Beriberi or Kahke
an endemic disease of the peripheral nervous system

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During the years 1886-91, whilst serving as Medical Officer of H.M.S. "Merlin," on the China Station, I had several opportunities of seeing cases of a disease which was at once much dreaded and comparatively little understood. It is familiar to everyone in that part of the world as Beriberi or Kakke, but though familiar clinically, I succeeded in obtaining very little information as to the nature of the disease until, on my return home in May 1891, I was able to take up the literature of the subject and study the course of investigations which have, during the last few years, been systematically carried out all round the world — particularly under the auspices of the Government of Brazil and of the Netherlands government in the Dutch East-Indies. The results of these investigations have been (1) a demonstration of the true nature of the disease, (2) it has been shown that at least two other epidemic affections have been denoted "Beriberi," (3) the adoption of rational precautions against the spread of the disease by both the Brazilian and the Netherlands Government.
It seems wonderful that a complaint which was known and written about so long ago as 200 B.C. (according to Hirsch) should have been allowed to remain so little investigated by methods of modern research: as a matter of fact until a few years ago the disease was only commonly seen in remote parts of the world by medical men whose chances of obtaining access to the literature of the subject already existent, or of discussing the phenomena of the disease and comparing notes with others are small.

The study of the disease during the last twenty years has no doubt been immensely stimulated by the sudden spread of the disease since 1860 throughout Brazil, where it was hitherto unknown, as through the Dutch East Indies; by the keen development of the scientific spirit which has been so marked in Japan during recent years; owing to the disease having been recognised during recent years at various places which might have been supposed to be outside the area of its sway, namely, Newcastle on Tyne, London, Paris, New York and San Francisco.
I had but one case under my immediate charge while in the East, which was rapidly fatal, but as I was frequently coming in contact with cases during our travels I became much interested in the subject and so have chosen it as a basis for my thesis.

To naval surgeons the malady is of particular interest, from the fact of its having prevailed with great severity in the navies of Brazil, China & Japan; while an outbreak of "acute epidemic drowsy" which occurred on board U.S.S. "Juno" in 1856, while cruising off Sydney, may have been the same disease.

I have only been able to find records of the disease having occurred on two occasions in this country; one in a case recorded by Dr. Bristowe in his "Diseases of the Nervous system"—a case which he observes parenthetically, might have been taken for a case of diptheritic paralysis had the history of diptheria not been absent & had the diagnosis of Kallike 1st been made by Dr. Suzuki of Japan. The other instance in which the disease has been seen in this country was an outbreak which occurred on board the Chinese transport "Tso Nan"; three sailors
Three sailors died of the disease soon after the arrival of the ship at Newcastle-upon-Tyne, and eleven others were successfully treated by Dr. Oliver of that town, for whose report see Lancet 1887 ii 165.

Definition - Beriberi or Kakke - a disease affecting the peripheral nervous system, frequently epidemic in the tropical or sub-tropical countries it affects, especially in overcrowded dwellings and amongst soldiers, sailors, prisoners, characterised by degeneration affecting the motor sensory, vasomotor and trophic fibres of the peripheral nerves, frequently without any great elevation of the temperature, manifested by paralysis, dysaesthesia, oedema, anaemia, oedema, pericarditis, effusions, and muscular atrophy, with oedema and palpitation of the heart, leading when acute to a fatal termination and when chronic to paralysis, deformity from muscular contracture.
Sympyos Loëmpoe (Java); Berias and Ahoi (Banua); Pantjukit Japoea (New Guinea); La paralysie de l'île de Panama P. Marie.

La maladie des ouvriers (French Antilles).

Barbier's is an old French term applied to the paralytic form & probably included other forms of paraplegia.

Bàëlz in 1881 described the disease as "franciscitis epidemica". Pekelharig and Winkler call it "Peripheral multiplier aritis".

Sir J. Fargen in the article on Beriberi in "Quain's dictionary of medicine" says it is the "sleeping sickness of Africa". But from a portion of the article on sleeping sickness by Dr. A. Gore, Brit. med. Jour. Jan. 2, 1875, it would appear that the two diseases have no points in common.

