td>Rev; Médical de l'Est. 25 No 2 Jan 15 1893 p 42.</td>
</tr>
<tr>
<td></td>
<td>2) Philip: Jour: of Science.</td>
<td>vol 1 Sept 1906 No 7 page 709-761.</td>
</tr>
<tr>
<td>HICHTET</td>
<td>Annual Health Report for Siam.</td>
<td>No 12.</td>
</tr>
<tr>
<td>KOCH</td>
<td>&quot;Research into the Etiology of Beri-beri&quot;.</td>
<td>Hongkong Government Report. 1906.</td>
</tr>
<tr>
<td>LASNET</td>
<td>Lancet April 3 1897 vol 1 page 975.</td>
<td></td>
</tr>
<tr>
<td>LAURENT</td>
<td>&quot;Rôle de l'insuffisance en matières grasses de la ration alimentaire dans l'étologie du béribéri&quot;</td>
<td>Archiv de Méd Nav. vol 71 1899.</td>
</tr>
<tr>
<td>Author</td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>--------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Miura</td>
<td>&quot;Der diastalische Arterienten&quot; Zeit d Med Ges zu Tokyo Jan 1891.</td>
<td></td>
</tr>
<tr>
<td>Nocht</td>
<td>Festschrift du 60e anniversaire de Koch.</td>
<td></td>
</tr>
<tr>
<td>Ross</td>
<td>1)&quot;Peripheral Neuritis&quot; Med: Chronicle vol 7 1900.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2)&quot;The Premonitory Symptoms of Alcoholic Neuritis&quot;. Lancet 1889 vol 1 page 1125.</td>
<td></td>
</tr>
<tr>
<td>Saldanha</td>
<td>&quot;Anoteon the etiology of Beri-beri and the presence of Arsin in the rice&quot; B.M.J. Nov 23 1908 page 1609.</td>
<td></td>
</tr>
<tr>
<td>Sitta</td>
<td>Journ: de Medicine de Prague 1901.</td>
<td></td>
</tr>
<tr>
<td>Travers</td>
<td>1)&quot;The theory of the causation of Beri-beri by a toxin conveyed by rice considered in the light of local experience of the disease&quot; Journ: of Trop Med: Vol 5 1902 p 231.</td>
<td></td>
</tr>
<tr>
<td>Ucherman</td>
<td>Reference Lancet page 26 1902.</td>
<td></td>
</tr>
</tbody>
</table>
REFERENCES TO LITERATURE.

WRIGHT, HAMILTON

1). "An inquiry into the etiology and pathology of Beri-beri" 
   Studies from the Institute of Research F.M.S. 
   vol 12 No 1 1902.

2). "A case of acute cardiac beri-beri" 
   B.M.J. May 12 1906 page 1095-1097.

3). "The Cause, Course and Treatment of Beri-beri" 
   American Medicine Oct 1905."
COMPLETE

INDEX.
INDEX

A.

Acute cardiac form. 125.
Age, effect of- 86.
Age, period chart 99.
Alcoholic neuritis, 183.
Alkalinity of blood. 163.
Allbutt 238.
Analysis of rice 43.
Andel 69.
Ankylostomiasis 182.
Arguments for rice theory 64 et seq.
Arteriograph 173.
Arterio-rectus chart 177, 178.
Arsenical neuritis 183.
Arsenical poisoning, theory of- 14.
Arsenic in hair 31.
Arsine in rice 41.
Aron 60.
Aspiration of heart 224.
Atrophic form 130.

B.

Baer 6, 27, 136, 151, 190, 221.
Bentley 68.
Biddle 78.
Blood, bacteria in-
Bleed counts 21, 155-156, 245, 261.
Blood, charts of-
Blood pressure 145, 245, 155, 271.
Blood, charts of-
Bourguignon 16, 41 et seq., 65, 89.
Braddon 90, 102, 109, 151, 159.
Brazil, beri-beri in-
Bright's disease 181.
Buchanan 65.
Bullmore 197.
Burma, neuritis in- 180, 181.

C.

Cardiac lesions 143, 185.
Carnegie Brown 76, 77.
Cassava 79.
Cerebro-spinal fluid 22, 141, 238-234, 239.
285.
### INDEX

#### C.

- **Cerebral symptoms**
  - 141.
- **Chloride-free diet**
  - 221.
- **Choquan, epidemic in**
  - 165.
- **CHRISTMAS ISLAND, references to**
  - 4, 12, 20, 26, 57, 58, 61, 70, 75, 90, 107, 108, 166, 195, 196, 199, 200, 201.
  - 207-210, 212, 214.
- **Chu Choy, case of**
  - 270.
- **Circulatory symptoms**
  - 142, 221.
- **Clément**
  - 212.
- **Coagulability of the blood**
  - 154-156, 214.
- **Constipation, effect of**
  - 67.
- **Croom**
  - 212.
- **Coolie house, photograph of**
  - 202.

#### D.

- **Dangerfield**
  - 28.
- **Daniels**
  - 36, 107.
- **Dantec le**
  - 30, 182.
- **Death, modes of**
  - 126, 127.
- **Dechambre**
  - 190.
- **Diagnosis of Beri-beri**
  - 170 et seq.
- **Diaphragm, paralysis of**
  - 222.
- **Diarrhoea**
  - 221.
- **Dieren van**
  - 50.
- **Dietetic tables**
  - 207-210, 216.
- **Diet in hospital**
  - 210, 214.
- **Distribution**
  - 7.
- **Dropsica epidemica**
  - 182.
- **Dropsical forms**
  - 182.
  - = photograph of
  - 129.
- **Durham**
  - 34, 35, 79.
- **Dykes**
  - 102.
- **Dyspepsia**
  - 221.

#### E.

- **Becke van**
  - 131.
- **Bijlman**
  - 102.
- **Electrical reactions**
  - 102, 156.
- **Electricity in treatment**
  - 225, 226.
- **Ellis**
  - 66.
- **Epidemic Dropsy**
  - 74, 182.
## INDEX

### E.
- Ergotism .................................................. 194.
- Etymology ............................................... 6.
- Evolution ................................................ 195.
- Experiments with animals .............................. 22, 23, 26, 30, 33.
- .................................................. 35, 46, 255, 276.

### F.
- Fat deficiency ........................................... 37.
- Feibig ...................................................... 11, 73.
- Fiji, Beri-beri in- ..................................... 73.
- Fink ......................................................... 60, opp. 190.
- Formate ..................................................... 156, 218.
- Formic acid ................................................ 217, 218.
- Forms of Beri-beri ....................................... 111.
- .................................................. relative percentage 124.
- Fowls, experiments with ................................ 18, 30, 41, 50.
- .................................................. 58, 59, 276.
- Fraser ....................................................... 53, et seq. 90, 102.

### G.
- .................................................. photographs of- 117, 118.
- Gastro-intestinal symptoms ............................. 150.
- Gerrard ...................................................... 28, 102.
- Geyet ......................................................... 63.
- Glögnner ..................................................... 10, 173.
- Goodall ...................................................... 212.
- Grey .......................................................... 62.
- Grimm ......................................................... 38.
- Gulpke ......................................................... 38.

### H.
- Hagen ......................................................... 65.
- Hicceugh ................................................... 222.
- Higuet ....................................................... 63.
- Hirota ......................................................... 86, 126.
# INDEX

## H.
- Halst: 108.
- Hong Ming, case of: 104 et seq.
- Rose: 40.
- Hospital dietary: 210, 214.
- Hygiene: 200, 212.

## I.
- Incubation period: 101.
- = = tables of: 104-106.
- Indican in urine: 102, 107.
- = = plate showing: 109.
- Infantile form: 126.
- Integumentary symptoms: 158.
- Isolation experiment: 54.

## J.
- Janeway: 120.
- Java pea: 69, 205, 212.
- Jeanselme: 65, 72.
- "Jongkok" Test: 112.
- Joynt: 73.

## K.
- Kakkeococcus: 95, 81.
- Kokubo: 224, 158.
- Kleistermann: 172.
<table>
<thead>
<tr>
<th>INDEX</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>IND X.</td>
<td></td>
</tr>
<tr>
<td>L.</td>
<td></td>
</tr>
<tr>
<td>Lactic Acid Bacilli</td>
<td>221, 266-268.</td>
</tr>
<tr>
<td>Landry's paralysis</td>
<td>182.</td>
</tr>
<tr>
<td>Lasnet</td>
<td>102.</td>
</tr>
<tr>
<td>Lathyrism</td>
<td>164.</td>
</tr>
<tr>
<td>Laurent</td>
<td>37.</td>
</tr>
<tr>
<td>Lead poisoning</td>
<td>133.</td>
</tr>
<tr>
<td>Leprosy</td>
<td>185.</td>
</tr>
<tr>
<td>Leslie</td>
<td>89.</td>
</tr>
<tr>
<td>Limley, case of</td>
<td>228 et seq.</td>
</tr>
<tr>
<td>Lipaemia</td>
<td>153.</td>
</tr>
<tr>
<td>Locomotor Ataxia</td>
<td>153.</td>
</tr>
<tr>
<td>Locomotory symptoms</td>
<td>142.</td>
</tr>
<tr>
<td>Lok Ley, case of</td>
<td>230 et seq.</td>
</tr>
<tr>
<td>Lok Sun, case of</td>
<td>229 et seq.</td>
</tr>
<tr>
<td>M.</td>
<td></td>
</tr>
<tr>
<td>Macroscopic appearances</td>
<td>165-167.</td>
</tr>
<tr>
<td>Magnesia, value of</td>
<td>217.</td>
</tr>
<tr>
<td>Malarial neuritis</td>
<td>166.</td>
</tr>
<tr>
<td>Malcolmson</td>
<td>6.</td>
</tr>
<tr>
<td>Malingering</td>
<td>155.</td>
</tr>
<tr>
<td>Manila, epidemic in</td>
<td>66.</td>
</tr>
<tr>
<td>Manson</td>
<td>11, 17, 19, 223, 233.</td>
</tr>
<tr>
<td>Map</td>
<td>78.</td>
</tr>
<tr>
<td>Marapuana</td>
<td>226.</td>
</tr>
<tr>
<td>Massage, value of</td>
<td>217.</td>
</tr>
<tr>
<td>Maveral</td>
<td>17.</td>
</tr>
<tr>
<td>Metchnikoff</td>
<td>266.</td>
</tr>
<tr>
<td>Meteorological conditions, effects of</td>
<td>29.</td>
</tr>
<tr>
<td>Miasmic theory</td>
<td>11.</td>
</tr>
<tr>
<td>Microscopic appearances</td>
<td>166-178.</td>
</tr>
<tr>
<td>Miller</td>
<td>32, 159.</td>
</tr>
<tr>
<td>Mitchell</td>
<td>219.</td>
</tr>
<tr>
<td>Miura</td>
<td>38, 66, 97, 169.</td>
</tr>
<tr>
<td>Mixed form, photograph of</td>
<td>150.</td>
</tr>
<tr>
<td>Mohr's method</td>
<td>161.</td>
</tr>
<tr>
<td>Mortality returns</td>
<td>155.</td>
</tr>
<tr>
<td>Motor symptoms</td>
<td>154.</td>
</tr>
<tr>
<td>Muntok, epidemic in</td>
<td>71.</td>
</tr>
<tr>
<td>Muscular symptoms</td>
<td>225.</td>
</tr>
<tr>
<td>Mustard, methods of applying</td>
<td>222, 235.</td>
</tr>
<tr>
<td>Myelitis</td>
<td>184.</td>
</tr>
</tbody>
</table>
# INDEX

## N.

<table>
<thead>
<tr>
<th>Term</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nansen</td>
<td>138</td>
</tr>
<tr>
<td>Nationality, effect of</td>
<td>84</td>
</tr>
<tr>
<td>Nerve lesions, histology of</td>
<td>170-173</td>
</tr>
<tr>
<td>Nervous symptoms</td>
<td>131 et seq.</td>
</tr>
<tr>
<td>New Caledonia, epidemic in</td>
<td>83</td>
</tr>
<tr>
<td>Noght</td>
<td>187</td>
</tr>
<tr>
<td>Norwegian commission</td>
<td>187</td>
</tr>
</tbody>
</table>

## O.

<table>
<thead>
<tr>
<th>Term</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oedema, causation of</td>
<td>139</td>
</tr>
<tr>
<td>Oedema of Beri-beri</td>
<td>137 et seq 220</td>
</tr>
<tr>
<td>Okata</td>
<td>24</td>
</tr>
<tr>
<td>Ophthalmoscopic examination</td>
<td>232</td>
</tr>
<tr>
<td>Optic Atrophy</td>
<td>233</td>
</tr>
<tr>
<td>Oxalate poisoning theory</td>
<td>17,163</td>
</tr>
<tr>
<td>Oxygen inhalation</td>
<td>224</td>
</tr>
</tbody>
</table>

## P.

<table>
<thead>
<tr>
<th>Term</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Padi, photographs of</td>
<td>205</td>
</tr>
<tr>
<td>Paralysis of Diaphragm, chart of</td>
<td>247</td>
</tr>
<tr>
<td>Parrots, Beri-beri in</td>
<td>60 opp.</td>
</tr>
<tr>
<td>Paris, seige of</td>
<td>190</td>
</tr>
<tr>
<td>Pellagra</td>
<td>184, 192</td>
</tr>
<tr>
<td>Pekelharing</td>
<td>21, 23, 89, 108, 151, 172</td>
</tr>
<tr>
<td>Pernicious anaemia</td>
<td>193</td>
</tr>
<tr>
<td>Phosphorus theory</td>
<td>53 et seq.</td>
</tr>
<tr>
<td>Port Arthur, seige of</td>
<td>139</td>
</tr>
<tr>
<td>Potassium citrate, value of</td>
<td>220</td>
</tr>
<tr>
<td>Probonlinggo, epidemic in</td>
<td>62</td>
</tr>
<tr>
<td>Prognosis</td>
<td>195 et seq.</td>
</tr>
<tr>
<td>Progressive muscular atrophy</td>
<td>185</td>
</tr>
<tr>
<td>Prophylaxis</td>
<td>198 et seq.</td>
</tr>
<tr>
<td>Pulse in Beri-beri</td>
<td>144, 145</td>
</tr>
<tr>
<td>Pulse tracings</td>
<td>146-148, 256</td>
</tr>
<tr>
<td>Putnam</td>
<td>16</td>
</tr>
</tbody>
</table>
INDEX.

