Sheets on Bëi Bëi
FRM Atkinson
Beri-Beri.

Its etiology, pathology, symptoms, treatment.

As surgeon in the Peninsular and Oriental Steam Navigation Company, I have had under my care cases of this complaint. I have seen others in many of the hospitals in the East. It is for this reason that I have taken Beri-Beri for the subject of my thesis. Indeed, hardly say that I can only hope to look at the disease from the clinical aspect, as post-mortem examination on board ship is in nearly all instances out of the question. At the same time, in order to make my thesis as complete as possible, I have studied the pathology, chiefly gleaned from Pettenkofer's earlier work.

Derivation of the term. Berchies, one of the earliest writers on the subject, called it Berchies. There can be little doubt that both diseases are identical. It has also been called
"The bad sickness of Ceylon" "The sleeping sickness of Africa" "Acute orchedia of Mauritius".

Cairns suggests for its derivation other 'Syncope' than 'a sailor' hence Brillieu would mean sailor's athen. Others, as Hinchlott, suggest the third instance word thiri, a sheep, as the joint oparous suffering from this disease, is supposed to resemble to some extent, that of this animal. In Japan, it is called hoitski it also Rakke. The most likely derivation seems to be from the Sinhalese word teri meaning 'weakness,' the repetition of the word 'junct weakness'.

Brillieu chiefly subject to Brillieu. The natives of India Throughout Hindustan, on the Persian Gulf are especially liable to it. It is common in Burmah, the Malay Peninsula, China, Ceylon, Japan, Africa, Mauritius. It has also been noticed in Australia & South America.
Europeans were at one time said to be rarely, if ever, affected so much as that they were. Gen. Maclean states, that he never saw a case in a European during his twenty-two years service. In India, &c.; Gen. Moore's experience is the same. Malcolmson had seen a few cases in Europeans. There is no doubt now that they are attacked not unfrequently. In individuals, it has been stated, once ever attacked immediately on their arrival in an infected locality. Six months' residence has been supposed to have sufficed. The necessary period of infection, but since Gen. Moore has seen it after six weeks' residence among Sepoys in the Persian Gulf and others in Rajputana.

Hygiene. Preceding Causes.
(1) Nationality, just referred to.
(2) Age. Very seldom occurs under adult life. Rennacy Roy, out of
Six hundred ruddle cases only observed in three children under seven, 50 under four years old. Dr. Crichton saw a case of it in Ceylon in a woman, office, a boy under twenty. Dr. Takahashi of Japan, has seen it in children of both sexes at young age.

3. Men are more susceptible to it than females. Women, after childbirth, are said to be very apt to suffer from it.

4. Inadequate ventilation of a room may lead to it by predisposing to denunciation of health.

5. Meteorological conditions usually become epidemic at the fall of one monsoon, the setting of another becomes epidemic in the wet monsoon.

6. Conditions spoil the world. It was stated not to extend inland more than sixty miles, but it has been seen in Rajasthan and elsewhere. Said the excerpt above.
two thousand or three thousand feet, but this is denied by D. Vallance, who states it can occur at any altitude. Insomuch in an infected locality may predispose to it, by deplacating the system. In fact, it may be stated generally, that any thing which tends to lower the vitality of the system, such as physical exhaustion, deprivation of food, debauchery, or will predispose to Ber. Biri.

Exciting causes.

Rodgers looked on it as a droplical affection beginning in the chest, malarial as a rheumatic affection beginning in the spinal cord, the dropsy paralysis being secondary results.

Scott a modification of acute dropsy.

Ranching a form of small anaemia, and there is no evidence of this, I have tested the urine in a dozen cases with nitric acid in the cold.
heat nitric acid or sulfuric acid never found albumin in any instance. Indeed also, that Dr. Bentley only found a trace in once case out of fifty he examined. This new must therefore be abandoned.

Chevers a specific fever fever.

Sir Guyart Hensil a result of malarial ascobic cachexia combined.

Macleod, M.R.C.S. Maclean a form pecular. I cannot hold with those who believe that Brem Brem is a form of scurvy. There are no sign of ascobic tendancy in Brem Brem patient, save maybe in slight spotting of the gums (a by no means common occurrence). This sign of itself is not sufficient demonstrative in coming to the conclusion that scurvy is present. Again, the better supply of proteins provided for the sailor cross a deep sea, the diet which contains anti-scurvy food has not prevented outbreaks of Brem Brem, although the occurrence of scurvy is much less prevalent.
Damp moisture. The outbreaks which occurred on board the SS "Ryujō" "Tanlue" entirely negative. I am inclined to think that damp and moisture are far the causes of their death. The following is a table of their voyages: for a fuller account see Dr. Bentley's book. "Ryujō" from Dec 19, 1882—Jan. 31, 1883 Rainy days = 3
Kakké case 3
Feb. 1st—Mar. 31st Rainy days = 8 Kakké case 7
April 1st—May 31st = 3 25
June 1st—July 31st = 0 125
Aug. 1st—Sept. 15th = 1 0
Total 271 days voyage rainy days 15 Kakké case 0 60
Tanlue" Feb. 3rd—Mar. 31st Rainy days = 7 Kakké case 5
April 1st—May 31st Rainy days = 2 Kakké case 7
June 1st—Aug. 1st = 5 1
Aug. 1st—Sept. 30th = 3 3
Oct. 1st—Dec. 16th = 2 0
Total 287 days voyage Rainy days = 19 Kakké case 0 16

Hence, therefore, that during the voyage of the "Ryujō" (271 days), there were 15 rainy
days & 160 cases of Pernic. During the voyage of the "Tsukuba" (287 days) there were 19 rainy days, but only 16 cases of Pernic. I think, therefore, from these results, this view must be abandoned.