Etymology: Kakke is a name composed of two Chinese words which mean legs and disease; the disease is mentioned in Chinese literature of the period 200 B.C. and is described, as of wet & dry varieties, in another work written at the end of the tenth century.
Beriberi appears to be first mentioned in 1645 by Bontius, who was familiar with the disease in Java. The name now in such common use is said by him to be derived from the Hindi Bhāre - a sheep - from the fancied resemblance of the gait of persons affected to that of sheep. Carter suggests Bhāre a sailor from Bahr the sea as an Arabian derivation. The Malay word 'binibi' means abrupt tripping gait, and the Laccadive words 'behr beeri' - extreme weakness have also been suggested as origins of the name. But Hiles in his report (1889) says that the name has no vernacular signification in Laccadive or any other dialect. Almost probable derivation seems to be suggested by D'Sinclair of Belangor namely from the Sanskrit Bhāra, an aight; patients usually describe their complaint as a weight in the chest, legs, or arms, as the case may be.

It would appear that while each of the local names may be used in reference to the same disease, the use of each is attended by the disadvantage of being popularly applied to any disease that may bear rough resemblance to the component epidemic cause of mortality in the particular region affected. Thus "Beriberi" is very commonly misapplied to very various diseases.
Geographical Distribution

As regards its manner of occurrence, Beri-beri has at some places the character of an endemic malady, as in Japan where it has recently been observed & written on by Vermicke, Buzze Simmons, Baerz, Schenbe, Tashiki & others. It is endemic in many parts of Southern China, though it has been stated (cowers, dis. of Nervous system) that the disease appears to have become extinct there. It has been written on by Rennie in Foochow, by Coulson at Shantow; also in Hong Kong.

It was very prevalent in the Southern squadron of the Imperial Chinese Navy in 1887 during their visit to Chemulpo (Corea) in the autumn of that year it was stated that 30 per cent of the men were suffering.

In North China Dr. MacKenzie did not know of the disease existing at Tientsin and Dr. Illison informs me that he has not recognized it at Har- kow. It is however prevalent in Korea. It was epidemic in the Locos Islands in 1870. In the Straits Settlements - at Singapore Dr. Irvine Howell reports that 1174 cases occurred
in the gulf there between May 1875 and May 1880. It occurs also in the Calabash islands, Malay Peninsula & in many islands of the Malay archipelago - Sumatra (described by Van Leent & Belpke) - Banda, Borneo - it was introduced into the province of Kutai by the Netherlands man-of-war "Pahang" in 1851 & has become endemic. In Labuan, a British possession, its occurrence is described by Roe & Barry. In Java, vide Clapham; but in Bali, which is separated from Java by only a narrow strait, the disease is unknown. In the Celebes & Molucca groups, its prevalence in the Dutch East Indies was the cause of a government commission to investigate the disease. Professors Peckham & Winkler of the University of Wroclaw have added much to our knowledge on the subject. India - the disease occurs in Andhra & was admirably described in 1835 by Dr. John Byard. Malcolmson in a "Practical essay on the History & Treatment of Beriberi" (Reprint Division Press) it is endemic in the sparsely - Grandjan to Trasulipatam - along the coast extending inland with diminishing severity for about 100 miles (see page ) to a less extent on the
Coromandel coast, plain of Carnatic & Malabar coast. In Bengal an epidemic which occurred in 77-80 was called Beriberi by some observers, but its identity seems very doubtful.

In Burmah and Assam, it is said to have been introduced into Lower Burmah by the British troops in 1824 (A. Davidson in his "Geographical Pathology"); a severe outbreak of the disease occurred in the jail at Mandalay, about 500 miles upcountry, in 1887. In this instance no anthroponites were found in the fatal cases (the two diseases have been were still much confounded).

In Ceylon, it appears to have been confounded with Anthroponitis by some recent writers, particularly Hyneq in "Rep on the anaemia of Ceylon or beriberi 1887".

In Mauritius it is said to have been epidemic amongst the British troops in 1813; an epidemic in 1878-79 of "acute anaemic dropsy" has been taken by some writers (Norman Chenev, Brit. Med. Jour. 84, 1871) to have been beriberi, but it appears to have been an exanthematosus fever quite a distinct disease.
In Madagascar, Zanzibar, and Mosi-oa-Tunya, the disease is isolated to the N.W. of Madagascar. In Reunion, the disease is stated to occur, but the epidemic described 30 years ago by Dr. Wilson as here here attacked children only. There is no allusion to dropsy, from which circumstances it seems probable that the disease just took more of the nature of epidemic cerebrospinal meningitis.