R.

Race, effect of 84.
Rainfall charts 92--98.
Relapses, indications of 196.
Respiratory symptoms 153, 224.
Rhombergism 135.
Rice dust 219.
= = photographs of 226.
Rice theories 42 et seq.
Rice varieties of 40 et seq.
= = photographs of 203, 204.
Rigor mortis 165.
Roll 16.
Ross 14.
Rudimentary form 112.

S.

Sago, as a cause 73, 74.
Saldahna 51.
Sanatogen 229.
Scheube 6, 37, 38, 111, 171, 195.
Scott South Pole expedition 188.
Scriba 62.
Scurvy 188.
Sensory symptoms 152-154.
Sex, effect of 94.
Ship Beri-Beri 52, 77, 82, 167--192.
Singapore Gaol, epidemic in 65.
Sitta 173.
Social conditions, effect of 83.
Spasmotic form 125.
Spastic paralysis 184.
Stanley 102.
Stanton 55 et seq 102.
St Helena, epidemic in 74.
Stomach contents, examination of 151.
Strachan's disease 185.
Summary of etiology 81, 82.
Syringomyelia 168.

T.

Tactile sense 108.
= = charts of 110, 241, 242, 263.
Takaki 36, 67.
Tapping value of 266.
Temperature chart 284.
## INDEX

**T.**
- Theories, table of: 9, 10.
- Tokyo, epidemic in: 62.
- Torup: 183.
- Toxil-infectious theory: 20 et seq.
- Travers: 102.
- Treatment: 212 et seq.
- = results of: 215.
- Trentlein: 17, 41, 163.
- Trophic changes, photograph of: 262.
- Tropical oedema: 199.
- = blood pressure in: 199.
- Tsuzuki: 31.
- Turner: 32, 159.

**U.**
- Usherman: 102.
- Urinary symptoms: 159--163.
- Urine, bacteria in: 31, 32.
- Urine, charts of: 274.

**V.**
- Venesection, value of: 223, 236.
- Voderman: 38, 62, 89, 102.
- Vomiting: 222.

**W.**
- Wrist-drop: 119.
- Women, beri-beri in: 67, 85, 86.
- Wellington: 71.
- Wright, Hamilton: 52, 53, 57, 72, 73, 101, 166, 172.

**X.**
- Yamagiva: 171.
VOLUME.
PATHOLOGY

of

BERI-BERI

"Scribendi recte sapere est principium et fons."

Horace.
MACROSCOPIC or POST-MORTEM APPEARANCES.

The condition of the body will vary according to the form and period of duration of the disease. In some cases the oedema is marked often accompanied by cyanosis, in others the body is pallid and emaciated. In acute cases there may be frothing at the mouth. Rigor Mortis comes on early and in cases where death has been sudden only lasts a short time.

On making the incision to open the abdomen one is struck:—

1). By the pale almost yellowish colour of the muscles
2). By the fluidity of the blood in the veins and its dark colour (saturated with Carbon dioxide).

On account of this condition of the blood hypostatic congestion occurs early.

3). In oedematous cases the presence of serous fluid in the cellular tissue.

Punctiform haemorrhages may be noticed over the visceral surface of the peritoneum, pericardium and pleurae, and effusion into these cavities is very common, especially into the pericardium. A certain degree of pericardial effusion is found in 95% of cases, but as a rule not enough to have caused death from mechanical means, but it may be regarded as one of the constant pathological signs of this disease.

ABDOMINAL VISCERA.

The Liver is often somewhat enlarged and congested, fatty degeneration may be present and it may have the typical nut-meg appearance.
The Gall Bladder contains fluid bile.
The Spleen is congested and frequently enlarged, this is probably of Malarial origin, for in Christmas Island a non-malarial locality there was no marked increase in the size of this organ.

The Stomach is as a rule dilated, its walls are oedematous and congested and at the tops of the folds haemorrhagic erosions may be seen. In acute cases a gastro-duodenitis is generally present, this point was particularly emphasized by Hamilton Wright, as being a constant pathological change in Beri-Beri and the portal of entrance of the specific virus of the disease. But while great hyperaemia is present in the acute fulminating cases, it is not a conspicuous symptom of those in whom the disease has been of some standing, and the condition when found is probably due to the general venous congestion.

In the Intestines there is little, the follicles may be swelled and small haemorrhages may be found. Intestinal parasites are frequently seen, but have no etiological significance.

The Kidneys are as a rule quite healthy, and the capsule strips readily, punctiform haemorrhages may be seen here as else-where and cloudy swelling may be present.

**THORACIC VISCERA.**

Lungs are usually pale, and frequently there is oedema and congestion at the bases. As a rule the lungs do not contain much air, but occasionally are markedly emphysematous. Here and there a patch of deep colour will point to an area of pulmonary apoplexy.
HEART. The pericardium constantly contains fluid varying in amount. The organ itself is always enlarged especially on the right side. The state of the myocardium will depend on whether the heart is hypertrophied or dilated. In the former cases the heart walls are reddish brown and firm, while in the latter they are pale and friable. Clots are often found on the right side passing into the pulmonary artery. In atrophic cases in which the death struggle has been protracted "chicken-fat" clots are often seen. The valves in advanced cases are not "tight", especially so is this the case with the Tricuspid, but there is no disease of the endocardium. The coronary vessels are much dilated. The changes in the heart are degenerative and not inflammatory.

BLOOD VESSELS. Arterial lesions are inconstant, occasionally there is a disintegration of the elastic network of the aorta.

NERVOUS SYSTEM. To the naked eye there is little change to be made out in the nerves.

VOLUNTARY MUSCLES. Marked atrophy even in those that appear large from the swelling, this being due to the infiltration of serum in the inter-muscular cellular tissue. The muscles when atrophied are yellowish in colour, when oedematous they present what has been termed a "marbled" appearance, yellow lines as a network throughout the redder flesh.
Pathology

MICROSCOPIC or HISTOLOGICAL APPEARANCES.

HEART

The cardiac muscle fibres may be affected to all degrees, the right ventricle shows the changes best, and in the same section one may see all degrees of degeneration; alongside healthy fibres one finds some showing fatty degeneration; others in which transverse striation is lost and others again with marked granular degeneration and fragmentation. In very advanced cases all the normal elements may be lost, the fibres are frequently seen to be vacuolated and sometimes the interstitial tissue is increased.

Dürrck reported, that in cases examined very shortly after death, acute changes in the myocardium were evidenced by the swelling of the muscle cells, they seem to be infiltrated by a homogenous vitreous mass in the form of bands, which blended insensibly with the striated protoplasm of the cardiac fibres. (see micro-photograph).

LUNGS

Unless congestion and oedema are present, these do not show any changes; nor is there any
There is nothing characteristic about the Spleen.

LIVER

Shows fatty degeneration and cloudy swelling, and there is congestion of the intra-lobular capillaries. Plahn has described what he terms "the interstitial hepatitis of Beri-beri," which consists in an infiltration of the interlobular fibrous connective tissue with small round cells.
KIDNEYS

These show cloudy swelling, but little other change. Miura found glomerular nephritis, but this is so uncommon, that it cannot be considered the cause of the anasarca of the disease.

MUSCLES

In the case of dry atrophic muscles, the fibres will be seen to be thin and to have lost their striation, this is one of the earliest changes. On cross section the characteristic polyhedral form will be seen to have become round or oval. Healthy and diseased fibres will be found to be lying side by side. Later there is a want of definition of the individual fibres and eventually they may resolve into a sort of granular mass, the sarcoplasm shrunken away from the sarcolemma.

In the case of dropsical muscles, the presence of the oedema enables one to dissect out the fibres with ease. These fibres are specially liable to develop colloid or serous degeneration, the sarcoplasm is swollen within the sarcolemma and under the microscope appears white, reflecting direct light more strongly than the normal fibre. The nuclei of the cells are increased, and there is decided proliferation of the interstitial tissue.

NERVES.

Here lies the principle changes in this disease, and to the nerve lesions can be traced all the symptoms, the paralysis, the cardiac symptoms, the fall of blood pressure.
pressure, the diminished urine and all the other phenomena that go to make up the disease that is known as Beri-beri.

The histo-pathological appearances necessarily vary as greatly as the lesions produced. In one section every form of degeneration may be seen and the changes that occur are those of a typical parenchymatous neuritis; true Walerian degeneration. The myelin sheath suffers first, the changes may be quite insignificant or very profound.

On longitudinal section the sheath can be seen to have broken up into droplets or beads, finally it disappears. When the degeneration of the myelin sheath is advanced, the axis cylinder suffers, it first gets distorted, then irregular and eventually can only be seen in the neighbourhood of the Nodes of Ranvier; later it disappears entirely.

The nuclei of Schwann's Sheath undergo karyokinetic proliferation.

Thus anything may be found from a simple swelling of the medullary substance with thickening and displacement of the axis cylinder to complete destruction leaving only an empty sheath of Schwann, studded with newformed nuclei.

On transverse section in advanced cases some of the cells are seen to be still normal, others still retain their axis cylinders, others again contain homogenous masses composed of myelin detritus, whilst finally others are represented by mere empty spaces.

(see microphotographs).
The connective tissue also increases with multiplication of the nuclei and their is some thickening also of the blood vessels.

In chronic cases there is sclerosis of the endoneurium and wrinkled bands stretch across the section. On account of these changes Scheube and Baelz conclude, that the process is not a simple degeneration, but that it is inflammatory. Yamagiva who did extensive work on this subject for Miura, upholds the original view of Pekelharing and Winkler, that the changes are those of simple degeneration.

If on cross section the nerve shows the presence of a number of fine fibres, it is said to be indubitable evidence of degeneration. The nearer one approaches the periphery the more advanced are the changes especially in the terminal twigs, as one gets up to the Spinal cord the changes are not so marked, so that it is difficult to be certain of any alteration in nerve Roots: the anterior Root is said by some never to be affected and the posterior only slightly.

It seems to me that clinically there are grounds for believing in the involvement of Posterior Roots in advanced cases, for acute girdle-like pains may be complained of and on examination the Cerabro-Spinal fluid shows decided increase in the Lymphocytes, which may be taken as a proof that a Radiculitis has occurred.

In the case of such important nerves, as the Vagi and Recurrent Laryngeals, advanced degeneration is
not seen. This is readily understood when one remembers how essential they are to the general economy. Hence in the transverse section of these nerves one seldom sees more than a few fibres filled with granular detritus.

In order of sequence the Peroneal, Tibial and Saphena nerves are first affected.

Pathologists differ as to whether the Spinal Cord is affected at all. Pekelharing and Winkler held that it was not, Wright on the other hand found "hazy cells with swollen nuclei and slight chromatolysis in the ganglion cells of the Cord and Bulb."

Küstermann found acute degeneration of the ganglia at the base of the 4th Ventricle.

Sitta found degeneration of the Cauda Equina in 5 subjects, who had died of Beri-beri.

Lesions of the cord are probably rare: the following have been described:

(1). Proliferation of the cells of the Ependyma and infiltration of nuclei the circumference.

(2). Atrophy or loss of Motor cells of the Anterior Cornua.

(3). Ascending degeneration of the Posterior Columns, in connection with atrophy of corresponding Sensory Roots.

(4). Vacuolation of Motor Cells of the Anterior Cornua.

(5). Accumulation of waxy bodies in the white substance of the cord.
It seems, therefore to me from clinical reasons as well as from pathological findings, that the primary degeneration is in the nerves, but that it must not be limited too narrowly to the peripheral part alone. While the periphery is by far the most frequent part to be diseased, yet the "infection" may be more extensive, and then the posterior nerve roots and even the cord itself may be implicated.

The nervous theory is admitted now by everyone, and so need not do more than mention the view of Clöünger who considered, that the disease was not a polyneuritis but a polymyositis. (Muskelerbruchkrankheit).

Such then in brief are the characteristics of the macroscopic and microscopic pathological changes met with in Beri-beri.
Section of Sciatic Nerve from advanced case (x250)

Consists almost entirely of vacuoles left by the degenerated nerves, here and there is a homogenous mass and a few black dots indicate an axis cylinder that has not altogether broken up. Thickening of the vessel walls with infiltration of nuclei.

Section of Optic Nerve. (x260)

Whole section consists of fibres in an advanced state of degeneration, the dark spots indicate those in which the condition is earlier. Vessels and interfascicular space thickened and infiltrated.
ACUTE BERI-BERI.

Longitudinal Section of Popliteal Nerve (x200)
Degeneration of Myelin Sheath.

(micro-photograph)

ACUTE BERI-BERI.

Longitudinal Section of Popliteal Nerve (x250).
Shows fragellation of Myelin Sheath with characteristic bead formation.