Brig. D. Rowell of Singapore, came to the conclusion from his experience during the outbreak at Singapore prison, that lack of nitrophenous food distinctly predisposed to the disease by causing deterioration of the blood. Hence a greater susceptibility to the poison, for he found that when the carbohydrates in the prison diet were decreased nitrophenous elements substituted. Pernic became less prevalent. Dr. Anderson of Japan, also on the other hand, that the coastie claps who eat more rice than either the 80 Wei or sailors suffer less than the latter.

Dr. Macleod says Major White stated, that this diet has nothing to do
with its production.

D. Simmons of Yokohama, found that those who can afford good and abundant food are most liable to the affection.

D. Takaki, of Tokyo, believes that the only cause of beri-beri is lack of nitrogenous food. His views briefly are as follows.

The reason why Japan beri-beri is more prevalent in summer than in winter is that very little nitroenous food is eaten by the Japanese during the summer months, the diet consisting chiefly of rice. It is hot water. He says that this prevalence of beri-beri is not due to the warm weather, D. Takaki, mentions the fact that in one of the islands in the north of Japan, beri-beri only occurs in winter, because in summer the diet of the natives is raised with fish off-aue in addition to rice or, whereas in winter, the seas and lakes are frozen over, rice is almost the only
article of diet.
The susceptibility of women after child
birth is, he considered, owing to the
custom of Japanese women to live
at that time entirely on rice. In
his own practice he feeds them on
barley water, meat. Since she has
not seen a case of milk fever after
parturition for years.
As far as my observations are concerned, I cannot believe that diet
has anything to do with the
production of this disease.
On board the Peninsular and Oriental
Steam Navigation Company's ships,
the patients that came under my
care were all freemen. The diet
could not be blamed for this,
with freemen the cases are given
exactly the same found, yet no
cases suffered from the complaint.
Again, the diet supplied contains
ammonium food as well as rice,
yet they still get the disease.
Their daily diet is as follows.
Rice 18
Shall 14
Shee 2
Salt 8
Curry Sauce 10
Onion sauce, 8
Vegetable 8
Sea ½
Sugar 1½

Salt treatment & Volunteers required.

An impure state may act as a predisposing cause, but there have been no facts brought forward to prove that it is an exciting one.

Exposure to cold. This is, I consider, the commonest cause on board ship. During the hot nights which frequently become cold in the early morning, most of the sailors lie on deck, hence the prevalence of the disease amongst them. This cause seems the only satisfactory factor in the reason why the seamen have only come under my advice suffering from the disease.

The work of the bascaro is finished some hours before bedtime, and therefore retire to rest quite cool. The seamen, on the other hand, come out of the engine room at all hours of
The day I might hot surprising and immediately throw themselves down on the deck, hence their greater liability to the disease. Another also that on board the "Ryuyo" all the stokers were down with the disease, which still further goes to support any contention that exposure to cold is an unimportant factor in the production of Bijin Bijin. This reason will also sufficiently explain the circumstance that in Japan, Bijin Bijin is more common in summer than in winter, when sleeping out of doors is of common occurrence. At the same time I do not consider that exposure to cold per se is sufficient to produce the disease.

Malaria. Mr. Joseph Townson, D. Bentley, and many others consider it a form of the malarial poison. I think there can be little doubt that the poison exists in the soil & is propagated by the air & perhaps fomites, but I do not consider that the same poison causes both malarial and
Beri Beri. The symptoms of the two diseases are so unlike that it is hard to believe that the same organism can produce such totally different complaints. The milder form of fever cannot produce measles, whereas can typhoid fever. Both diseases which resemble each other much more closely than malaria and Beri Beri.

Again Beri Beri is essentially a non-scarlet fever which is a further instance of the non-similarity with malaria.

Lastly, I would lay stress on the fact, often proved, that cold-cutting monsoon will produce epidemics with malaria and Beri Beri, but it is extremely rare for both epidemics to break out at the same part of the state, at one part malariaprevails and at another, Beri Beri will prevail. I think, however, that Beri Beri and malaria may be said the analogous in that the incidence of the former is apt to be increased by all the agents.
necessary for the production of the disease. From these considerations, it will be gathered that I hold strongly to the opinion that there is only one exciting cause, a microbe peculiar to this disease in Bini and in the same alone, propagated from the soil obtained by the air (perhaps from the air), but especially attacking those exposed to cold (the commonest predisposing cause on board ship) or in any way debilitated from previous disease.

Microbe Theory. Dr. Lacenda was the first to conclude from observations that Bini Biri was due to a microorganism, which, he stated, resembled that found in rice.

Since that time many observers have worked at this side of the question with various results, for which I am chiefly indebted to Dr. Bentley, Mr. Pitharkland, and Whipple's works.