Livingstone, in his last journal (1872), describes an epidemic of acute dropsy amongst the Kanganas and most of those who took the disease died; there is no mention of paralysis. I can find no satisfactory account of an occurrence of the disease on the continent of Africa.

It occurs in Australia, New Zealand, and New Guinea.

In the Western Hemisphere, the disease was first recognized in South America at Bahia (Brazil) in 1860. In 1874, it existed in Bahia only, hence it spread until now (it was said at the Brazil congress) it would be easier to stamp out yellow fever than here here. In 1875, it was hardly known.
by name in the province of San Paulo, now it is disseminated from North to South in inland towns as well as those of the Maritime zone, where it is common (Sodé Pareira). The disease appeared about the same time in Guiana & Paraguay & has been written on by Dr. Pareira, Sibon Lima, Lacerda - who claims having first discovered the microccus causing the disease & others.

The disease is now endemic throughout Brazil, in Argentina a law has been passed making all vessels with lepers on board "suspect" liable to quarantine. There have been outbreaks at San Francisco & amongst the fishermen on the Grand Banks of Newfoundland.

In the French Antilles, according to a report of Dumont published by Living there are occasional cases of the disease amongst the Chinese & negroes. It is known there as "la maladie des excrécences" & occasionally epidemics. In the West Indies it has occurred in the islands of Guadaloupe & Cayenne. In Cuba it raged with great violence during the year 1873 amongst the negroes causing mortality stated to be from 60 to 75 per cent.
Geographical Distribution (cont.)

The disease affects by preference the coast line & the shores of large rivers.
It has been observed that in many places where the disease is endemic the soil is
peculiarly rich in saline materials —
magnesia, lime chlorides, alumina & xron,
but that these have any determinant effect
is not clear.

Etiology — Contagion, the antagonism
of opinions on this point seem
to have arisen from a confusion of diseases
under the same name Periherin. Pekelharing and
Winkler state that “almost universally has
this affection been regarded among medical
residents as a disorder of infectious origin;
and they concluded, after long study of the
disease, in the Dutch East Indies that while
the disease is not readily transmissible from
individual to individual it may be so trans-
mitted by wearing apparel &c.; usually
infection does not show itself until after
a residence in an infected locality for 6-12 months. The intensity of the virus seems to become much aggravated by overcrowding, insufficient ventilation & want of cleanliness; outbreaks on board ships, in barracks & in prisons have been particularly fatal; under such circumstances the period of incubation & of prodromata becomes very much shortened. Leslie (on her herein F.C. Press Sinla 1891) considers that the usual period of incubation is 3-5 weeks.

Climate - although the endemic areas affected by the disease are confined to latitudes lying within about 37° of the Equator, north & south, the disease may persist & even develop beyond those areas. At Hakodate, in Japan, the disease is endemic & the climate there is much the same as ours in Britain.

The disease usually develops at the changes of the monsoon & is worst during the hot, rainy monsoon (the S.W. monsoon N of the Equator) & the approach of the cold dry season has nearly always a good effect on the violence of the outbreak.
Climate. The following table shows the distribution according to season of 572 cases, admitted into Military Hospitals in the northern division of the Madras Presidency during a period of three years (from Waring).

Table of Admissions for Bengali at Seasons

<table>
<thead>
<tr>
<th>Hot season</th>
<th>Rainy season</th>
<th>Cool season</th>
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<tbody>
<tr>
<td>March</td>
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<td>December</td>
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<td>April</td>
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<td>86</td>
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<td>390</td>
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<td>or 15.03 percent</td>
<td>or 68.2 percent</td>
<td>or 15.7 percent</td>
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The same fact is demonstrated in D'Irvinie Rowell's report of the more recent epidemic in Singapore, the disease was always worst during the rainy (S.W.) monsoon. See Table page over.
<table>
<thead>
<tr>
<th>Month</th>
<th>1875</th>
<th>1876</th>
<th>1877</th>
<th>1878</th>
<th>1879</th>
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<tbody>
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<tr>
<td>Total</td>
<td>127</td>
<td>110</td>
<td>86</td>
<td>14</td>
<td>102</td>
<td>142</td>
</tr>
</tbody>
</table>

Percentage of Deaths to Admissions:
- 1875: 11.81
- 1876: 16.28
- 1877: 100.00
- 1878: 16.20
- 1879: 20.63
- 1880: 4.47
Exposure to cold is a predisposing cause of the disease; it is very common for patients to give a history of having been exposed to chill by sleeping out, getting wet etc. The fact of its having been experienced may very frequently be found on inquiry. "Cold" was much complained of in the ship "Farje Allum," in which 35 cases occurred among a crew of 65; also by the sailors of the Brazilian cruiser "Guanabara" on board which the disease developed at New York.