(micro-photograph)
SUB-ACUTE "DRY" BERI-BERI

Longitudinal Section of Popliteal Nerve. (×550).
Marked degeneration of Myelin sheath,
(micro-photograph)

SUB-ACUTE BERI-BERI

Transverse Section of Peroneal Nerve with Nerve Sheaths.
Considerable loss of nerve fibres, (A.A)= intact fibres
(B.B)= blood vessels, (C)= Nerve sheaths.

(Photograph from drawing after Pekelharing)
SUB-ACUTE BERI-BERI.

Transverse Section of Branch of Anterior Crural Nerve. Most of the nerve fibres have been destroyed, leaving empty spaces surrounded by solid interstitial tissue. A few of the fibres are replaced by a swollen granular mass (A.A) and a few (coloured red) are still intact. (Photograph from drawing after Pekelharing)

SUB-ACUTE "WET" BERI-BERI.

Section of Myocardium, (×500.) Shows clearly the marked degeneration of the cardiac muscle fibres with fragmentation and segmentation. (Micro-photograph)
DIAGNOSIS.

and

DIFFERENTIAL DIAGNOSIS.

"Non enim tam auctoritatis in disputando, quam rationis momenta quaerenda sunt."

Cicero.
The Diagnosis of Beri-beri does not rest upon any one symptom, but on a number of clinical signs. It is not as a rule difficult, but the rudimentary forms are apt to give the most trouble. It is a matter of importance, that the diagnosis be made early. In the fully developed cases, whether atrophic or oedematous, the picture is too typical to be mistaken. From one's own observation in the Federated Malay States and elsewhere there is far too great a tendency to label almost everything as Beri-beri, cases which are suffering from Climatic Oedema, Malarial Neuritis, Anaemia, Ankylostomiasis and General Debility. This is especially the case in native managed hospitals.

I think then that the following can be regarded as the essential early signs of the disease, upon which diagnosis can be based.

1). Puffiness of the face.
2). Slight pretibial oedema.
3). Slight paraesthesia over the outer side of the leg.
4). Slight hyperaesthesia of the calves on firm pressure.
5). Easily accelerated cardiac action.
6). Slight epigastric fulness.

These signs are nearly always all present and taken together may be said to be pathognomonic of the disease.

DIFFERENTIAL DIAGNOSIS: After eliminating the aetiological factors, that the following in a fairly con-
DIFFERENTIAL DIAGNOSIS.

I think, that the following is a fairly comprehensive list of the possible diseases, which might give rise to any confusion in the diagnosis of Beri-beri.

I think, that those considered first are by far the most important and apt to be the most misleading, while those towards the end should seldom give rise to any difficulty.

A. MALARIAL NEURITIS.

In Burma, as Fink has shown, and in many other places where Malaria is rife, a Peripheral Neuritis of undoubted Malarial origin occurs.

In Burma this is known locally as "Htone Na" and shows itself in two forms "So" or wet and "Chauk" or dry.

At first sight these suggest at once to the observer the two chief types of Beri-beri. In all the cases there is a strong antecedent Malarial history, and the spleens are enlarged.

Further, there may be no fever at the time of the onset of paralytic symptoms and owing to the weakness of the extensor muscles, the foot-drop, wrist-drop, and "squatting" tests are all positive. There is anaesthesia varying in amount, loss of Knee jerks, hyperaesthesia of the calves of the legs and muscular wasting are all present.

On the other hand if there is any oedema it is always very slight, there are no gastric symptoms and no CARDIAC SYMPTOMS, nor is there any dyspnoea.
In other words the vagus nerve is not affected; this is of the greatest differential diagnostic importance, because of the similarity otherwise in the symptoms.

In Fink's cases the patients were, and had been for some time previously, eating freshly husked rice. As a further aid in the diagnosis, in some cases blood examination will prove of value, this is not of course of paramount importance, for in the malarial cases it may be negative, whilst in cases of Beri-beri, if the patient has Malaria as well, it may be positive.

Then lastly there is the all important therapeutic test, for the malarial cases as a rule clear up rapidly with the exhibition of quinine, the Beri-beri cases do not.

So important is the differential diagnosis between these two diseases and so often are they mistaken, that I have no hesitation in saying, that many of the cases diagnosed as Beri-beri may be attributed to previous attacks of Malaria.

The whole point of the matter lies in this, that in one form of neuritis the Vagus is affected in the other it is not. Therefore it is of importance to remember the course of this nerve, composed as it is of both Motor and Sensory Fibres, and supplying the larynx, pharynx, lungs, oesophagus, stomach and heart.

One note of warning I should strike and that is the possibility, that in the gastric form of remittent malaria the end fibres of the vagus to the gastric mucosa may be irritated and so cause obstinate vomiting, but evenn
Diagnosis——

the many other signs of Vagus affection so typical of Beri-beri will be wanting.

E. EPIDEMIC DROPSY.

There is marked oedema and frequently diarrhoea and vomiting. Nearly always there is a considerable initial pyrexia. There is never any paralysis, the knee-jerks are present and there is no anaesthesia or paraesthesia of the skin and no muscular hyperaesthesia.

C. ANKYLOSTOMIASIS.

In this disease the onset is insidious, though it is sometimes ushered in with a swinging temperature. The anaemia is progressive and it is on account of the blood condition, that any similarity between this and Beri-beri exists. The appetite is perverted, there is no paralysis, anaesthesia or hyperaesthesia. As a rule the ova will be found in the stools, but I should mention, that in the latter stages of the disease parasites may not be found and yet symptoms remain, but the blood in these cases always shows a marked eosinophilia.

D. LANDRY'S PARALYSIS.

This disease can be very like an acute attack of Beri-beri in many of its symptoms. But there is no oedema, no wasting of the muscles and sensation is rarely lost. Complete paralysis occurs in a few hours, and there is as well Fever, Splenic enlargement, Albuminuria and Skin eruptions.
E. ALCOHOLIC NEURITIS.

In this case there will be the previous alcoholic history, the tremulousness, the digestive disorders, and the albumen in the urine.

F. ARSENICAL NEURITIS.

The question of the analogy between Arsenic and Beri-beri has already been discussed when considering the etiology of the disease. Again there is the history to guide one, the marked diarrhoea, the abdominal pain, blood in the urine, discoloration of the skin and the absence of oedema.

G. LEAD POISONING.

Here one has the classical symptoms to guide one, the pains in the joints, the severe abdominal colic, the wrist-drop without any corresponding foot-drop, the blue line on the gums, and the absence of pain in the muscles.

H. LOCOMOTOR ATAXIA.

The girdle pains of this disease are very characteristic, there is probably a very definite history to aid one, the course of the symptoms, the anaesthesia of the soles of the feet and the presence of the Argyll-Robertson pupil will all make the diagnosis easy.

I. BRIGHT'S DISEASE.

There is really not much similarity between these two diseases, a mistake might arise in a very oedematous case, but in Bright's there is no loss of sensation, no hyperaesthesia, no loss of knee jerks and there is albumen in the urine.
Diagnosis.

J. PELLAGRA.

History of eating maize, (I am not contending that this is the cause of this disease), but apart from the history there are very definite differences the knee-jerks are increased, marked tremors are present and often tetany, there is distinct inco-ordination, and there is no tenderness of the muscles and the other cardinal symptoms of Beri-beri are also absent.

K. ERGOTISM.

Again the history will be of help, the consumption of rye; as regards the symptoms besides the many negative points, there is the tingling of the skin and the typical gangrene.

L. LATHYRISM.

In this case there is a history of eating pulse, then again the knee-jerks are increased, and negatively there is no tenderness, no inco-ordination, no paralysis, no anaesthesia.

M. MYELITIS.

The rapid onset is characteristic, bladder troubles are prominent with incontinence of urine, there is no pain or tenderness in the muscles.

N. SPASTIC PARAPLEGIA.

Here the picture is very different, there is the slow onset, the characteristic stiffness, the great increase of the knee-jerks, and the absence of tenderness, of oedema, and of muscular atrophy.
O. PROGRESSIVE MUSCULAR ATROPHY. (Aran-Duchenne)

There is really not very much similarity between these two diseases. The Progressive Muscular Atrophy takes years to form, there is an absence of hyperaesthesia in the muscles and no anaesthesia.

P. SYRINGOMYELIA.

The onset is very slow, the knee-jerks are increased, there may be scoliosis and there is the characteristic thermo-anaesthesia.

Q. STRACHAN'S DISEASE.

In the acute form of Beri-beri the symptoms are somewhat similar to those of Strachan's disease, but in the latter there is no oedema, no paraesthesia, no acute cardiac crisis, and the paresis passes on to atrophy.

R. LEPROSY.

The disease is similar in that there are areas of anaesthesia, but there is little other similarity, the slow progress is different, the skin and trophic changes, the perforations, the bone reabsorption. (In very rare cases bone reabsorption has been seen in Beri-beri?).

S. CARDIAC DISEASE.

There may be oedema, but it is of the feet, there will be murmurs over the cardiac areas, albumen in the urine and no nervous symptoms, no paralysis, no anaesthesia.
T. PERNICIOUS ANAEMIA.

As already stated in advanced cases of Beri-beri the blood findings may be very like those of Pernicious Anaemia, but in the latter disease there are none of the other symptoms of Beri-beri, no paralysis, no anaesthesia, no true muscular hyperaesthesia.

The only other possibilities, that one can think of need only be mentioned, namely Trichina and possibly Spinal Meningitis.

MALINGERING.

Apart from recognized diseases, I think that the possibility of malingering should be held in mind. In areas where the disease prevails, it is no very uncommon thing to find coolies attempting to get off work on the plea of Beri-beri, they know many of the cardinal symptoms and induce cardiac irregularities by the use of drugs. Such cases can be most suggestive of Beri-beri, especially if examined hurriedly, but a careful inquiry into the nervous system, with examination of the knee jerks and the alterations of sensation will overcome any difficulty so long as the possibility is held in mind.

Briefly then these are the possible diseases, which might be mistaken for Beri-beri in one or other of its phases. I have not gone into this matter in any detail, but I think the points mentioned are sufficient to show the main distinctions, and before passing on to deal with the treatment one would like to briefly discuss the question of Ship Beri-beri.
NOTES ON SHIP BERI-BERI.

I have not included either this or Asylum Beri-beri amongst the differential list owing to the controversy, that still exists on the subject. The opinion of medical men differ very much, as to whether Ship Beri-beri is to be regarded as true Beri-beri or not.

Le Dantec in a paper published in 1905 referred to an outbreak as Nautical Beri-beri, drawing a distinction between it and the ordinary form. Bullmore from cases seen at Falmouth also did not consider the disease to be true Beri-beri, from his observations he further held, that it was neither infectious nor due to rice, but to general digestive disturbances caused by long sea voyages on a poor dietary. In cases seen by Nocht at Hamburg the morbidity was as high as 62.5% and the mortality 15%.

A commission was appointed in Norway to go throughly into this matter, and as a result of their investigations they came to the conclusion, that two forms existed.

1). Of vegetable origin caused by the eating of faulty rice.

2). Of animal origin caused by the eating of bad stale meat and fish.

As a result certain prophylactic measures were advocated including Government inspection of food and the limitation of certain necessary articles, thus compelling more frequent re-provisioning.
Nansen and Holst considered Ship Beri-beri to be a form of Scurvy. It occurs practically only on sailing vessels engaged in long voyages during which fresh food is unavailable. Symptoms typical of Scurvy and common to Ship Beri-beri are haemorrhages, loosened teeth, pain when the muscles are grasped, loss of reflexes and even anaesthesia. Holst considers that it is due to eating food deprived of its antiscorbutic properties by boiling and drying.

It is fairly generally recognized that the taking of Lime Juice prevents true Scurvy, but it does not always prevent Ship Beri-beri, as was shown by the Norwegian Beri-beri committee.

Professor Torup's theory is that scurvy is a chronic poisoning due to the decomposition of food. Dried foods allowed to get wet are apt to generate Scurvy, dogs are very susceptible to it as was seen in Scott's South Pole expedition, for the dogs, that were fed on dried fish allowed to get wet always developed the disease; so also Nansen himself developed it from this cause. In the same way on board ship food from damaged or rusty tins is far more liable to cause it. Norwegian ships are especially prone to it as they purchase tinned food condemned by the London inspectors and have no regulations regarding the taking of Lime Juice as in the British service. Italian ships are on the other hand very free from it and this is undoubtedly explained by the fact, that their dietary is very largely supplemented by onions and wine.
In the Journal of the American Medical Association (May 30, 1906) a case was reported of "Beri-beri" occurring without a definite rice factor in a Dutch ship, which came into San Francisco with the crew suffering from this disease. The point stress was laid on was that there was no rice on board either as cargo or as food. The conclusion, that was arrived at was, "that other vegetables besides rice may be factors in the production of Beri-beri". This is not to be denied and one has already attempted to show, that it may be true of Sago.

But this is the point, that I would lay most emphasis on,—the sole diet of the crew in this vessel consisted of ship's bread, salt meat and dried beans, which were stated to be mouldy. Thus the probability here, as in all these cases of Beri-beri on ships, is very strong, that the disease in question had a very large element of Scurvy in it.

Epidemics of so-called Ship Beri-beri are often due to Tropical Oedema from lowered blood pressure. A case of this came before my notice at Christmas Island. A cargo vessel came to load phosphate, and the captain sent a message to say, that a number of the crew were suffering from Beri-beri. The crew were principally Australians, the food on board was not very first class and there was a great lack of fresh vegetables. The men on examination displayed oedema, which in some cases was very considerable, and most of them had refused to work.
Besides the oedema there was some pain on pressure over the muscles, there was however no anaesthesia and no cardiac affection, the urine was free from albumen. The Blood Pressure was lowered in every case, in some to below 100 mm. of Hg. There may have been a certain scorbutic element present, but they were not suffering from Beri-beri.