Onaka of Tokio describes a schizomycete in the spinal cord, nerve, and blood. The bacilli are smaller than those found in malaria, chiefly straight, forming spores.
generally wounded. In a later stage of the disease, the \textit{organism} shows a cloudy appearance, making a ring round the wall of the tube. In a later stage, they appear as a vigorous cultivation on the surface as well as in the interior of the substance. The superficial colony was sharply circumscribed, irregularly defined, yellowish white in colour, lustrous in appearance, smooth, but later becoming faintly wrinkled. The organism did not grow at room temperature, mice and dogs inoculated died after from 12 hours to some days. Death apathetic, spastic in parts, hind feet swollen. Leopold found (1) \textit{Staphylococcus} profunco aureus, (2) \textit{Micrococcus} chain form, (3) A small \textit{Streptococcus}, colorless, fungus on character, difficult of cultivation, (4) \textit{Micrococcos}, which by inoculation in animals, causes \textit{pneumonia}. Dogs cause a defibrinative hemorrhage, and is
described as the typical micrococcus of Berkeley. Intracerebral injection of the micrococcus produced necrosis, paresis of limbs, atrophies, atrophy of muscles, especially adductors, stiffness in consistence while Pekelharing's observations are as follows:

Organisms were never found (16 cases) in any of the organs save in two cases when large bacilli were found in the heart. The blood was examined in the living subject of granules. Rods were found in most cases. The rods were not uniform either in length or thickness, sometimes irregular. The granules were spherical in most cases, but in some elongated ones were met with. Granules and rods were frequently clustered together in the former resembling diplococci. Rods were often divided into two seemed to have the power of movement. These bacteria were considered to have some connection
with the disease as they were not found in a healthy person who had lived for years past in a non-Beri-Beri district on the day of his arrival at Atjih, but after a residence at Atjih of a few weeks. Both granules and rods were found in the blood. Again, they took observations of the blood of five people, four healthy, one ill with Beri-Beri. The blood of all these contained bacilli whilst at Atjih, but could not be discovered when they returned to Batavia.

As regards the cultivation of the bacillus: in fifteen cases, hitherto showed only cocci; three rods like bodies. The rods differed in each specimen. Since there were some bacilli with a characteristic ornament, although in one of the two cases they were much smaller than in the other. The third species of rod was very peculiar. In all the plants, in those which developed on a solid
I substance as well as those which developed in a liquid, these rods, which were of very different lengths, which were often not perfectly cylindrical, but a little broader in the middle or at one of their ends, alternated with some spherical granules which presented considerable differences in their diameters.

Cultivated on agar, agar, these bacteria formed a thick bed with a shiny surface, at first white, which soon became reddish, after a few days thickened in colour.

In the twelve or twenty cases, micrococci were found; they developed best on the solid substances, forming a white bed with a shiny surface in ten cases, but in both they were of a yellow colour.

Amongst the ten cases in which white granules were obtained, the were composed of a single colony of micrococci. The members which differed partly from each other inside.
The different colonies developed on gelatine did not all resemble each other. On one occasion seven colonies were found, two of which formed white or fine, formed yellow from this. Four of these latter, had a yellow, brown but on the micrococci generally had an elongated form, while the fifth had a yellowish brown, two formed micrococci larger than the others, arranged for the most part in bands of four. This last form agreed with those we had obtained in the above cases, where the blood gave rise to two colonies, in the other three colonies, all of micrococci, which had a yellowish brown, which had the tendency to the formation of bands of four were noticed.

These observations were unable to come to conclusion whether these micrococci were varieties of the same species or not. They came to the conclusion that the
white micrococci are the cause of the disease; as a result of experiments on our rabbits and dogs, they found that injections of these micrococci produced an emetic identical with Bini-Bini. They state it is difficult to find in the blood extracts probably gain access to the system by the respiratory tract and to destroy in the blood but not before it has had time to start anemia. The micrococci live as a saprophyte upon all kinds of nutritive substances provided there is a sufficient supply of oxygen that the temperature is not too low. Heat and humidity both seem necessary for its growth; it also possesses great power of resistance can be carried by clothes or otherwise, hence its frequent appearance at sea. Further observations are necessary before it can be certain that the white micrococci described by these observers is the true microbe of Bini-Bini.
Anaemia, though considered that
Beri Beri was of the nature of a
pernicious anaemia owing to the
following considerations.
The blood in Beri Beri he believed,
(1) did not form rouleaux
(2) in many instances presented
spurious margins intermingled
with which were a number of faintly
colored minute bodies frequented
and lastly, there are met with in the
blood large, peculiarly brilliant
masses of doubtful significance.
It has since been discovered
(1) that the corpuscles do form rouleaux
(2) to more spurious forms
exist that are always viable in
rapidly dried blood
(3) the large brilliant masses are
simply composed of an agglomeration
of blood corpuscles.
(4) that anaemia, though common,
is by no means always present in
Beri Beri. Hence, the new must be
abandoned.
Varieties of Biên-Biên. Biên-Biên was originally classified into three types: (1) Acute (2) Subacute (3) Chronic. When the disease became better understood it was found that this division was unsatisfactory, and it is now more to describe the following varieties:

(1) Acute Dystrophic.
(2) Atrophic or paralytic.
(3) Mixed.

Which have been added:
(1) The opsomotic or convulsive.
(2) The Acute Pernicious.

The acute dystrophic is distinctly characteristic for prominent edema, but paralytic symptoms are also well marked.

The following case will illustrate this type:


31st. Came home complaining of pains in the calves of the legs, shortly after
health, difficulty of walking. States he has been ill for ten days with fatigue, inability to do hard work.

Condition is as follows. Conjunctiva: a) Pupils pale, the latter slightly tender to the touch, but do not bleed. Ankle and legs swollen especially over the knees where the skin is compressed, face puffy especially under the eyelids. Temp: 97.8. Tongue pale, covered with a white fur; appetite good. Faeces congested. Bowels confined. Abdomen swollen: dull in the flanks, tympanitic in front when the patient lies on his back. Liver normal. Anemia well marked.