Age — there is perfect unanimity of opinion among the authorities that the occurrence of beriberi in children under fifteen years of age is exceptional; in many epidemics no such cases are met with. So well established is this that it now appears doubtful whether the epidemic in Réunion, described by Vison, which attacked children only could have been beriberi; certainly it is possible for the Protestant Orphanage at Surabang in 1864 there were 48 cases among the 235 children 9 of which were fatal.
and, in the Missionary schools at Donkiga in Japan a large number of girls, between the ages of ten and sixteen were attacked, and one died. On the other hand on board "L'Inde" there were 575 emigrants, among them 526 children up to fifteen years of age; there were 118 cases of beriberi but only one of these was in a child (lungs); and, in the Jacques Carrié, the whole of the 55 children of the crew escaped the disease, while of 332 adults, 44 took it (Richard).

Childhood was found to be exempt from the disease in Lebanon, according to Barry, during the epidemic of 69-70.

Of 35 fatal cases at Tokio only one was in a person under fifteen; of 433 patients at Tokio 15 were between ten and fifteen years of age, 753 or eighty percent were between sixteen and thirty and 89 between thirty and forty, the remainder were over forty (Heiser).

Of 581 patients treated by Sekrke at Kioto 35 were children.
Sex. women are less liable to the disease than men; in women the disease is comparatively rare. On board the 'Indien' 111 out of 385, or 29 per cent of the men were attached (10.4 per cent died) while only 6 out of 118 women or 5.15 per cent were attacked and 2 died out of the 118 female passengers.

On board the 'Jacques Lécuyer' the proportion of cases among men to those among women was as 12 to 7.

Of Schenkel's 5841 cases fifty were females, or as 1 to 11.

Among 2224 cases healed in the Hospital at Tokio, between the years 1879-1881, there were only 68 women, or 1 to 31.7 male (Baile).

Pregnancy & the Perinatal period render women more liable to the disease. Dr. A. H. Bentley attended a Princess of the Imperial family of Jochore who died having contracted the disease after childbirth. In this connection it is interesting to note that D'Handford in Brit. med. jour. 28 Nov. 91 records three cases of multiple neuritis, where the onset seemed to be determined by childbirth.
Occupation, of 333 patients treated by Schenle in 1877-78 no less than 46 per cent were either scholars, priests, teachers or writers, while 41 per cent were in business & 13 per cent artists or artisans: the conclusion which Schenle draws is that those occupations predispose especially to the disease which involves a sedentary life.

Verrich also draws attention to the frequency of the disease amongst the student class.

Féris had the same experience in Brazil: he says "la vie sédentaire comme une cause secondaire de la maladie"... is no doubt the reason why the disease so frequently attacks the lettered class.

The same predisposing cause attains in the case of soldiers (under some circumstances) sailors & prisoners amongst whom so many epidemics have occurred.
Natives are more liable to the disease than Europeans or Americans; while I was at Hong Kong I was informed that a case of the disease had not been known there in either of the latter races; a case occurred shortly afterwards, however, in the person of an Englishman from Shanghai. Varying estimates the proportion of native cases and Europeans at 6 to 1 but later authorities have observed a far greater proportion of cases amongst natives than this.

The predisposition to an attack increases up to a certain point with the degree of acclimatization; new comers do not at first as a rule contract the disease. Hamilton states there is no instance of an individual being attacked with the disease immediately upon his arrival in India. Malcolmson considered a residence of from 9-12 months as usual before the disease could develop. Bailey had the same experience in Japan; among the Japanese who came from the interior to the coast, which is the home of the malady,
it never appeared until after they had resided some time, but after that, the cases became more frequent amongst them than among natives of the coast: wherein lies the explanation of the large number of cases that occur among soldiers, sailors, students, etc.

It is found in Brazil also that strangers are exempt from attacks for 6-12 months after they have come to reside in one of the infected centres.

Debility does not appear to be often a predisposing cause, the disease often attacks those who have had exhausting attacks of fever, dysentery, etc. On the other hand it is often seen in those who have previously been in good health. Rupert writing of the disease in Sumatra records, "the fact was brought out that there were no doubt a few weakly individuals among the patients, but that the great majority were strong persons between the ages of twenty and thirty, so that it was often the strongest and best nourished who were attacked."
Many of the worst epidemics that have occurred have been among soldiers and sailors—who are a hardy, rate not debilitated people not predisposed by previous exhausting disease.