Warm weather produces vaso-motor dilatation from diminished tone of the vaso-constrictors and thus tends to the development of oedema in the dependant parts. It has been clearly shown, that the blood vessels of the skin and those of the abdominal viscera frequently act in direct antagonism to one another (Janeway). Thus the dilatation of the peripheral vessels is not always accompanied by a fall in the blood pressure, but when balance does not occur, there is a fall in the blood pressure with congestion of the viscera and transudation of fluid. This is so frequently seen in the Oedema of the Tropics.

In most of the notable sieges of history a condition resembling Ship Beri-beri has occurred amongst the besieged. Dechambre has shown how a disease resembling Beri-beri was rife during the siege of Paris, whilst Baelz reported the same in the case of Port Arthur.

It seems therefore, that so-called Ship's Beri-beri may frequently be but Tropical Oedema, whilst on the other hand the cases which are seen on vessels arriving in temperate climates, whilst they may have a Beri-beri
element present, are largely complicated by Scorbutus.

Consider for a moment the surroundings, the damp almost hermetically sealed, badly ventilated fo'castle full of perspiring Lascars and at once one has a combination of many of the predisposing causes, and add to this a dietary deficient in Phosphorus, (whatever form that dietary may take) and with the further addition of a food deficient in anti-scorbutic properties. Is there any wonder, that mixed symptoms arise? Some of which point to Beri-beri and others to Scurvy, for in addition to the oedema, hyperaesthesia, and para-aesthesia of Beri-beri, there are many of the typical evidences of Scurvy the spongy gums, the loosened teeth, and the subcutaneous haemorrhages.

On the other hand, the reviewing of a large number of reports of so-called Ship Beri-beri brings out this important point, that in a great number of cases the symptoms do not correspond to the typical picture of Beri-beri at all. While oedema is a prominent symptom it is unaccompanied by atrophy of the muscles or any evidence even of paresis. In fact most of the symptoms in these cases too point to a pseudo-scurvy, the additional feature being caused either by a form of chronic ptomaine poisoning or else deficiency of phosphorus.

In every case a decided inferiority of the food supply can be proved.

One must not be supposed to infer from this, that true Beri-beri never occurs at sea for it undoubtedly does.
But in these cases there is a definite rice factor and the symptoms are those of the disease that has been described throughout this paper (and it is not by any means common to find authentic cases) the disease described as Ship-Beri-beri is not the true disease.

NOTES ON ASYLUM BERI-BERI.

Reports of an outbreak of a disease analogous to Beri-beri occurring in different asylums for the insane, have been published from time to time.

While the disease resembles true Beri-beri in many particulars, yet the picture presented is not that of the disease as seen in the Tropics.

From reading the accounts of such, one is struck briefly by the following points.

The oedema was not the same for it is hard and brawny in consistence, and does not pit on pressure.

Paralytic symptoms only shewed themselves in one third of the cases. There was no atrophy of the gastrocnemii. The bladder and rectum were early affected and often seriously so. The skin of the hands and face became brown suggesting Pellagra, and there were patches of Erythema and early trophic troubles, bed-sores, purpura and the like.

Further at the post-mortem examinations, besides the nerve degeneration, there were distinct changes in the cord, the large motor cells in the Anterior Cornua were swelled and were in a state of chromatolysis, the protoplasms were vacuolated and the nuclei eccentric.
Diagnosis---

Finally motile organisms were isolated from the Liver, Spleen, and Cerebro-spinal Fluid, which resembled Proteus Vulgaris.

How far this organism was the cause of the disease I am not in a position to state, but from the symptoms displayed the disease was not true Beri-beri. There were many things, that pointed to the possibility that the Beri-beri element was present, the over-crowding, the deficient hygiene, the faulty dietary. But here as in the case of Ship Beri-beri something else was undoubtedly superimposed either of a Scorbutic or Infective nature.
PROGNOSIS AND EVOLUTION

of

BERI-BERI.

"Vir sapit qui paucha loquitur".

SENECA.
Prognosis

E V O L U T I O N A N D P R O G N O S I S.

The commonest complications of Beri-beri are Tuberculosis, Dysentery and Malaria. Although most cases recover generally there is a lot of subsequent weakness and anaemia, with an absence of the knee-jerk often for a long period after apparent "cure" has taken place.

The prognosis is as a whole good, but it varies widely in different epidemics, and owing to the fact, that acute cardiac symptoms may supervene at any period, it must always be extremely guarded.

Amongst the unfavorable symptoms, which must be noted are: -- Vomiting, marked dilatation of the heart, and irregularity of the pulse.

The mortality returns vary exceedingly. In Sumatra at one time amongst the Chinese coolies it was as high as 60 or 70 %, and in Hongkong, taking the average over a number of years, it reaches the figure of 49·5 % in Males and 35·4 % in Females. On the other hand amongst the troops in the Dutch Indies it was only from 2 to 6 %, and according to Scheule in Japan it was 3·5 %.

In the Russo-Japanese War it was 2 % and at Christmas Island the average over a period of 9 years was 8·5 %, (the highest being 33·7 % in 1901 and the lowest 1·2 % in 1909).

The duration of illness also varies, some cases are very prolonged, others developing acute symptoms die suddenly.
Prognosis——

The average in the Tung Wah Hospital, Hongkong, is 47 days, on Christmas Island it was 43 and under a form of treatment to be described it fell to 36 days. Relapses are frequent and points, that call for extra care are:

1. Non-return of the knee-jerk.
2. A persistent paresis.
3. Fatigue on the slightest extra exertion.
4. Localized areas of hyperaesthesia.
5. Slight pre-tibial oedema.
6. Loss or feebleness of the sexual appetite.
7. Acceleration of the pulse on the slightest exertion, a violent apex beat with a feeble radial impulse.

N.B. This is of the greatest importance and so too is the next point.

8. A persistent diminution in the amount of urine excreted, almost oliguria.

In a large percentage of cases the ultimate usefulness of the coolie is not impaired, a certain percentage on account of cardiac trouble are only fit for light work, whilst some, who on account of a protracted attack, have developed deformities talipes etc, are not much use to the employer at all, although even in these good results may be obtained by tenotomy of the Tendo Achilles or with fixation of the ankle joint.
PROPHYLAXIS.

OF

BERI-BERI.

"Principis obsta, sert medicina paratur
Cum mala per longas convaluerer moras."

OVID.
After all that has been written, there can be little doubt, that the form of Rice in daily use is the one paramount all-important point in connection with the prophylaxis of this disease. It is no exaggeration to say, that if "chred" rice be employed the disease can be prevented from occurring. All that is required is a rice, that is not highly polished, which therefore retains a great deal of its pericarp, and so yields a generous supply of Phosphorus. I have endeavoured by the citation of a number of cases from a variety of sources, to prove the incontrovertability of this statement.

Sooner or later the big rice mills of Rangoon, Siam and elsewhere will realize that, though a pure white rice is still demanded by the European market, yet they must produce cheaply for the contractors' coolie a rice, which though it may have a dirty appearance when cooked, retains the all-important property so essential for health.

Large sea ports would do well to prohibit the importation of a rice, which falls short of the specified requirements, by this means alone can the disease, which annually forms such a large percentage of hospital admissions, and in Hongkong such a large mortality, be exterminated.

When one considers the crippling of work in big estates and in mines, which Beri-beri causes by its devastation of the Chinese labour, one wonders how long employers will continue to use a rice, which is
so toxic, when so easily they could at least give the other form a trial.

Coupled with the use of "cured" rice one would advocate a liberal and varied diet. I append a few lists to show the different articles of food in Christmas Island, with their respective Calorific Values and Energy producing properties.

None of these articles will cause much additional expense, the coolies will be fed better than ever before in their lives, and one will have the great satisfaction of watching the under-developed man, who arrives, becoming month by month a strong, sturdy and efficient coolie.

This is not the picture of a fancied Utopia, for on Christmas Island I used to take regular chest measurements of the coolies as they arrived, and it was surprising to see the change, that occurred. There was no doubt, that the power to do work was more than doubled, and in a very short time the original extra cost was repaid ten-fold.

I have already mentioned, that in Christmas Island there were certain difficulties in the introduction of the sole use of "cured" rice, personally I do not think it would have taken a great effort to overcome these difficulties, but even with the partial addition of "cured" rice to the daily dietary the number of cases of the disease fell progressively each year.

There is one point, which I have omitted to touch on and that is, that some employers have found
that the use of a rice, which retains practically all
its pericarp, is apt to set up a rather irritable form
of diarrhoea. It is not necessary to use such an
irritating form, all that is required, is a grain, which
is not highly polished and still retains \(\cdot 4\% \) of \(P_2O_5\),
such a rice never sets up diarrhoea.

Apart from the dietary the general hygiene
must be kept up to the standard.

The coolie houses should be well built, raised
from the ground and thoroughly dry. The roof should
be prolonged out to protect from the sun and the rain
and keep the verandah dry.
The houses should be so built, that with all windows
closed there is still an abundant circulation of fresh
air and should if possible face the prevailing wind.
I append a photograph of one of the latest type of
coolie houses now being built on Christmas Island, and
all these points have been attended to. As will be seen
it is built on slopping concrete, which drains into
an open gutter, which in turn passes directly into the
sea.

The houses should be regularly washed out with
some form of disinfecting fluid, this precaution will
certainly do no harm and will often prove most bene-
ficial. At Christmas Island the method employed was
to start at one end of the coolie lines and do 3 houses
throughly each day, until all were completed and then
to start from the begining again.
Efficient drainage must be provided and proper methods taken for the destruction and removal of night-soil. In Christmas Island the coolie lines, which were built at the top of the hill some distance from the sea, were provided with a most efficient home-made incinerator, which was cheap and worked admirably.

A good and abundant water supply should be provided.

Thus by careful attention to the form of dietary in use, coupled with ordinary hygienic precautions, the prophylaxis of the disease will be assured.

In the case of Ships the same points regarding food, sanitation and disinfection hold good.

I append a few photographs to show the different forms of rice. So much has been said throughout this paper about rice, that it seemed to be of interest to show as clearly as a photograph can the naked-eye differences. In the case of the Indian rice the branny dark coloured pericarp can be seen and compared with the white look of the Rangoon or Siam rice.
Photograph of most recent type of Coolie House as erected at Christmas Island.

This may be taken as almost a model for this type of building.

Built on concrete, sloped so as to drain readily into a gutter in front of the building and so into the sea. Raised well above the ground and so easily kept clean, thoroughly ventilated having 9 windows, and an air space of 2 feet at the bottom all round and 3 feet at the top, so that with all the windows closed there was still an abundance of pure air in circulation.

Covered with corrugated iron, which proved much more efficient in stormy weather than the original "attap" roofing and extended so as to keep the verandah dry as well. The extra heat from this form of roofing material as houses not occupied during the day, but could be easily met with by covering the surface with a layer of attap or a composition of tar, sand and white-wash.
Photographs of two forms of Rice.

(1). Sample of finely polished Siam rice, natural size. This is the rice that is so largely eaten by Chinese coolies. It is cheap and not of the first quality, as seen from the large number of fragmented grains.
Phosphorus percentage = 0.210 Cost = 6d. for 10 lbs.

(2). Sample of Indian Rice, slightly magnified.
This grain is not nearly so finely polished and so retains a great deal of the pericarp, the red colour of which can be seen in the print. It is the grain used by Indian coolies.
Phosphorus percentage = 0.469 Cost = 1 1/2d. for 10 lbs.
Photographs of two forms of Rice.

(1). Sample of ordinary third grade Rangoon rice, finely polished. A cheap variety largely purchased by coolies. Natural size.
Phosphorus percentage = \( \cdot243 \)  Cost = 5\( \frac{1}{2} \)d for 10 lbs.

(2). Sample of Chinese rice from Shang\( \ddot{\text{h}} \)ai, this is also a polished rice, but a certain amount of the finer pericarp remains adherent. It is largely used by the Chinese for making cakes with. Natural size.
Phosphorus percentage = \( \cdot261 \)  Cost = 3\( \frac{1}{4} \)d for 10 lbs.
(1). Sample of ordinary "padi", prior to milling, this is used by the natives of Malaya and China in the villages, being husked and winnowed by primitive methods. \footnote{1 lb. to 10 lb.}

(2). Sample of "Kadjang Idju" or the "Java Pea". This legumen is mixed with a form of sugar known as "Gula Aren" and boiled to a gruel so as to represent a rice diet. It is given largely in Java, and when used Beri-beri does not occur.

(Botanical name = Phascolus radiatus).
Photographs of two forms of Rice "Dust".

(1). This is the pericarp of the rice removed by the coarser form of milling, a lot of the fine pericarp remains adherent to the grain. There are some small fragments of rice amongst the sample. In Singapore used to feed pigs, in Rangoon made into cattle cakes for exportation.

Phosphorus percentage = 4.1.

(2). Similar to the above, but taken from a very finely polished rice, on which no pericarp was left at all.

Phosphorus percentage = 4.5.
---Dietetic Tables No 1---

Table to show the Amount of Food-stuffs (with daily variations) supplied per man per day.