Breathing 25 per minute. Abdomen:

Thoracic: slight dullness at the base of lungs.
Urine dark. Specific gravity 1022. Urates, uric acid

Genito-urinary phenomena. Feeling opaqueness
in the left. Anaesthesia over the
dorsum of the feet, extending up to the
knee, a limb deep in the right leg,
not going beyond the knee and ble in the
left. Anaesthesia over the backs of the
hands, tips of fingers.
Hyper-aesthesia well marked over
the calves of the legs on deep pressure.
Gastrocnemius bulge considerably.
Sensation lost. The others intact.
Gait staggering; on walking he keeps
his legs apart when taking a
step, his feet up and away
as if they were stuck to the ground.

Jan. 1st. Eye: staring. His anaesthesia
of the face, deep. Temp. 97.6. Pulse 110.
Bedroom of the legs increasing. Hands
and feet swollen. Heart dilated to
the right side of the right margin of
the sternum. Very sensitive to touch.
Condition at nurse the same as yesterday.

2nd. Breathing 30 per minute.
abdominal than thoracic. Pulse 112. Temp. 97.

Great difficulty in walking. Abdomen more swollen. Pulse 85, in the lungs and funding.

3rd. Forehead increasing rapidly. Difficulty in breathing.

The lungs extend up to 8th rib posteriorly on left side, 87° on the right side.

Abdomen dull all over, save for a small area round the umbilicus. Pulse 114.

Deep, gurgling, deep more or less. Patient has stopped up in bed breathing rapidly.


The deep breaths extend to the third rib. Pulse 130, very feeble.

Deep, 87, died at 2 p.m.

The above was chosen as the best case under my care of sedentary or optical

Dini Ben. It shows the rapid progress of the disease, not alleviated by the

usual remedies.

The atrophic or paralytic form consists chiefly a marked atrophy of the

muscles, rendering the patient almost
helped sometimes, in a very short time.
Oedema is usually very slight, if present
at all. The following case will illustrate
this variety.

**Rwan Chekh, Male age 23. First seen
April 12th. Sent to hospital April 20th.
**

Complains of fever, slight swelling of the
legs, numbness of feet, pain in
the calves. The deprivability to walk
without difficulty.

Condition is as follows: No oedema in the
face. Slightly anaemic conjunctiva,
loose coarsened with a white fur. Face
congested. Appetite poor. Bowels
constipated. Anaemia slight.
Pulse 110. Heart thumping feeble.
Duplicate 2 & central sound.
Increased cardiac dullness to the
right side of sternal margin, extending
1 ½ inch to the right of the right side
after margin. Apex beat best felt
between 7 & 8th ribs. Opposition
palpation. Lungs normal. No
rattles. A few phosphates.

**Genus organisms. Anaemia will**
marked over the dorsum of the right foot extending up to the ankle. No anaesthesia in the left foot. Arm normal, walking difficult. Decrease of pain after the reflexes intact.

The paralysis became more marked when he reached hospital, his condition was as follows.

All the systems were the reverse, remained about the same. Haemorrhage over the fibriae disappeared altogether.

Nervous. Anaesthesia extends up to both knees, but is more marked in the right leg than in the left. Above the knee, sensation is normal. The feet are inverted, when the patient hangs his legs over the side of the bed. He cannot get up from the sitting posture as his legs cannot support his body. He is unable to walk altogether. In the upper extremities, sensation is lost in the tips of the fingers, but is slightly felt in the tips of the right thumb. There is marked anaesthesia in both hands.
do deep urin. In straightening the
hand does flexion of the fingers at
the metacarpophalangeal joints
occur. The gap of the right hand is
depth than the left, tendon reflex lost.
The spinothalamic reflexes also lost, the
other reflexes remain intact.
No thrilling, twitchings, or diurnal
contractions. No areflexic pappen
ship. The muscles of the body are
weakened, especially the calf muscles
which are tender to deep pressure.
There are felt circumscribed
swellings. The patient is very sensitive.

On seeing the patient a few weeks
in hospital, his condition had
considerably improved. The
areflexia in the lower extremities
was only present in the foot of the
right leg. An abnormal reflex noted
above the ankles in both legs.
The upper extremities remained the
same. The muscular power had
increased, so the patient could stand & walk unaided. In walking, the legs were kept apart & the feet brought off the ground with a jerk; when touching the ground again, the heel was brought down slightly before the toe, which was pointed upward.

The reflexes remained the same. The muscles were still wasted & hypertrophied; no trace of muscular twitching could still be detected in them. These cases generally recover, but recovery is usually slow.

Dystonic form. This is the commonest form met with. Dystonic and paralytic symptoms are both well marked in these cases.

Spastie or convulsive. I have never seen a case of this type, yet seen. It is extremely rare, notwithstanding Dr. Bentley's views to the contrary. Paralytic symptoms are well marked, preceded in most instances by spasm of the muscles of the arms, legs, back, some or all, & sensation in
Acute pernicious. This type usually gets in at the end of a case suffering from one of the other varieties. The prominent features in this form of the disease are marked collapse, great failure of the heart's action, progressive dyspnea. There is frequently some fever. Edema is usually slight. Paralytic symptoms are prominent. It was well seen in the following case:

**Mohamed Hoosain age 30. First seen June 29th. Died July 2nd.**

Transferred from 55 'Australian' 65 'Roxton' at Colombo. Received no previous history of this case. State he has been ill for four days. Always commenced one morning with chills, fever, pain in the calf of the legs, a high pain in the muscles, slight difficulty of locomotion, with difficulty in walking. Slight oedema was noticed over the tibiae.


This type is the most dangerous of all, nearly always ends fatally.

Symptomatology. Before the symptoms of Bini. Bini became well marked, the patient has usually suffered for a shorter or longer time from premonitory symptoms, which chiefly consist of asthenia, inability to perform slight pain feeling sharp knaps in the calves of the legs, perhaps also a slight oedema over the tibiae or under the ankle may have been noticed, with these maybe a little fever, which may or may not be ushered in by rigors.