Bailey in 1881 treated 626 cases, at the Japanese Hospital Tōkyō & classified them as being 593 robust, 27 of moderate strength & 6 weakly.

Influence of Diet—most of the older writers attribute great importance to diet as being among the predisposing if not among the exciting causes of the disease.

Malcolmson (1835) was an exception to the rule. He said, and the argument is a strong one, “when we reflect that these (cereals) are standard aliments all over India we cannot carry our deference to his (Herklotz’s) experience so far as to admit that they can produce in those districts only so singular a train of symptoms.”

Rupert from his experience in Borneo, in 1880, came to the same opinion that if an improper diet or a preponderance of any saccharous vegetable food played the chief part in the production
of Beriberi, the disease would have to be a very general one, as much as rice is the staple food of the people of India & the East Indies, but the fact is that the disease is restricted to certain regions or spots, mostly on the coast or in immediate proximity to it.

The consensus of opinion however, seems to have suspected the sufficiency of the dietary of those who suffered. C. Morehead considered the disease to be an intense manifestation of the scorbutive diathesis & points out that all the cases which occurred in the "Farje Allum", after she had been two months & eighteen days at sea, affected those amongst the crew who refused to take vinegar, pickles, or any anti-scorbutics, while the passengers & others who consumed these articles escaped: his experience of the disease seems to have been somewhat limited & the scorbutive theory may be considered relinquished as untenable.

Mugliston (1886) states that a diet of inferior rice was also deficient in fruit as an exciting cause of the disease; "the disease has not attacked the men of a batch of coolies living..."
"under apparently the same circumstances"
and on enquiring it has been found that those
attacked have not eaten fruit, while others
of the batch have done so. Those enjoying
a better diet though eating no fresh fruit
have not been attacked so soon as those
who have squandered their money in
gambling or opium smoking also obtained
less food. By far the majority of patients
have been Chinese who eat in their usual
diet less nitroasensous food a fruit- than
do the natives, though otherwise living
under precisely the same circumstances.
I have thus been forced almost to believe
in the scorbatic origin of the disease.

Overheert de Heezer attributed outbreaks
of the disease to consumption of salt meal and
Pop spoke in the same way about outbreaks
in Dutch men of war. Blendijk was of opinion
that herberi appear on board ships when
ever the food ran short or became spoiled.
These views are still upheld by D'van
leent in the Dutch East Indies and by
D'Iskaki of Tokio."
Dr. Van Leest regards the "diabetic error as the one and only cause of Beri-beri, by producing a morbid composition of the blood, in consisting of a too small proportion of albuminous substances." 

This theory was put to the test by Onishi, who also believed that the disease was produced by too great a proportion of rice and cereal diet—his experimental observations were carried out in the Japanese Navy & the apparent result has been that the disease has almost entirely disappeared. The Sei Kuni Medical Journal publishes a table giving all the cases of Beri-beri in the Navy from 1878 to 1889 inclusive in which the result of this change of diet is strikingly shown. The change was effected in 1884; previous to that year the number of cases had ranged from 1163 to 1978, or from 23.12 to 40.45 per cent. of the entire force. In 1884 the number fell to 718 or 12.74 per cent. of the force, while in 1889 there were but 3 cases or 0.03 per cent. of the active force. During 1890 the number of sufferers from Rakke in the Empire was unusually small & this Yakakei
attributes to the fact that the common people were obliged to use much less rice than usual on account of its high price, eating in its place wheat & beans. He is at present investigating the quality of food consumed in public boarding houses, in relation to the number of cases of Rachke. Table I. pg. 92.

Kernich & Magest also attributed blame to the large consumption of rice.

 Kearney raised the question of disease or bad quality in the grain producing the sickness, in a manner comparable to the production of Ergotism or Lathyrism, but no support attended this theory & the more common view was that the large consumption of rice to the exclusion of albumens & fats resulted in the production of the disease.

On the other hand D'Urville Rowell in his Report on the outbreak in Singapore said points out that the dietery was one on which many of the prisoners gained weight.