<table>
<thead>
<tr>
<th></th>
<th>Sunday</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sam Rice</td>
<td>28</td>
<td>Sam Rice</td>
<td>28</td>
<td>Sam Rice</td>
<td>28</td>
<td>Sam Rice</td>
<td>28</td>
</tr>
<tr>
<td>Indian Rice</td>
<td>2</td>
<td>Indian Rice</td>
<td>2</td>
<td>Indian Rice</td>
<td>2</td>
<td>Indian Rice</td>
<td>2</td>
</tr>
<tr>
<td>Salt Vegetables</td>
<td>24</td>
<td>Salt Vegetables</td>
<td>24</td>
<td>Salt Vegetables</td>
<td>24</td>
<td>Salt Vegetables</td>
<td>24</td>
</tr>
<tr>
<td>Beans</td>
<td>1/2</td>
<td>Beans</td>
<td>1/2</td>
<td>Beans</td>
<td>1/2</td>
<td>Beans</td>
<td>1/2</td>
</tr>
<tr>
<td>Onions</td>
<td>1/2</td>
<td>Onions</td>
<td>1/2</td>
<td>Onions</td>
<td>1/2</td>
<td>Onions</td>
<td>1/2</td>
</tr>
<tr>
<td>Pork</td>
<td>1/2</td>
<td>Pork</td>
<td>1/2</td>
<td>Pork</td>
<td>1/2</td>
<td>Pork</td>
<td>1/2</td>
</tr>
<tr>
<td>Fresh Vegetables</td>
<td>3/4</td>
<td>Fresh Vegetables</td>
<td>3/4</td>
<td>Fresh Vegetables</td>
<td>3/4</td>
<td>Fresh Vegetables</td>
<td>3/4</td>
</tr>
<tr>
<td>Jams</td>
<td>1/2</td>
<td>Jams</td>
<td>1/2</td>
<td>Jams</td>
<td>1/2</td>
<td>Jams</td>
<td>1/2</td>
</tr>
<tr>
<td>Macaroni</td>
<td>1/2</td>
<td>Macaroni</td>
<td>1/2</td>
<td>Macaroni</td>
<td>1/2</td>
<td>Macaroni</td>
<td>1/2</td>
</tr>
<tr>
<td>How Cake</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**NB**

**Extras**

Under this heading comes Fruit (Papaya, Banana, Guava, etc.), eaten either as a fruit and as a vegetable and Juices, bought from Kongsi such as Mills, flour, Rambutans, Dried Apricots, Dried Oranges, "Juko Maltaca" etc.
Table to show the approximate Analysis of Food-stuffs in daily use, with their Energy Value per ounce in Calories, and the Calorific Value to be obtained from the amount supplied per man per day.

<table>
<thead>
<tr>
<th>Food Stuff</th>
<th>% Dry Matter</th>
<th>% Fat</th>
<th>% Alcohol</th>
<th>% Ash</th>
<th>Energy Value per ounce</th>
<th>Calories per man per day</th>
<th>Calorific Value Daily Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>32.7</td>
<td>5.0</td>
<td>0.419</td>
<td>0.3</td>
<td>98.0</td>
<td>30 oz</td>
<td>2940</td>
</tr>
<tr>
<td>Pork</td>
<td>60.9</td>
<td>12.3</td>
<td>26.2</td>
<td>0.7</td>
<td>81.6</td>
<td>4/3 oz</td>
<td>367</td>
</tr>
<tr>
<td>Lard</td>
<td>10.0</td>
<td>-</td>
<td>89.5</td>
<td>0.5</td>
<td>240.0</td>
<td>1 1/3 oz</td>
<td>312</td>
</tr>
<tr>
<td>Salt Fish</td>
<td>16.5</td>
<td>62.5</td>
<td>0.7</td>
<td>10.4</td>
<td>99.4</td>
<td>2/3 oz</td>
<td>228</td>
</tr>
<tr>
<td>Vermicelli</td>
<td>10.0</td>
<td>12.5</td>
<td>0.875</td>
<td>0.7</td>
<td>96.0</td>
<td>1 oz</td>
<td>96</td>
</tr>
<tr>
<td>Bean Stick</td>
<td>10.5</td>
<td>12.5</td>
<td>0.5715</td>
<td>5.0</td>
<td>96.0</td>
<td>3/4 oz</td>
<td>72</td>
</tr>
<tr>
<td>Dried Peas</td>
<td>19.0</td>
<td>21.0</td>
<td>1.8554</td>
<td>2.6</td>
<td>91.2</td>
<td>3/4 oz</td>
<td>68</td>
</tr>
<tr>
<td>Fresh Vegetables</td>
<td>92.8</td>
<td>1.0</td>
<td>0.3.36</td>
<td>0.9</td>
<td>6.0</td>
<td>10 3/4 oz</td>
<td>64</td>
</tr>
<tr>
<td>Macaroni</td>
<td>12.0</td>
<td>10.8</td>
<td>0.675</td>
<td>0.5</td>
<td>100.0</td>
<td>2 oz</td>
<td>50</td>
</tr>
<tr>
<td>Dried Beans</td>
<td>13.0</td>
<td>25.0</td>
<td>17.304</td>
<td>3.3</td>
<td>61.7</td>
<td>3/4 oz</td>
<td>60</td>
</tr>
<tr>
<td>Sweet Potatoes</td>
<td>72.9</td>
<td>1.6</td>
<td>0.3225</td>
<td>-</td>
<td>28.7</td>
<td>2/3 oz</td>
<td>66</td>
</tr>
<tr>
<td>Yams</td>
<td>79.6</td>
<td>2.2</td>
<td>0.5158</td>
<td>21.7</td>
<td>2/3 oz</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>Onions</td>
<td>89.0</td>
<td>1.6</td>
<td>0.3.63</td>
<td>-</td>
<td>9.5</td>
<td>1 3/8 oz</td>
<td>16</td>
</tr>
<tr>
<td>Prawns</td>
<td>25.0</td>
<td>2.0</td>
<td>0.4</td>
<td>6.2</td>
<td>1/4 oz</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Salt Vegetables</td>
<td>75.5</td>
<td>-</td>
<td>3.5</td>
<td>21.0</td>
<td>3.9</td>
<td>2/3 oz</td>
<td>8</td>
</tr>
</tbody>
</table>

NB. The above are not all supplied on the same daily ratios are shown on Table 0.
---Dietetic Tables No. 5---

Table to show the Total Potential Energy in Calories to be obtained from the Daily Food Supply with the relative percentages of Proteids, Fats and Carbo-hydrates.

<table>
<thead>
<tr>
<th>Day of Week</th>
<th>Food Stuff</th>
<th>Calories Value of Daily Supply</th>
<th>Percentages of Proteids</th>
<th>Percentage of Fat</th>
<th>Percentage of Carbo-hydrates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday</td>
<td>Rice</td>
<td>2940</td>
<td>5.0</td>
<td>0.1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>Pork</td>
<td>367</td>
<td>12.3</td>
<td>26.2</td>
<td>—</td>
</tr>
<tr>
<td>Sundays</td>
<td>Lamb</td>
<td>312</td>
<td>—</td>
<td>89.2</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Sweet Potatoes</td>
<td>66</td>
<td>1.6</td>
<td>0.3</td>
<td>22.9</td>
</tr>
<tr>
<td>Tuesdays</td>
<td>Macaroni</td>
<td>59</td>
<td>10.8</td>
<td>0.6</td>
<td>75.7</td>
</tr>
<tr>
<td></td>
<td>Irish Vegetables</td>
<td>21.3</td>
<td>1.0</td>
<td>0.3</td>
<td>3.6</td>
</tr>
<tr>
<td>Thursdays</td>
<td>Game</td>
<td>49</td>
<td>2.2</td>
<td>0.5</td>
<td>15.8</td>
</tr>
<tr>
<td></td>
<td>Onions</td>
<td>16</td>
<td>1.6</td>
<td>0.3</td>
<td>8.3</td>
</tr>
<tr>
<td></td>
<td>Prawns</td>
<td>8</td>
<td>4.0</td>
<td>0.2</td>
<td>—</td>
</tr>
</tbody>
</table>

Total Potential Energy = 3866 of which:
- Proteids supply 11%
- Fat 36%
- Carbo-hydrates 52%

---

<table>
<thead>
<tr>
<th>Day of Week</th>
<th>Food Stuff</th>
<th>Calories Value of Daily Supply</th>
<th>Percentages of Proteids</th>
<th>Percentage of Fat</th>
<th>Percentage of Carbo-hydrates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday</td>
<td>Rice</td>
<td>2940</td>
<td>5.0</td>
<td>0.1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>Lamb</td>
<td>312</td>
<td>—</td>
<td>89.2</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Salt Fish</td>
<td>228</td>
<td>62.5</td>
<td>0.7</td>
<td>—</td>
</tr>
<tr>
<td>Tueday</td>
<td>Vermicelli</td>
<td>96</td>
<td>12.5</td>
<td>0.8</td>
<td>75.5</td>
</tr>
<tr>
<td></td>
<td>Boneduck</td>
<td>72</td>
<td>12.6</td>
<td>0.5</td>
<td>71.5</td>
</tr>
<tr>
<td>Fridays</td>
<td>Dried Beans</td>
<td>68</td>
<td>25.0</td>
<td>1.7</td>
<td>58.4</td>
</tr>
<tr>
<td></td>
<td>Onions</td>
<td>16</td>
<td>1.6</td>
<td>0.3</td>
<td>6.3</td>
</tr>
<tr>
<td>Saturdays</td>
<td>Prawns</td>
<td>9</td>
<td>2.0</td>
<td>0.4</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Salt Vegetables</td>
<td>8</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Total Potential Energy = 3749 of which:
- Proteids supply 23%
- Fat 20%
- Carbo-hydrates 57%
Hospital Dietary

Daily Supply per coolie with times of meals.

N.B. Hospital coolies were thus provided with an abundant and varied diet, considerably more than that of the ordinary working coolie and possessing an even higher Calorific Value.

<table>
<thead>
<tr>
<th>Time</th>
<th>DIETETIC LIST</th>
<th>Food Stuff. Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.45 a.m.</td>
<td>Milk, Boiled Hour, Sugar.</td>
<td>Milk 12a</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>Puffed (boiled)</td>
<td>Puffed 10b</td>
</tr>
<tr>
<td>11 a.m.</td>
<td>Green Peas (boiled) Sugar &amp; Fat.</td>
<td>Peas 4b</td>
</tr>
<tr>
<td>2 p.m.</td>
<td>Tea (no solids, no milk)</td>
<td>-</td>
</tr>
<tr>
<td>12 p.m.</td>
<td>Sunday</td>
<td>Pork 4a</td>
</tr>
<tr>
<td></td>
<td>Monday</td>
<td>Fresh Potatoes 7b</td>
</tr>
<tr>
<td></td>
<td>Tuesday</td>
<td>Bran 5d</td>
</tr>
<tr>
<td></td>
<td>Wednesday</td>
<td>Preserved Peas 2a</td>
</tr>
<tr>
<td></td>
<td>Thursday</td>
<td>Green ‘Rice’ 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vermicelli 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>White Beans &amp; Peas.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rice 20a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vermicelli 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Beans 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>White Beans &amp; Peas.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rice 20a</td>
</tr>
<tr>
<td></td>
<td>Monday</td>
<td>&quot;Indian’ Rice 20a</td>
</tr>
<tr>
<td></td>
<td>Tuesday</td>
<td>Vermicelli 12a</td>
</tr>
<tr>
<td></td>
<td>Wednesday</td>
<td>Sweet Potatoes or Greens 3a</td>
</tr>
<tr>
<td></td>
<td>Thursday</td>
<td>White Beans &amp; Peas.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rice 20a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vermicelli 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Beans 12a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>White Beans &amp; Peas.</td>
</tr>
<tr>
<td>6 p.m.</td>
<td>Milk (no solids)</td>
<td>Milk 12a</td>
</tr>
</tbody>
</table>

Additional Information:

1. Pigeon Broth...frequently...
2. Fresh Fruit (Banana, Papaya, Pomegranate)....daily...
3. Fresh Vegetables (Long Beans, Chinese Cabbage, Radish, Vegetables grown at Bornigal)....frequently...
4. Juiced Pineapple...once a week...
TREATMENT
OF
BERI-BERI.

"Nam, quoniam variant animi, variamus et artes;
Mille mali species, mille salutis erunt".

OVID.
From what has been said it will have been gathered, that if the prophylactic precautions indicated are taken, the necessity for treatment will steadily become less. However in view of the great prevalence of the disease at the present day it may be as well to make a few statements upon the subject, with special reference to certain modes of treatment, which have been adopted with success and not mentioned in the literature on the subject.

A. HYGIENIC.

The lines already mentioned should be followed out. If possible the patient should have a change of dwelling, if not of locality. I do not think the importance of this is as great as was formerly insisted upon, but a change of air and surroundings must have a certain beneficial effect. In Christmas Island good results frequently followed the removal of a patient from Loading Point up to the hill hospital at the top of Phosphate Hill (altitude about 1000 feet), which faced the prevailing wind.

The Beri-beri patient should be placed in a well ventilated and thoroughly dry ward, if possible one kept entirely for this disease. This I mention more for the protection of the case of Beri-beri, than for the other patients. The disease is not infectious, no coolie ever acquired Beri-beri in hospital at Christmas Island, but on the other hand a man suffering from
Beri-beri is far more liable to develop some other complication if exposed to it, most notably dysentery and tuberculosis.

Care in selecting the site for a hospital should be taken to see, that the drainage is efficient and that the water supply is pure and abundant.

The patient should be out in the sunshine as much as possible and avoid all forms of dampness.

In mild cases and during convalescence he should be encouraged to take gentle exercise, provided no cardiac condition prohibits it.

B. DIETETIC.

The daily diet should be carefully regulated. Many convalescent patients demand rice and they should be given nothing but the "cured" form.

The Java Pea (PHASEOLUS RADIATUS) forms an excellent substitute and when cooked with Gula Aren (a form of a sugar) can be made to resemble a sort of Rice mash, which is most palatable.

Care should be taken to see, that the stomach is never overloaded, only a moderate amount of food being taken at each meal. The principle meal should be in the middle of the day.