These symptoms may last for a week, fortnight, or even one or two months, before the initial stage commences.

This stage begins by marked paresis
in the back of the calf of the legs, with inability to walk freely. There is also numbness in the legs usually preceding a localised oedema over the site when the skin feels pressure. The oedema plateaus on extension over the abdomen. The upper extremity is followed by anæsthesia and paralysia with hyperæsthesia of the muscles. The body then begins to swell, the abdomen becomes full and tense. Dyspnœa marked: the pulse becomes quick, the heart bounding with thumping, a feeling of approaching death. If the case is going on to a fatal termination, the dyspnœa becomes more and more urgent, the pulse rapid, widely dilated, the straining muscles of inspiration are brought into play, the face becomes livid, the extremities swollen and cold. The whole body bloated the patient soon dies of suffocation. Such generally speaking are the
symptoms of Bēn Bēn. All these symptoms, however, do not necessarily exist in every case. In some the oedema may be slight; paralytic symptoms most marked; in other the opposite condition is met with; in others, again, spasms of the muscles of the body especially the back rendering the patient almost rigid are chiefly noticeable, but this form of the disease is rare.

If the acute symptoms are recurred from the patient may pass into the paralytic stage or recover completely.

Details of symptoms. Oedema. Oedema nearly always appears after the nervous symptoms is by no means present in all cases. In some it may be absent altogether, in others slight, in others it is the most prominent feature. It usually begins in the front of the face and spreads to the
fat, extending also to the back of the hands & finally affecting all the * subcutaneous tissues & muscles. Oedema of the face is common especially under the eyelids, usually appears later than the oedema of the lower extremities. Alimentary system. The appetite is usually good save in the delirious form where it is impaired. The tongue is furrowed, the bowels confined, diarrhoea being uncommon. I have frequently noticed the faeces, composted as Dr. Bentley originally pointed out. Gastric disturbances especially vomiting are dangerous omens, and generally usher in a fatal termination. Vomiting is probably due to affection of the terminal end of the vagina. The liver may be enlarged, but is usually normal. Haemopoetic system. Anaemia is frequently present, but it is not uncommon to find it absent.
Circulatory system. Palpitation is nearly always present. The patient commonly complains of a thumping feeling over the apex beat. The cardiac dullness is increased to the right side and the heart is faintly dilated hypertrophied. The first sound is prolonged and the second especially at the apex, reduplicated. Murmurs, chiefly asystolic ventricular, are not uncommon. There is a marked contrast between the painfully beating heart and the feeble pulse which is not usually fast when the patient is at rest, but on the slightest movement reaches 90, 100, or more beats per minute.

Pericarditis may occur as a complication at the termination of a case. The heart may dilate very rapidly, sudden death from cardiac paralysis being a common ending of the disease. The heart signs just enumerated are to a better or left apex, met with in every case of Bini Bini, and are one
The constant feature of the disease. The cause of the rapidly beating heart is probably an affection of the cardiac nerves which become implicated in the same way as the other nerves in the body.

Respiratory system: there are no signs in the lungs of any change until the disease sets in, when they become affected with the rest of the body. There is then dyspnoea, rapid breathing, and all the signs of effusion. Sudden death from respiratory paralysis is a common ending in a case with the other evidences of atrophy of the respiratory system.

Integumentary system: the skin over the fihrae is frequently shiny. There is often a scaly eruption over the body. Sensitivity to cold is nearly always present, causing in some cases cutis anserina, fall of the hair shaft and modifications in the functions of the skin, due to anomalous influence in many instances.
Urinary system. The urine may be acid or alkaline, usually acid.
The specific gravity varies from 1010 to 1020 or more. It may contain phosphate,
crystals, or an excess of chloride. Albumin is rare.

Sensory system. Sensory phenomena.
The sensory disturbances in some cases precede the motor; in others, the
motor precedes the sensory. Reducing
the sensory symptoms, the first
thing complained of is usually a
feeling of numbness which may be slight local, or more marked and
general. The numbness is often accom-
panied with a feeling of pins and
needles or the pain may become
more intense, so severe indeed, that
the sufferer shudders when anyone attempts to touch him.
The pain may be of an acute character,
but is usually of a dull, aching,
numbing kind. It is most marked
in the muscles of the legs; it awakens,
increased at night, and by walking
or making a movement of any kind. It is also aggravated by pressure. The numbness is followed by anaesthesia of the skin, hyperaesthesia of the muscles. These disturbances are first noticeable in the lower limbs, especially in the calves of the legs, then extending to the hands and arms and finally to the body generally.

In the face, the commonest place to find anaesthesia is in the upper lip, but I have not found it so common as Dr. Bentley would lead one to suppose.

Paresthesia. These frequently resemble those of locomotor ataxia. The conduction of nervous impulses is markedly slow. The prick of a pin is often not felt for some time after application of the stimulus. The pain may then be felt in a different spot that prick. Allolchna has not been observed. Cold and hot tubes applied to the skin are often mistaken for cold.
being called hot, or ice numb.
Gentle but not lightning pains have
been noticed in some cases.
Sensation is usually lost in the
following order.
Internal aspect of calf, dorsum of foot,
internal aspect of leg, sole of the feet,
outer side of leg. The skin of the skin below
it never seem to be sensitive.
In the arms, 1st Anterior aspect of wrist,
thenar hypotenuse, venae comitantes, proximal
phalanx of thumb, little finger,
fourth finger, index finger, palmar
of the hand and digits of fingers.
Internal
aspect of forearm, inner side of elbow,
joint. On the chest there may be a
square anaesthetic patch which may
reach below the umbilicus. The
skin of the back never seem to be
affected" (Shelburne Hinkle, 1893.)