Dr. J. H. Bentley in his Thesis cites the occurrence of an outbreak in the Royal family of Jodore as proof that poverty or want of variety in diet is not a necessary factor in the etiology.
Sir Robert Kerr speaking of Brazil says that people of the higher classes of society may be more attached to the disease than those at the other end of the scale, who are struggling for food.

Schickle points out that the Ainos, (the ancient indigenous race of Japan) who are mostly hunters or fishermen & whose diet is richer in albumen & fat than that of other Japanese natives, suffer equally. Baily observes that the disease is worst (eg commensal) where there is most animal food that is to say at the seashore.

D'Andison of Tokio says that the Cooke class who live more exclusively on rice than the soldiers sailors or traders suffer far less from the disease than these do.

Surgeon Major White who had experience of the disease in Ceylon concludes that chiefly nothing whatsoever to do with its production.

D'Shares Summons of Yokohama says that those in a position to afford a good abundant supply of food are most liable to the disease.

He adds however that rice is often badly tolerated by the patients. 'Perhaps on account
of its constipating qualities or the deficiency in the grain of potash.

Rupert speaking of the epidemic amongst the troops & men of war in Borneo says that they had fresh beef twice a week, poultry, eggs & coffee in their rations besides fish, salt meat, potatoes & rice; at the same time that the labourers of the country living on rice & dried fish were entirely free from the disease.

Dried fish, a common staple article of diet, has not eschewed attack in this connection; the consumption of it is universal in the East equally by the healthy as by the diseased.

Bentley in his account of 67 cases of the disease gives the dietaries of several of his patients, & he found that they were up to an average standard.

**Drinking Water** has frequently been supposed to be a medium by which the disease is disseminated; in many instances however epidemics have occurred where the supply has been beyond suspicion; at Singapore for instance.
The water supply is particularly good and could have played no part in the outbreak which occurred in the yard. D'Clapman mentions, of the outbreak in Java, that the water supply there was good, being derived from deep Artesian wells.

Malaria as a factor in the etiology

Surgeon Major White, who had great experience of the disease in Labuan, and many older writers, regarded the disease as an intense form of malarial poisoning; this view is combated by Hirsch who in summing up says that it has not the very smallest point in common with malarial diseases, that it occurs in India in places that are wonderfully dry, but does not occur in certain other places which are wetter: that the disease has spread at various places during the last 30 years without there being any changes in the soil and that it becomes epidemic on board ships where there can be no question of influence of the soil. Excellent as is the summing up of Hirsch on other points it seems to me that much can be said in answer to this view.
in the first place many diseases which are certainly malarial in type become prevalent on board ships: Intermittent & Remittent fevers both do so; Malaria fever is extremely common on board ships; Rabies resembles yellow fever in its persistence about the coast line, round the shores of rivers, & in prevailing in circumscribed localities as well as in the manner in which it spreads in localities in which it has once become established: Secondly, the disease is usually found under conditions which give rise to malarial affections generally.

"Duane Simons of Yokohama who has for a long time been investigating this subject says that his researches justify the conclusion that the exciting cause of the disease is a specific miasm or furred exhalation. He goes on to say - which is well worthy of quotation as showing the conditions under which the disease thrives " on my arrival in Yokohama 14 years ago, four months after the opening up of the country, I found a small fishing village stretching along the shore of a deep bay at the mouth of one of the numerous valleys
valleys with high bluffs on either side such as everywhere break the coast line of this sea girt Empire. Behind the somewhat elevated gravel belt occupied by the town and stretching inland for 3 or 4 miles were low rice fields on either side of a sluggish stream. As the town grew these fields were filled in by soil from the bluffs until today a city of 50,000 inhabitants rests on this new made land, in some parts below the sea level. This is the description of the parts where the disease is most prevalent. As may be seen drainage is difficult and the soil is saturated to within a few inches of the surface with brackish water. One of the results of this exposure of sea earth, much of which was not immediately covered by houses, was the appearance of the disease in severity and frequency before unknown. Their number remained proportionately small for some years, but the disease later on took an epidemic form. He goes on to say that the spores of the disease are wafted by the air to a distance from the source. He instances as example of a low lying valley in the town with a hill on either side which is the dump S.W.
monsoon the germs are conveyed in the atmosphere up the slope of one bluff in the direction of the wind attacking the inhabitants, whilst those residing on the opposite side of the valley enjoy complete immunity.