In the Tung Wah hospital Hongkong, patients may choose, which ward they prefer in one "cured" rice is eaten and in the other "uncured". In 1918 the mortality in the former ward was 19% in the latter it was 41%. The appended chart shows the progressive fall in the
number of cases of Beri-beri on Christmas Island, the result of improved hygiene and of gradually increasing the percentage of "cured" rice in the food.

The list following it is the dietetic scale of hospital rations in the Government Hospitals of the Straits Settlements.
### DIET FOR CHINESE

<table>
<thead>
<tr>
<th>Item</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>1½ lbs daily.</td>
</tr>
<tr>
<td>Fresh Fish</td>
<td>3 oz 5 times a week.</td>
</tr>
<tr>
<td>Salt Fish</td>
<td>3 oz 2 times a week.</td>
</tr>
<tr>
<td>Fresh Beef or Pork</td>
<td>4 oz daily.</td>
</tr>
<tr>
<td>Vegetables</td>
<td>3 oz daily.</td>
</tr>
<tr>
<td>Salt</td>
<td>½ oz daily.</td>
</tr>
<tr>
<td>Onions</td>
<td>½ oz daily.</td>
</tr>
<tr>
<td>Garlic</td>
<td>¾ oz daily.</td>
</tr>
<tr>
<td>Lard</td>
<td>¼ oz daily.</td>
</tr>
</tbody>
</table>

### DIET FOR MALAYS

<table>
<thead>
<tr>
<th>Item</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>1½ lbs daily.</td>
</tr>
<tr>
<td>Fresh Fish</td>
<td>3 oz 5 times a week.</td>
</tr>
<tr>
<td>Salt Fish</td>
<td>3 oz 2 times a week.</td>
</tr>
<tr>
<td>Fresh Beef or Goat</td>
<td>4 oz daily.</td>
</tr>
<tr>
<td>Vegetables</td>
<td>3 oz daily.</td>
</tr>
<tr>
<td>Dhall</td>
<td>½ oz daily.</td>
</tr>
<tr>
<td>Salt</td>
<td>½ oz daily.</td>
</tr>
<tr>
<td>Curry Stuff</td>
<td>1 oz daily.</td>
</tr>
<tr>
<td>Coconut Oil</td>
<td>½ oz daily.</td>
</tr>
</tbody>
</table>

### DIET FOR BENGALIS

<table>
<thead>
<tr>
<th>Item</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flour, Kanak</td>
<td>1 lb daily.</td>
</tr>
<tr>
<td>Chee</td>
<td>½ lb daily.</td>
</tr>
<tr>
<td>Vegetables</td>
<td>6 oz daily.</td>
</tr>
<tr>
<td>Dhall</td>
<td>1 oz daily.</td>
</tr>
<tr>
<td>Salt</td>
<td>½ oz daily.</td>
</tr>
<tr>
<td>Curry Stuff</td>
<td>1 oz daily.</td>
</tr>
</tbody>
</table>

### DIET FOR SIHKS

<table>
<thead>
<tr>
<th>Item</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flour, Kanak</td>
<td>1½ lbs daily.</td>
</tr>
<tr>
<td>Chee</td>
<td>¼ lb daily.</td>
</tr>
<tr>
<td>Milk</td>
<td>½ pint daily.</td>
</tr>
<tr>
<td>Sugar</td>
<td>2 oz daily.</td>
</tr>
<tr>
<td>Kacjang Idju</td>
<td>2 oz daily.</td>
</tr>
<tr>
<td>Vegetables</td>
<td>1 lb daily.</td>
</tr>
<tr>
<td>Curry Stuff</td>
<td>1 oz daily.</td>
</tr>
</tbody>
</table>
C. MEDICINAL.

This is almost entirely symptomatic, but the following general lines of treatment should be adhered to, as being of the utmost importance.

(1). The bowels should be kept freely open. For all time the saline purgatives have been held in honour in this respect especially Magnesia. Occasionally Castor Oil or Jalap may have to be resorted to and care must of course be taken not to overdo it so as to increase the weakness especially in oedematous cases.

(2). Massage at least twice a day with some simple linament. I cannot lay sufficient emphasis on this. I have seen most splendid results by thus keeping up the tone of the muscles, and it is a precaution which is all too apt to be neglected, in places where a large amount of coolie labour is employed. It is so easy to do and many a Chinese coolie will be found to be a born masseur.

(3). The administration of Tonics during convalescence. During the acute stages when there is much hyperaesthesia of the muscles Strychnine is contra-indicated, but in the later stages it is of the greatest value. The combination, that I found to be far the most satisfactory was Strychnine and Formic Acid (25%). The results, that one obtained were occasionally most striking, and others to whom I have recommended a trial of this formula, have expressed their great gratification at the effects produced.
The patients felt that they were improving, which is half the battle in the treatment of any disease, and especially such a depressing complaint as Beri-beri. They expressed a real sense of well-being.

Taking the results taken over 12 months, it was found, that the average period in hospital under this form of treatment was reduced from 47 to 34 days.

Cases in which it was found, that there was oxaluria present were given Calcium Formate in the hopes, that the Calcium would combine and form the insoluble Calcium Oxalate and liberate the Formic Acid. I think from what I saw, that the choice of drug was good, but I have not seen a sufficient number in which oxaluria was present to be able to judge the real therapeutic value of this line of procedure.

FORMIC ACID.

The question of the therapeutic value of Formic Acid is still quite undecided. Many glowing accounts of its use in certain diseases have been published and, from the good results obtained by Croom in Diphtheria, a trial of it in this disease seemed to be indicated.

It was originally held, that Formic Acid and the Formates, when properly given, had a powerful stimulating effect upon the voluntary muscular system increasing its vigour.

Clement of Lyons in a paper read before the Paris Academy pointed out, that the use of Formic Acid caused a feeling of increased strength, vigour and activity. He referred to its action "toni-musculaire" and asserted
and asserted that, using a Mosso Ergograph, an increased power of work could be demonstrated, even as much as 85 kilogrammeters. Further he stated, that the eagerness for work became greater, sleep more refreshing, movements more brisk and causes, which lead to exhaustion of mind and body, felt less.

Later Muchard after 2 years treatment and experiment confirmed these views.

More recent work has shown, that the Formates do not increase cardiac tonicity, but they do increase the contractility of the heart. It is only right to mention, that the work of Goodall and Mitchell (1906) disposed of many of these claims of Formic Acid and they summed up with the conclusion, that in the doses stated Formic Acid had no demonstrable effect on the circulatory or muscular systems.

Apart from any experimental evidence whatever and judging simply by the clinical results obtained, I should certainly be inclined to the view, that the therapeutic power in certain cases is anything but small, it was frequently found to be of very exceptional value, and I should be sorry now to be compelled to do without it in the treatment of Beri-beri.

In acute cases it was found of value to administer the rice "polishings" made up with Aqua Chloriform as a medicine. Coolies who refused to take the polishings in their food could always be got to take them in this form. By this means it was often prescribed throughout the whole course of the disease.
Occasionally one tried Sanatogen, but the price of this preparation is too prohibitive to enable it to be given to coolie patients, otherwise it should prove of value in this disease, for, I believe, it consists of Phosphoric Acid and pure Casein, the phosphorus being entirely in the organic form, thus supplying this essential element for the physiological well-being, which is so deficient in Beri-beri.

Such then are the general lines of treatment in this disease, for the rest it is purely symptomatic, emergencies being dealt with as they arise. We will then pass on to consider the special treatment of the different prominent symptoms of the disease.

**OEDEMA.**

So great in amount may this be, that the most energetic treatment is often called for. In the ordinary cases a simple diuretic is all that is necessary; of those in common use I have found Potassium Citrate to be decidedly the most useful, especially so on account of its non-irritating action on the kidneys, while any action it may have is at the expense of the oedema. This was used as a routine in combination with Infusion of Digitalis. In these cases spontaneous diuresis was the first and surest sign of convalescence. Tapping had on some occasions to be resorted to, and one case in which the urinary secretion had fallen to extremely low rallied rapidly after being tapped on several consecutive days.
I am not convinced of the value of vapour baths they cause a lot of discomfort in the Tropics and aggregate the vascular weakness. Though by this means a lot of water can be got rid of, yet the amount of Urea lost is small, so the kidney is left with a more concentrated mixture to deal with; this of course can be partly met with by reducing the amount of Protein in the food.

In dropsical cases it was customary to insist on a chloride-free diet, which according to the views at present held ought to have had some beneficial effect. Pilocarpine was occasionally resorted to and this drug in combination with Sodium Salicylate was highly spoken of by Baelz.

GASTRO-INTESTINAL SYMPTOMS.

DYSPEPSIA.

This is often met with in early cases and can usually be dealt with by dietetic means plus the exhibition of such drugs as Rhubarb, Bismuth and Soda.

DIARRHOEA.

This in my experience is not met with so frequently, but it may become a prominent and even urgent symptom at any stage of the disease. It can usually be controlled by a careful attention to the diet accompanied by initial purgation and subsequent use of astringents and sedatives. I found, that in such cases, if the symptoms were irresponsive to this line of treatment, the administration of Milk curdled by the Lactic Acid organisms often proved of the greatest therapeutic value and would always employ this method in obstinate cases. (see special case).
HICCOUGH.

This is often a prominent and ominous symptom in advanced cases. In order to combat it when it arises Cocain is often the best drug to use, and this can be considerably aided by the application of mustard over the epigastrium. By experience one found that the best way of applying this is to rub the dry powder into some cotton wool and then apply it without moisture to the skin, and cover it with oil-silk. The moisture of the body frees the sulpho-cyanate of allyl into action, and the result is that one gets a good reaction without any discomfort.

Tractio of the tongue is recommended and may be resorted to.

VOMITING.

In advanced cases one learnt to look upon this as a particularly dangerous symptom, in fact persistent vomiting at any stage is a thoroughly bad omen.

The drugs recommended for Hiccoough are all useful, especially Cocain, but sometimes the whole medical armamentarium may be tried and prove equally inefficent.

The sucking of small quantities of Ice may allay the symptoms and gastric lavage is sometimes the only means of coping with the difficulty.

PARALYSIS OF THE DIAPHRAGM.

By the time that this symptom has occurred, I do not think, that the patient has very much chance. It is said, that the best results have been obtained
by electricity. One pole is applied to the epigastrium, the other to the root of the neck over the Phrenic Nerve. The application is made for a period of 10 minutes several times a day.

CARDIAC SYMPTOMS.

In mild cases the cardiac condition may not require any special treatment. Digitalis, if reliable preparation used, is always of the greatest when palpitation or any cardiac weakness shows itself.

In the Acute Fulminating Form a number of drugs have been advocated by different clinicians, a certain sign that none of signal value.

When the cardiac distress was very great, I have seen temporary relief from the inhalation of Amyl Nitrite, and Nitroglycerine (gr-1/100th) every ½ hour was recommended warmly by Manson. I have tried this method, in fact the dresser was instructed to have it always in readiness, and it proved of the greatest value in one case, in which the patient eventually made what appeared to be an impossible recovery. In other cases it has failed signally to produce the slightest reaction.

Strychnine provided that the muscular hyperaesthesia is not too acute is one of the sheet anchors and subcutaneous injections of Camphor will sometimes prove most beneficial.

The question of VENESICTION has to be considered. It is not by any means always advisable, especially in anaemic cases with a low blood pressure. In others
Treatment——

where there is no contra-indication the drawing off of 10 to 20 ounces of blood is often most advantageous, thus giving the engorged right heart a chance.

In cases where contra-indications exist good results can be obtained by practising venesection upon one arm and transfusing at the same time into the other.

Or an excellent method in atrophic cases is to apply a bandage of elastic to one limb in such a way as to obliterate the venous return, but not to interfere with the arterial supply, by this means the limb swells up and quite a large quantity of blood can be cut off from the system, this gives the heart a chance to rally, and when matters have somewhat improved, the blood supply can be gradually returned to the general system by putting on the bandage at lower levels.

In cases where there is considerable pericardial effusion, to such extent that the cardiac action is embarrassed, paracentesis must be employed, as giving the patient the only possible means of recovery.

In two cases I tried ASPIRATION of the Right Ventricle, and in one of these the patient, who was comatose at the time temporarily recovered consciousness.

RESPIRATORY SYMPTOMS.

The dyspnoea that is so often present is usually cardiac in origin. Of whatever origin striking use can be got by the method of dry-cupping. The inhalation of wet Oxygen has been recommended, but I have had no opportunity of judging its therapeutic value.
In cases where the dyspnoea is a marked symptom, a hypodermic injection of Morphine in combination with Digitaline should be tried, also the pleuræ should be frequently examined to ascertain if any effusion has occurred, which is such a constant cause of urgent dyspnoea, and if present to deal with it by tapping if necessary.

**MUSCULAR SYSTEM**

When the pain is very acute Strychnine is contra-indicated, but some relief can be obtained by the applications of hot fomentations and it may be necessary to give Morphia hypodermically. I have already laid stress on the value of massage to preserve the muscular tone. If Talipes Equinus occurs Tenotomy of the Tendo Achilës may have to be resorted to, or Fixation of the ankle joint, in order to give the patient a useful limb.

During convalescence Electricity should be of value: with the battery at my disposal I could form no adequate judgement of the full possibilities of this line of treatment, but imagine it might in some cases be almost indispensable. If muscular excitability still exists to some extent, one would recommend the Faradic Current otherwise the Galvanic.
CONVALESCENCE.

This has already been touched upon, but I would just sum up the remarks again. Massage and Electricity, graduated exercises with cold packs and douches recommended, and the ordinary tonics Strychnine, Iron and the like administered.