Motor disturbances. Motor symptoms
neuro occur alone, they are always
accompanied with some of the
sensory disturbances previously
described. The motor phenomena
are involved in with twitching of the muscles of the leg, arm, or body. In some cases these twitchings last throughout the illness, in others they pass into the paralytic stage. An entire limb is never affected, some of the muscles being attacked, others remaining unaffected. The muscles are attacked from the periphery, especially the tendons giving rise to drop and foot. The opposite leg and arm never seem to be affected at the same time, though Dr. Bentey cites a case in which the patient was found to be suffering from drop and of the left arm, followed in twelve days by the same condition in the right hand. There is always marked wasting of the muscles involved often followed by contractures. In some of the other muscles marked bulging with circumscribed swelling in the substance of the muscles is nearly always present. The swelling is due to enlargement with degeneration of the
Muscular fibers will show effusion in between them. Idio-muscular contractions brought about by shifting fibers heavily have been seen but not fibrillary twitching. In tendon reflex there is no increase of tendon reflexal test; lateron it is lost; the plantar reflex and knee reflex may also be affected, but the cremasteric and abdominal area rarely affected. Hypertension has been noticed in many cases. Vasoconstriction phenomenon. There is no tendency to the formation of bed-sores. Coldness of the extremities and pretensionness to cold generally are common features of the disease. The rapid osenna metrict in the complaint has been considered, not without probability, as due to changes in the vasomotor system. Fall of the hair, beard with modifications in the secretion of the skin are common. Acute swelling of the submaxillary, parotid glands occurs sometimes.
at the end of a case of serious significance. They are probably due to parasitism influence.

Temperature. I have included temperature under this heading as it seems very probable, as Dr. Bentley suggests, that the great varieties of temperature met with in Bêl Bêl are due to parasitism influence. Bêl Bêl is typically a non-febrile disease. The temperature is usually subnormal, as low as 94° F, having been noted. The acute pernicious variety is the type in which a rise of temperature is most commonly met with. It has been known to rise as high as 107.2.

Electrical phenomena. The value of electricity has lately been visited upon by Petelhauen, and I cannot do better than quote their own words regarding it. "The initial phase of Bêl Bêl is characterized by quantitative..."
changes consisting of a diminution of irritability generally for the two kinds of current. Very often also qualitative modifications of the electrical reactions of the dorsal muscles of the foot characterized further by an augmentation of the diameter of the tactile zone at a fixed point in the leg.

These changes are "suppression of irritability." The Faradic current whether applied directly or indirectly to the muscles supplied by the external and internal popliteal nerves. Considerable diminution of the indirect muscular irritability by the galvanic current is still greater diminution of its direct irritability. In addition, a contraction, more or less slow, which appears when the current is uniform, at the clamps as well of the anode as of the cathode, characterizes the deviation as
Qualitative. It is evident, therefore, that in Bini Bini we have a special form of the reaction of partial degeneration.

"That which is particularly peculiar is the considerable amount of diminution of the irritability of the muscle to the galvanic current, a condition exactly opposite to what one sees in a complete peripheral facial paralysis. In the mixed forms electrical modifications are at times very diverse; they may consist only of a simple diminution of irritability of the nerves or muscles, affording a non-typical reaction of degeneration, although the typical partial reaction is not wanting. In atrophic Bini Bini, on the contrary, typical partial reaction, or reaction of complete degeneration, is the rule."
my characteristic. The leg is brought up off the ground with a jerk as if the foot was thrust to the ground. The foot is brought down again almost flat, the heel, however, touching the ground slightly before the toes which appear to be shot upwards. In the later stages, no particular pain can be said to be peculiar to Bell's Palsy.

Muscles. The muscles supplied by the external popliteal nerve always show degenerative changes. The flexors of the knee are commonly affected and with them the extensors of the knee & the plantar, but flexion of the knee can always be performed. The adductors & flexors of the thigh are usually the last to be involved. In the upper extremity, the extensors of the hand & fingers are the first to lose their power, later, on the triceps, flexors of the hand & fingers, the triceps and pectoralis major are usually the last muscles to become involved.
The trapezius, sternocleidomastoid, and rarely affected. The facial nerve is frequently attacked. The muscles around the mouth, the orbicularis oculi, palpebrarum, the frontal, rectus, and orbicularis oculi have all been known to have been invaded. Difficulty in swallowing has also been observed. The Buccinator, the lip of the tongue, have suffered in some instances. Affection of the hypoglossal nerve is very rare. The extracortals and diaphragm are always affected in the severe paralytic states. Death from paralysis of the laryngeal muscles is not uncommon. The sphincters never seem to be involved.

Pathology. Naked eye appearance.

As is only to be expected in a disease with so many different stages, and exhibiting such various types, the naked eye appearance, not with very considerably.

The body of a patient who has died from the atrophic form shows marked
wasting accompanied with here and there slight swelling of the muscles of the calves, generally a little oedema over the tibiae. In the acute dropical form, the whole body is swollen, and enlarged, the eyeballs protrude, the lips are livid, the conjunctivae echymoscid, the poplar veins stand out prominently, the face puffy, bloated with all the other signs preceding death from suffocation. Rigor mortis does not usually set in for two or three hours.