A drug much used in Brazil during convalescence and most highly spoken of, is the Fluid Extract of Marapuana (Livicosma ovata), given in doses of 10 to 20 drops a day.

Any other symptoms that arise such as Bedsores, Ulcerations, Convulsions (rare) and Laryngitis must be treated by ordinary means.

Other drugs, that have not been mentioned and may prove useful are Urotropine, in cases where some bladder trouble has arisen and Nuclein. This later drug I have tried hypodermically and a leucocytosis certainly resulted, but no marked alteration in the symptoms were noticed.

Such then are the general lines of treatment, that one would recommend, though but briefly referred to. Most of the drugs mentioned have at one time or another proved of conspicuous value, only unfortunately when put to the test on another occasion to prove singularly inefficient.

There is thus no specific remedy for the disease, and some cases will demand all the therapeutic resources available and call forth all the physicians ingenuity in their treatment.
It is really remarkable how on many occasions, when all seems hopeless, one may strike a remedy, that seems to act upon that particular patient like a charm and he makes a steady though always a slow recovery. Whether it is the drug, that one has administered that has really helped him round the corner or the VIS NATURAE, the saving effort of nature to retain the species, is not always a matter easy to decide, but it is always comforting to the physician to take the credit to himself, to wipe off a little from the list of those who have slipped through in spite of everything.
HISTORIES and NOTES on SPECIAL CASES

of

BERI-BERI.

I have added these notes on special cases of this disease, as I think, that they may be of some interest and value. They are copied from one's own Case book and there is in consequence a deal of repetition, but as some of the cases dealt with exhibited symptoms, which are of great rarity in Beri-beri, I do not think that they are altogether out of place.
Atrophic Beri-beri with double Post-beri-beric Optic

Atrophy and Paralysis of the Diaphragm.

AGE 27
OCCUPATION COOLIE
NATIONALITY CHINESE—CANTONESE
ARRIVED C.I. FEB. 1908.

History.

On May 23rd 1908 he was admitted to hospital suffering from a mild attack of Beri-beri. He complained of numbness of the legs and fingers with weakness on exertion accompanied by some palpitation.

On examination there was comparative anaesthesia to all sensory stimuli down the anterior of the tibiae and over the tips of the fingers, hyperaesthesia of the calf muscles and complete loss of knee jerks. The heart's action was accelerated, but the valves were closed.

There was no oedema, no alteration in the Skin reflexes, and the Arm jerks were still present, but he was unable to perform the "squatting" test.

Placed under suitable treatment he improved steadily and was discharged apparently quite well on July 10 1908. The knee jerks were present though slight and he could do the "squatting" test, while the heart was regular and well balanced. He returned to light work for 4 weeks, during which time he was under supervision and then went back to the quarries.

On October 22nd 1908 the patient was re-admitted on this occasion his only complaint was increasing loss of vision. With oblique illumination both lenses appeared clear.
The pupils reacted readily to light and accommodation. There was no conjunctivitis and the only abnormality was an Internal Pterygium of the right eye, which did not interfere with the line of vision.

Ophthalmoscopic examination at this stage proved negative, a note was made, that the discs seemed to be rather pale, but this was attributed to his general condition, for he was distinctly anaemic at the time. On testing his vision he said, that he could not discern even large objects at a distance of 10 feet, and could not read any of the test letters, but could accurately count fingers at 18 inches from the Left eye and 12 from the Right.

As regards other evidences of nervous disorder none were present, the knee jerks were active and the heart was sound.

He was admitted to hospital and placed on tonic treatment, at first it was thought, that there was a large element of malingering about him, for at night when no one was about he was able to play Chinese cards with the other patients, but soon it became apparent, that he was steadily losing his sight.

Ophthalmoscopic Examination was frequently made and a slowly progressing double optic atrophy was seen to be occurring. This was a simple degenerative change, at no time was there any apparent neuritis preceding the atrophic change, no hyperaemia of the discs and no loss of definition of their margins.

On Dec 20 1903 the eye condition was as follows:—

1. The fundus was anaemic, vessels both arteries and
veins were much diminished in size.

2. The disc was a pearly white colour with sharp clearly defined margins.

3. Vision was lost and the loss had apparently been concentric, he could still recognize a bright light when flashed on the retina.

An attempt to show the appearance of the right fundus is appended.

The general health remained perfectly good, his appetite was excellent and he seemed quite happy.

On January of 1909 arrangements were made to send him home, as he especially desired it, these took some time to complete as a lot of correspondence was necessary.

On March 29, 1909 when everything was finally settled, he developed a second attack of Beri-beri. It is interesting to note, that though in hospital only "cured" rice was eaten, yet the patient developed the disease, but on inquiry this interesting point came to light, that as there was nothing apparently wrong with the patient save his blindness, he had been allowed to go down to the coolie lines as much as he liked and had been practically eating nothing but "uncured" rice, for some time previously. It may be considered, that the progressive degeneration of the Optic nerves, was an evidence of existing Beri-beri, but I am rather inclined to look upon it as a post-beriberic atrophy, for during all these months in hospital, when he had been under careful observation, he had none of the other manifestations of the disease. His knee jerks were active, locomotion was unimpaired,
the "squatting" test could be readily performed and the cardiac condition was highly satisfactory.

The onset of active symptoms of the disease seems to me to point to a fresh infection.

This attack, which was not preceded by any rise in temperature, nor was there any subsequently, was ushered in with Gastro-intestinal symptoms, and was at first diagnosed as being probably dysenteric.

On March 29th then he vomited 3 times and his bowels moved 16 times, the stools contained a trace of mucous but no blood. He received Castor Oil followed by Morphia, Chloroform and Bismuth. On the 30th he had 14 motions and on the 31st 19, after that the stools became less frequent, lost the trace of mucous and on April 4th had returned to a normal well-formed motion.

The faeces were examined for the ova of Ankylostoma duodenale, but with negative results.

Two days later on April 6th he first complained of numbness of the hands and legs, and a feeling of oppression in the pit of the stomach with weakness of the limbs.

EXAMINATION.

Definite anaesthesia amounting to considerable blunting of sensibility and delay in perception to all stimuli. Loss of perception to Tactile and Thermal stimuli over the anterior and external surface of the legs, as far up as the knee (not of the feet.) and of the finger tips and thenar eminences, all of which were almost symmetrical on both sides (fig 1). There was marked hyperesthesia of the calf muscles.
which felt flabby and toneless. The knee jerks were just perceptible, but the arm jerks were quite active. There was no alteration in the superficial reflexes, the gait was of the "steppage" type, he walked on a broad base, and raised the feet high in order to clear the ground. He was quite unable to perform the "squatting" test and testing the push of the foot showed the weakness of the extensors. He had no cardiac subjective symptoms and during repose the heart's action was regular and steady, only on exertion was there some cardiac irritability present, after attempting to walk a few steps the pulse rate would rise from 75 to 125. There was no oedema and no dyspnoea present.

The Eye. Pupils equal, moderately dilated, inactive to light. Sight by this time had completely gone.

Ophthalmoscopic Examination.

The fundus was paler and the vessels even more constricted and along them a white line could be seen to run on either side far out from the disc. The disc itself was sharply defined, slightly cupped and of bluish white colour. (There was no further alteration in this condition).

The Cerebro-spinal Fluid was clear, SP GR 1001, under no pressure, microscopically an occasional epithelial cell, no lymphocytosis.

The treatment at this time was on general lines, tonics including Formic Acid with absolute rest, massage, and a generous easily digested diet.
The patient continued to get steadily worse and by April 22nd the anaesthesia had spread to the level of the umbilicus and over the anterior surface of the forearms (Fig 2), this loss of sensibility to stimuli was complete over the legs from midthigh downwards and much impaired over the rest of the affected area.

The muscular hyperaesthesia was much more marked, no longer being limited to the calf muscles, while at night he suffered from cramps in the legs sharp and excruciating, causing him to cry out loudly, and requiring injections of Morphia to give relief. All the deep reflexes were absent, the superficial were present.

He was unable to stand alone and foot-drop was apparent, while increasing weakness of the forearm muscles could be demonstrated. The heart was normal in size and position and the sounds remained closed, but its action was increased in rate, the rhythm being almost embryonic from the shortening of the diastolic pause. There was no oedema. Occasionally there were attacks of dyspnoea and a cough had developed, but no physical signs of any pulmonary disease.

The Blood, which had been examined frequently, was becoming progressively more anaemic (vide chart) and the Urine, which had hitherto been quite clear, became alkaline, pale, thick, foetid and had a deposit of phosphates.

Thus, with the more extensive involvement of the nervous system, the downward progress of the disease went on, presenting a clinical picture of striking interest.
On April 28th information regarding cutaneous sensibility could not be obtained, but there was general paresis of the limbs and marked muscular atrophy; the cramps though not so frequent were still severe and his whole body seemed to be extremely sensitive. Wrist-drop was very apparent and the feet had assumed the position of equino-varus. He had to be constantly shifted on his bed, speech was reduced to a hoarse whisper and coughing became more and more difficult owing to the paralysis of the laryngeal muscles and those of forced expiration. The heart was little altered in size, but the apex beat was displaced outwards into the axilla. The action was rapid and the first Mitral sound was replaced by a soft blowing murmur propagated out into the axilla as far as the Post-scapular line, the second sound was short and sharp. In the pulmonary area a systolic murmur was also audible. The pulse was still regular, but the Maximum Systolic Pressure was steadily falling. Slight oedema of the ankles present, but no hepatic enlargement or tenderness. The urine had rapidly cleared under the administration of Urotropine, but now retention set in and catheterization had to be employed. The blood remained much as before, but there was a steady increase of the coagulation period (vide blood chart). There was loss of control over the rectal sphincters and incontinence of fæces.

The Cerebro-spinal fluid was clear, watery, SP GR 1007, trace of albumen, and microscopically epithelial cells with an occasional lymphocyte.
On April 29th the last scene was ushered in with paralysis of the diaphragm, the costal arch became unduly widened, while the epigastrum was drawn backwards and upwards with each inspiration, all over this area there was marked tympanites and the apex beat was displaced outwards, (an attempt to show the condition present will be seen in the diagram).

There were frequent attacks of dyspnoea and complete aphonia. The superficial reflexes were lost. The patient lay stretched out and motionless, his sightless eyes "gazing" upwards, but consciousness was still present and nourishment could still be administered. The apex beat was still further out and the left side of the chest became dull. Blood Pressure continued to fall. The pulse at the wrist became impalpable, while auscultation over the heart revealed its tumultuous action with bruits in all areas.

On May 2nd a condition of coma came on, the extremities became perfectly cold and slightly livid, respiration shallow and ineffectual, while the cardiac action suddenly became slower and death silently inter-vened.

**BLOOD CONDITION** dealt with on separate chart, coagulation period advanced from 3 to 9 minutes.

**URINE CONDITION**
- Dec 27 Acid, clear, amber, no abnormality.
- Mar 29 - - - Indican in abundance.
- Ap 10 - - - less.
- Ap 22 Alk, turbid, pale, Indican trace, Phosphates abundant, pus.
- Ap 28 Acid, clear, amber, slight urate deposit, no pus.
- May 1 (catheter) 15 oz in 24 hrs, ac, 1017, clear, no abnormality, trace of Indican, Urea 53.25 grs in 24 hrs, Chlorides 30 grs, Micros: no organisms, 3 "hyaline bodies".

May 2 (P.M. Spec) Urea 4.90 grs to oz, Chlorides 40 grs.
NOTES on the CASE.

A full history of this case seemed to be of considerable interest, as it illustrates in many ways the salient points of Beri-beri, and at the same time possesses many symptoms not commonly seen in the disease.

The question of Treatment has been almost entirely disregarded in the description of the case, as it had little bearing on the matter, but throughout it was symptomatic. Before the second attack of Beri-beri occurred the patient was kept in the open air, given a generous dietary coupled with such tonics as Arsenic, Iron and Cod Liver Oil and under this his blood condition and general health improved. After Beri-beri symptoms developed treatment was directed to keeping up the tone of the muscles and avoiding deformities, by daily massage and the administration of Formic Acid, combined with general tonics. The cramplike pains were treated with Lead and Opium dressings and Potassium Bromide internally, and finally injections of Morphine. Later the failing heart required energetic and frequent stimulation, Strychine, Ether and Nitro-glycerine, Venesection also in combination with Adrenaline, proved of value.

In anaemic cases in which the venous blood pressure was high, it was not always deemed advisable to employ venesection, but a good substitute was found to be obtainable by applying an elastic bandage round the limbs, tight enough to obliterate the vein but not the main artery. By this means a large amount of
blood is, so to speak, cut off from the general circulation and the venous pressure falls and the heart obtains relief for the time. The blood is only allowed to return by degrees, namely by altering the position of the ligature, and thus a too sudden over-loading of the right side of the heart is avoided.

To return to the case in hand, it was pre-eminently Beri-beri of the True Atrophic Type, the clinical picture was complete, as shown by the ever-increasing anaesthesia of the skin, the stage of the acute cramp like pains, the flabby wasted muscles, the daily loss of flesh, the progressive loss of muscular power, with the Foot- and Wrist-drop. The retention of consciousness also was characteristic, retained indeed, till with incontinence of foeces, paralysis of bladder, diaphragm and larynx, attacks of dyspnoea and falling blood pressure, death closed the scene.

To the following points I would draw special attention.

The Progressive Anaemia.