In incision, in the acute dropical form, brunn erosion from the muscles subcutaneous tissues; in the atrophic form the muscles show yellowish patches of degeneration. Brunn is found in numerous cases, in the peritoneum cavity, in most cases, the liver may be enlarged and slightly but depressed. The gall bladder is frequently sedentary. The anchylostomata duodenal cicerosphake. As far as frequently
met with in the intestines. The
esophagus may be enlarged. The blood
is of a dull-red color, saturated
with carbonic acid; it has been
stated by some to be tarry, though this is
denied by others. There is nearly
always fluid in the pericardium.
The heart is always hypertrophied;
slightly dilated usually on the right side,
but the left may be also affected.
The muscular coat is pale, and
sometimes shows signs of fatty degenera-
tion. There are usually ecchymoses
on the pericardium, _pleurae_, the
caulis of the right auricle and
ventricle contain clotted blood.
The veins are dilated engorged.
The aorta may show signs of indur-
atration, * Oedema of the lungs is
common. The kidneys are usually
normal, but may show signs of
Convulsion.
Effusion is found in the ventricles of the
brain, on the surface of the cerebrum.
Effusion frequent by exists under the
Demase of the spinal cord. The spinal cord is stated by Dr. Bentley, the always soft, different with hemorhages, markedly edematous. It is, however, denied by Pekelharing-van-Miller.

The meninges are with swollen and atrophied, with hemorhages and between them, atrophied. They may show yellow patches of degeneration: if hyperatrophied, a marble mottling of yellow lines and patches.

The constant naked eye模糊 fluid in the pericardium septum, hyper trophy and dilatation of the heart, is seen in between the muscles. According to some, part engagement of the spinal cord which is soft and edematous.

Microscopic appearance. Granulation, the condition of the nerves in Bini Bini has chiefly been worked out by Pekelharing and Blinks, whose results are as follows.
The nerve fibre has become a little thicker, the medullary sheath is no longer a black cylinder with a double outline, but has broken up into masses. In Ranvier's node, the medullary substance appears to be altogether wanting, but midway between two nodal points it is collected into prominent masses. The necrotic sheath of Schwann remains intact around this substance as if it is agglomerated into masses. In some instances, one finds instead of the internodal nuclei, two or three nuclei imbedded in a mass of protoplasm somewhat swollen. The axis cylinder of the nerve is to be found only in the vicinity of Ranvier's node, while the medullary substance has often disappeared over a large space. In more advanced stages of degeneration, the medullary sheath has almost entirely disappeared, but there are only rosettes of it found in little masses with Schwann's nucleated sheath stretched over them.
Still more characteristic is it to find at certain places, that the nerve fibres are filled with a number of little globules, coloured black, brown, or dirty yellow mixed intimately with a frothy mass. In this mass, or by its side, one always finds a nucleus, generally several are seen. The nucleated sheath of Schwann becomes duplicated upon itself, because of the absence of the medullary sheath substance in the fibre. The contents of this fibre consist of a peculiar mass upon which are seen numerous long, straight lines.

Haemorrhages were rarely found in the nerve. In addition to the signs of degeneration, signs of regeneration are also visible.

The nearer one approaches the periphery, the greater the nervous degeneration, the nearer one gets to the centre, the less distinct is it. In therent nerve trunks in the root, no change are found" a condition
which one would expect from a purely clinical study of the disease.
The cardiac vagal branches of the vagus show the same changes. "The
number of thicker fibres is less than in the normal vagus nerve, if these
have been replaced by a large number of fine fibres."
Roots of spinal nerves. Despite again from the same source, "we have not been
able to ascertain any changes in the anterior roots or at most only
very slight ones. On the other hand, we have received the impression,
that long duration of the disease leads to an atrophy of the fibres in the part
of the nerve situated between the nerve trunk and ganglion of the root.
The peripheral portion in fact of the posterior root. In the ganglion itself,
we found signs of change in cases of long duration, not only so but in
the fibres proceeding therefrom in a central direction along the central
portion of the root. "The columns of Sole."
Central nervous system. The central canal is frequently closed by a development of cells around the lumen derived from the epithelial lining normally existing there. Vacuolating the cells in the anterior horn has been noticed by more than one observer.

In transverse section, the gray substance of spinal cord is often found rich in fibers provided with a medullary sheath. The same is applicable Clarke's column. In the medulla oblongata sometimes a large number of corpuscles may be found in the floor of the fourth ventricle but have no shape or care.

The muscle of the body show all the signs of marked degeneration. So sum up the microscopic appearances met with in the nervous system following show: remark as follows: 'An extreme and very extensive affection of the peripheral
nerves predominate; as one approaches the central nervous system the pathological evidence of nerve change diminishes. The anterior roots of the spinal nerves are always healthy; but in the posterior roots one sometimes meets with a slight atrophy of the fibers which is always infinitely dep on the postural side of the intervertebral ganglion than on the distal prolongation of the nerve. In the spinal cord, some variations of secondary importance are met with in the large nerve cells of the anterior columns, but more constant — still is a slight loss of fibers in the extension of the posterior roots in the two lateral zones, unaccompanied by any swelling of the axis cylinders, from a degeneration of cells or multiplication of nuclei."

We are now in a position to consider whether Bin Bin is a disease due to central lesion or is it a 'multiple peripheral neuritis'?
As far as a central lesion is concerned, it can only be anterior poliomyelitis acuta, or an inflammation of the cord & membranes affecting both sensory & motor nerve. The sensory phenomena met with in the disease are so strongly against its being an anterior poliomyelitis acuta, that we can safely put that view out of consideration. The researches of Bekherman & Hidaka, are all against the view held by Dr. Bentley that Bēri Bēri is "a cord lesion which involves both motor & sensory, functions of central origin, a sub-acute inflammation of the spinal cord & its membranes," a view chiefly based on the naked eye appearance, met with by them in the spinal cord.

Regarding this theory, it does not seem that congestion due to perineal condition of the spinal cord & its membranes are so common as Dr.
Bentley has found. Again, it is generally acknowledged that the ordained in
Biri Biri is always preceded by nervous phenomena. That being the
case, it seems likely that it is set
up by some sort of influence; hence
its presence in the cord may be
accounted for is quite insufficient
to explain the congestion so that Dr.
Bentley describes.

Thirdly, no cord lesion has ever
found in Rickettsian Shrinklers
research. The predominant feature
being degeneration of the peripheral
nerves, but this, of course, is not
sufficient to prove that no cord
lesion exists.

Lastly, from what we know regarding
nerve degeneration, there is no
reason to suppose that if a nerve is
cut off from its centre it will
degenerate from the periphery
to the centre, as we have seen
the chief signs of degeneration to
be found in the peripheral endings.
of nerves, it seems more than probable that Böni Böni is not due to a central lesion but is rather a "peripheral multiple neuritis".

Cause of death. As will be gathered from the preceding pages, death may result from paralytic shock, respiratory muscle or larynx from cardiac emboli, lastly ashen, the two former being the commonest terminations of the disease.

Propagation of the disease. From the general mass of material at our disposal, it seems certain that the body of a Böni Böni patient cannot be considered contagious. This is borne out by the following considerations.

In the hospitals in the East, Böni Böni patients will be seen lying in the same wards with those who are suffering from other complaints, yet none of these latter ever catch the disease. Again, the attendants, on those ill
with Bêi Bêi very rarely take the disease, which is a still further proof of its non-contagiousness. To cite another instance, when the disease was rife in Singapore last year, a number of the patients were sent to many prisons, intimately associated with inmates, yet none of these latter contracted the complaint. On board the H.M.S. Revenge under which I have served, infection can have played no part, as in only instance when one patient has suffered from the disease, it has never spread to anyone else. The fever seems to be spread by the air (perhaps the clothes, but this latter, means of conveyance seems doubtful). It is no doubt increased in virulence by overcrowding in prisons, rooms, tents, barracks.

Diagnosis. The only disease which resembles Bêi Bêi very much is mistaken for it is locomotor ataxia,
that only in the earliest stages.

Here, the premonitory symptoms lasting a shorter or longer time, the pain in
the calves of the legs (perhaps electrical
phenomena), the edema localized
attract to the tibiae, absence of
lightning pains, spastic crises,
and Bigsby-Robertson phenomenon
efficiently differentiate the
case. In the later stages, it cannot
be mistaken for any other
disease.

Prognosis. As regards prognosis, the
atrophic form is most frequently
recalled from them the other
types. Gastric troubles, especially
diabetes vomiting are especially
prominent. Inflammation of the parotid
duodenal gland is generally
when in a fatal termination.
The acute edematous form is
very frequently fatal, but if the
acute stage is passed, recovery
is not uncommon. The acute
pernicious type warrants a very
unfavourable prognosis.

Treatment. The treatment of Bubi Bubi divides itself into two heads, the prophylactic and the medicinal.

Prophylaxis. This is the most important. As the poison seems to be carried by the air and fomites, all sanitary measures calculated to destroy the fomites in a room or house should be stringently carried out. There should be no overcrowding in dwellings. The houses should be thoroughly drained and ventilated. At the same time the clothing of an infected patient should be disinfected, the rooms inhabited undergo a thorough cleansing. This is best done, as recommended on board the P&O ships by corrosive sublimate (in 1000), which should be sprinkled over the walls, floors, and roofs, where the sublimate

Cannot be used, owing to its corrosive properties, purification by Sulphur may be employed instead.

Under these conditions it seems probable that Bëri Bee can be stamped out of an infected building.

The amount of its infection, however, of buildings, will prevent an outbreak of the disease, if the soil is thoroughly contaminated with the forms.

The root of the evil must then be attacked, this can only be done by thorough drainage of the soil. If this is carried out efficiently, there seems good reason to hope that the disease may be considerably decreased.

General Treatment. Open air exercise should be obtained as far as possible, and removal of the patient from an infected district is of vital importance.
A sea voyage often proves of value.

Medicinal treatment, no specific has yet been discovered which will cure the complaint. The patient should be treated on ordinary principles.

If there is much oedema, a hot-bath, hot drinks, citrate of potash (20 p), or citrate of potash (30 p) with a strong purgative (10 p of Colonel, 3 p of salts, jalapine co., or 8 p of saltpetre) are beneficial.

If there are any signs of cardiac failure, etc., should be injected into the heart, at the same time stimulants, as ammonium, etc., digitalis, strophanthus valerol should be given internally.

In the paralytic cases, morphia is the best remedy. Intravenously cimic is said to be of value when depurare is 먼저.

Blum origium is a substance called shrek saroth, the
exact composition of which is unknown. They were at one time said to be of value, but they have now fallen into disuse.

The article of diet should be light and nutritious, milk, chicken, broth, beef, tea, jelly, milk puddings, bread and milk, &c, or alcohol as required. I have found [schematically], in church of digitalis and in the hydrochloride, that the most beneficial remedies are alcoholic.

Local treatment. For relief of the pain in the leg, bandaging from the ankle up, or rubbing with olive oil and chloroform, or a B. C. liniment are the most valuable.

If hyposthenia is present, a blister over the chest proves sometimes grave.

Blister or the cutting to the spine, mustard plaster to the chest, &c, &c. Of the legs are all of value in
Specifically, such is the usual treatment employed in a case of Bini Bini. In many cases, however, all medicine are useless and the physician is as helpless as in the most malignant case of small-pox or scarlet fever.

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