As shown by blood examinations taken over a considerable period. It was held at one time by some, that Beri-beri might be caused by anaemia, but it is now certain, that the anaemia is a sequel of the Beri-beri, the toxin of which disease has apparently a very constant deliterious effect on the corpuscular elements of the blood. During the quiescent stage, when there were no apparent active symptoms of the disease, considerable improvement in the blood condition did occur, but hand in hand with the appearance
of acute symptoms there was progressive anaemia, which
did not lend itself to treatment at all. There was de-
crease in R.B.C.- Hh -and in the W.B.C. this last is
of interest for as a rule there is a certain amount of
leucocytosis; finally the blood film almost resembled
Pernicious Anaemia and the Color Index was 1, and besides
the alterations in shape, size and staining power a few
nomoblasts were also seen.

THE DOUBLE OPTIC ATROPHY.

This symptom is of great interest on account of
its rarity, for beyond the pneumogastric the cranial nerves
seldom become affected in Beri-beri. In Allbutt's
System of Medicine, Manson in discussing this disease
saya "the cranial nerves as a group and the higher
nerve centres being exempt" and "neither, unless very
exceptionally, is there any implication of the centres
or nerves of sight". Searching through the literature on
the subject I can find no mention of the Optic nerves
having been implicated.

On the other hand there is little reasonable
doubt, but that the complication was a sequel to the
first attack of Beri-beri, the onset of atrophy being
slow and progressive, a pure degeneration of the nerve,
to the naked eye unaccompanied by any inflammatory pro-
ceess. The nerve supply of the extrinsic and intrinsic
muscles of the eye was unaffected.

Other points worthy of note, in the review of the
case on account of their comparative infrequency, are...
The complete anaesthesia of the legs to all sensory stimuli during the later stages of the disease. This occurs most often in atrophic cases such as this, but is not a common symptom and appears to me to be a probable sign, that a Radiculitis had occurred, similar to the degenerative process in the nerves. The Sensory Roots having become affected, especially as the specimen of Cerebro-spinal Fluid removed in the post-mortem room contained a distinct lymphocytosis, as compared with that examined during life before the advanced symptoms referred to had made their appearance.

The terminal loss of superficial reflexes also of interest. The paralysis of the diaphragm with the characteristic tympanites and falling in of the abdominal wall on inspiration, is another point of note. Also the slowing of the pulse just before death and the sudden final development of cardiac bruits in all areas, due possibly to cramp of the heart muscles themselves, for there was no post-mortem evidence of any valvular disease.

The manner in which structure after structure supplied by the Vagus became affected, formed a clinical picture of wonderful interest and completeness.

APPENDED ARE:

1. Post-mortem Notes on the Case.
2. Two Charts to show the increasing change of Cutaneous Sensibility.
4. Diagram of the Fundus Oculi.
5. Table of Blood Counts.
7. Diagram to show Paralysis of the Diaphragm.
8. Micro-photograph of Transverse Section of Left Vagus Nerve.
9. Micro-photograph of Transverse Section of Right Optic Nerve.
May 2nd. Necropsy at 2 pm, 4 hours after death, weather hot and dry, marked emaciation of body. Rigor mortis complete. Pupils equal and medium. All superficial veins contain fluid blood, abdominal muscles pale.

**ABDOMEN.** Liver small, firm, anterior border sharp, bile runs freely, kidneys healthy capsule strips freely. Spleen small, firm, cuts with some resistance. Stomach no congestion of mucosa, but marked pallor and friability with a few areas of necrotic absorption. Duodenum first part slightly reddened. Rest of intestines normal, no worms.

**THORAX.** Lungs and pleurae healthy, Pericardium contains slight excess of clear fluid, is non-adherent. Heart right side much dilated, full of dark, semi-fluid, very friable post-mortem clot, muscle wall of all chambers atrophied, pale, flabby and attenuated. Coronary vessels normal, cardiac valves healthy but aortic and mitral incompetent.

**MUSCLES.** Of lower limbs especially pale, wasted and flabby, no oedema of muscle or subcutaneous tissue. Cerebro-spinal Fluid removed post-mortem was clear and under no pressure, Sp Gr 1008, trace of albumen, no coagulation on boiling. Microscopically after being centrifurilized, presence of a few cell elements epithelial and polymorpho-nuclear leucocytes, only a few lymphocytes (2 to a field).
Blue colour denotes impaired Sensibility
Red colour denotes complete inanition

April 22, 1909.
NOTES.

Blood Pressure readings registered during the last 10 days of life, taken by Martin's modification of the Riva Roci Sphygmomanometer. Chart shows the steady fall of the Blood Pressure and the rather rare terminal fall in the Pulse Rate. The final Blood Pressure reading was only approximate. All readings taken from the right arm with the body in the prone position.
Primary Optic Atrophy (Case of Lok Loy)

Fundus. Pallor towards the disc.
Vessels both veins and arteries diminished in size and showing characteristic white line.

Disc. Pearly white, slightly cupped, edges well-defined, lamina cribrosa well seen.
### Blood Counts

<table>
<thead>
<tr>
<th>Date</th>
<th>Red Corpuscles</th>
<th>Red Corpuscles per cent</th>
<th>Hæmoglobin</th>
<th>Hæmoglobin per cent</th>
<th>Colour Index</th>
<th>Leucocytes per c.mm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1908</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oct 30</td>
<td>3,700,000</td>
<td>74%</td>
<td></td>
<td>74%</td>
<td></td>
<td>1,000</td>
</tr>
<tr>
<td>Dec 27</td>
<td>4,400,000</td>
<td>88%</td>
<td></td>
<td>85%</td>
<td></td>
<td>0.965</td>
</tr>
<tr>
<td>1909</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feb 15</td>
<td>3,200,000</td>
<td>64%</td>
<td></td>
<td>70%</td>
<td></td>
<td>1.093</td>
</tr>
<tr>
<td>Mar 15</td>
<td>2,900,000</td>
<td>58%</td>
<td></td>
<td>58%</td>
<td></td>
<td>1.000</td>
</tr>
<tr>
<td>Apr 23</td>
<td>2,500,000</td>
<td>50%</td>
<td></td>
<td>57%</td>
<td></td>
<td>1.040</td>
</tr>
<tr>
<td>May 1</td>
<td>2,500,000</td>
<td>50%</td>
<td></td>
<td>30%</td>
<td></td>
<td>1.000</td>
</tr>
</tbody>
</table>
PARTicular CASES——

LOH SUN.

Graphic Chart to show the result of Blood Examinations.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>8,200</td>
<td>5,000,000</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8,000</td>
<td>4,750,000</td>
<td>95</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8,000</td>
<td>4,500,000</td>
<td>90</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,800</td>
<td>4,250,000</td>
<td>85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,600</td>
<td>4,000,000</td>
<td>80</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,400</td>
<td>3,750,000</td>
<td>75</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,200</td>
<td>3,500,000</td>
<td>70</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7,000</td>
<td>3,250,000</td>
<td>65</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,800</td>
<td>3,000,000</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,600</td>
<td>2,750,000</td>
<td>55</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,400</td>
<td>2,500,000</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,200</td>
<td>2,250,000</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6,000</td>
<td>2,000,000</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Temp: 86º F  Coagulation Period: 4½ 4 4¾ 6½ 7 9

Alkalinity: N4/140 N7/100

\[ \begin{align*}
\text{W.B.C.} : & \quad - \quad - \\
\text{R.B.C.} : & \quad - \quad - \\
\text{Hb} : & \quad - \quad - 
\end{align*} \]

NOTES.

Changes in the blood vessels were progressive, in the last film (May 1st) the appearance was almost that of Pernicious Anaemia. There was marked pallor of the R.B.C. with alterations in size (macrocyes and microcytes) in shape (poikilocytosis) and in colour (polychromatophilia). Vacuolation was also present and 2 Normoblasts were seen.

Differential

- Leucocytes =
  - Polymorphs --- 63 %
  - Lymphocytes --- 18 %
  - Mononuclears --- 14 %

- Count
Loh Sun: Paralysis of the Diaphragm

Diagram to show
Paralysis of Diaphragm

- Area of Hepatic Decline
- Area of Sympanites
- Apex Beat
Transverse Section of Left Vagus Nerve (x 260)

Two bundles of nerve fibres separated by band of Endoneurium.

In the bundle nearest to the observer degeneration is advanced, and there are many large empty spaces with here and there a black dot indicating the remains of an Axis Cylinder.

In the bundle farthest off degeneration has also occurred, but is not so advanced, a few healthy fibres still exist.

The various degrees can be well seen in the original section, but the micro-photograph fails to bring them out very clearly.
Transverse Section of Right Optic Nerve. (x 250)

Advanced degeneration, most of the nerve fibres have lost their axis cylinders, here and there throughout the section a dark spot indicates the remains of one less degenerated than the rest. The remainder of the section is a mass of vacuoles some containing degenerated fibres as a homogenous mass.

The endoneurium is sclerosed and wrinkled bands run across the section.
Case of Beri-beri with marked Hyperaesthesia and Laryngeal Paralysis.

Name: LOK LOY
Age: 24
Occupation: Coolie
Nationality: Chinese--Kheh.
Arrived: Feb 1909

HISTORY.

April 10 1900. Admitted to hospital with mild attack of beri-beri, improved rapidly and went out May 23 to do light work, as the heart still somewhat excitable. For the next 6 weeks he did not come near hospital.

July 1st he came complaining of fever and headache, his temperature was 102 F and he was admitted. The knee jerks were present and there was no complaint of numbness.

Three days later however he complained of pain in the skin of the legs, the knee jerks were much diminished, and there was a trace of oedema over the tibias, the calf muscles were not hyperaesthetic as far as one could make out.

July 9th considerable alteration in the condition, there was paraesthesia amounting to anaesthesia, symmetrical on both sides and rising to two inches above the knee, above this there was a zone of acute hyperaesthesia, the knee jerks were lost, but the arm jerks were present. There was no increase of the oedema.

The cardiac condition on admission was satisfactory, all the sounds were closed and regular. \[ \frac{11}{2^3} \]

Max: Syst: Press: = 120 mm of Hg.

Pulse Tracing--no change.
July 12th the zone of hyperaesthesia had spread up to the Manubrium Sterni, the slightest pinch or touch with a pin causing acute pain, the numbness passed up to the mid-thigh. There were no cramps. The arm jerks were lost and there was marked weakness of the extensors. The heart was considerably more rapid and excitable in action, though still regular, over the pulmonary area the 1st sound was replaced by a blowing murmur propagated downwards. Some enlargement of the Right Heart. $\frac{2/3}{3}$

This condition became progressively worse, the weakness of the muscles more marked and the cardiac condition more and more unstable, until dyspnoea set in.

July 19th has been getting steadily worse at 12 noon today he was breathing rapidly and with great difficulty, Resp = 42, Pulse = 136, Temp normal, Shoulders moving spasmodically, face white, pasty and puffy. Voice inaudible, heart irregular, very rapid with wild precordial pulsation. Skin of Abdomen, Thorax and Arms acutely hyperaesthetic. Right side of heart much dilated, venous pulsation in the neck. $\frac{2/3}{3}$

Other symptoms were not marked, the tongue remained clean, the bowels regular, slight oedema about the legs and face.

Urine no abnormality except for reduction of the Chlorides (Amount 23 oz), no Indican present. Blood examination shows slight anaemia no leucocytosis, but an ever increasing period of coagulation, having gone from $3\frac{1}{2}$ to 7 min in 3 days.

Patient was bled 30 oz and transfused 25 oz NaCl Sol: followed by Nitro-glycerine gr 1/100th every ½ hour.

At first after the Nitro-glycerine, there was a great
difference in the pulse tracing and he lay quietly instead of trying to toss about, but the improvement was only evanescent.

July 21 patient died, remaining conscious to almost the last.

NOTES on the CASE.

The points of interest in this case are the acute hyperaesthesia of the skin, the slightest touch causing agony and the terminal paralysis of the laryngeal nerves and muscles. This may have been due to pressure on the Right Recurrent Laryngeal nerve by the dilated right heart, but it seemed at the time to be more likely due to a toxic degeneration of the nerves themselves, and subsequent histological examination of the nerves proved that they were in a state of degeneration. The initial temperature was also of interest, it is said to occur in Beri-beri, but in my experience is very seldom seen. Lastly note the retention of consciousness right up to the closing scene.

Appended are:

1. Notes on the Post-mortem examination.
2. Temperature Chart showing initial rise.
4. A progressive series of Pulse Tracings, which are rather interesting as showing the increase in Rate passing on to Irregularity.
Post-mortem Notes.

At 9 30 am 12 hours after death, weather cool and damp (T 84). Rigor Mortis complete, no hyostatic congestion, body well clothed, pupils equal and medium, veins contain fluid blood.

Adhesions between Pericardium and Diaphragm, and Left Pleura and Diaphragm. No pericardial effusion. Heart considerably dilated. Rt Auricle and Ventricle contain antemortem clot extending $1/2$ in: into Pulmonary artery, Pulmonary orifice incompetent, Aortic competent. IN section Rt Vent: wall thined, not friable, left Vent wall some slight fatty infiltration, no endocarditis. Coronary vessels and Aorta healthy.

Lungs pale, fully crepitant, in section passive congestion on both sides.

Abdomen. Liver and Spleen enlarged, firm, congested.

Gall Bladder contains fluid bile. Stomach dilated contains large quantity of inodorous fluid deeply coloured with bile, muc: membrane pale, except close to pylorus where there are several petecial submucous hemorrhages.

No erosion of muc: membrane, Duodenum no congestion.

Muscles pale and flabby, especially of calves.

No oedema of glottis or of the subcutaneous tissue.

A few enlarged glands in the groin.

Inoculation Experiment.

The serum obtained from bleeding the patient was injected into a guinea-pig (10cc), Result animal seemed unwell for about a week, but developed no paralysis or even weakness of the limbs, and quite recovered.