"Dr. Irvine Rowell in his Report makes the following statement of the conditions at Singapore gaol at the time of the outbreak " the disease was first recognised in May 1875 " it has been present in the gaol every year since then except in 1877 when only two cases were treated. The prevalence & mortality became so alarming in 1878 & the first four months of 1880 that the native portion of the gaol was condensed as unfit for occupation & abandoned in April of the latter year. The malady first confined itself to long sentence men - Chinese Malags & Indians alike, but no Europeans or native female prisoners suffered. The death rate was 16.20 per cent of admissions in 1878 & rose to 20.63 in 1879. The year 1877 in which two cases only occurred was an unusually dry year & the prisoners were employed extremally.

The European & native female prisoners were lodged in a higher & drier part of the gaol than
the native male prisoners. The prison is built on an old mangrove swamp, at an average level of only two and a half feet above high water mark, surrounded by high walls & along one side of it runs a tidal stream which is much contaminated with organic impurities. A sluice gate was put on this stream after which the disease became worse. Verb. Ind. Ind. Aug. 1. Dec. 81.

Damp surroundings alone are probably to some extent predispositive: a large number of Bentley's patients had a history of having been at work in wet places, or in freshly broken ground: at Singapore the amount of disease varied exactly with the amount of rainfall (Non-tropical) & was much less during the comparatively dry year 1877 than in previous or subsequent seasons.
General sketch of the Disease

The disease presents such a variety of clinical features that a general sketch which will include most of the phenomena that may be present is difficult to make. Some writers describe the disease as of two varieties, a wet & a dry, others make three varieties. Dr. B.J. Bentley describes five as follows:

1. wet, or dropical,
2. dry, paralytic or atrophic,
3. spasmodic,
4. acute pernicious,
5. mixed.

There are however no fundamental differences in the character of the disease at the various places of its distribution area.

The onset of the disease is usually gradual & preceded by a feeling of weakness, lassitude & languor, but it is sometimes abrupt; it generally commences with a feeling of numbness, sense of weight & slight weakness in the lower extremities, which are usually affected before the upper & at first feel merely stiff, below the knees; on examination slight oedema of the feet
& legs may be found, especially along the inner sides of the tibia, this often appears later than symptoms of pareses: the patient will complain of various dysesthesia, muscular pains, twitching of the limbs or formation.

The walk soon becomes unsteady & irregular even though there may be no great amount of weakness or pain & the knee jerk will be found to have disappeared: the flexors of the ankle & extensors of the wrist suffer earlier than do their opponent groups of muscles but later all may become affected. The edema soon affects the face & hands so that the general expression & gait will often of themselves allow a diagnosis to be arrived at: with the spread of the dropary the pericardium becomes affected & shortness of breath, pain in the epigastrium & palpitation make their appearance. Frequently these symptoms will abate & the patient recover if a change of residence is affected. Frequently after a remission of longer or shorter duration they will recur with greater severity, the numbness extends over the abdomen to the face & lips, there is great aversion to movement, spasms
occur in the calves & may become general and affect respiration or speech, there is a sense of oppression & pain at the precordium & the lower extremities become completely paralysed, a condition of wrist-drop & foot-drop is established. The adenæ extends & becomes general, extreme dyspænea & inability to lie in bed & anæmia supervene; perhaps after several fainting fits the patient dies. The skin is often cold & the patient very susceptible to draughts; the urine is nearly always lessened in quantity & may be almost suppressed; the appetite usually remains particularly good but sometimes there is gastric irritability, vomiting & diarrhoea are rare & extremely unfavorable symptoms. Constipation is usually present & there is often pain & tenderness in the abdomen. Effusion into the peritoneum is not so common as into the pericardium & pleura. Even in chronic cases death sometimes supervenes very rapidly or fresh & more severe symptoms may suddenly appear by which the patient is rendered completely bedridden, perhaps to survive for many months paralysed and in pain.
General notes. The temperature of the patient shows but slight departure from the normal frequency there is a slight evening rise but considerable depressions may also occur. The disease is endemic in circumscribed localities between latitude 37° N (Japan) & 33° S (Sydney) prevalent mostly near the sea, is worst during the summer rainy season, attacks chiefly young adults men not readily communicable from individual to individual. Wet, tropical chills, overcrowding & malaria predispose to it.

Symptomatology. The onset of the disease may be apparently very acute, as it was in Case I page 52.
C. Morehead reports a case which occurred in the "Tarjei Allum" which was fatal in 3 days & others mention even more rapidly fatal cases when the disease assumes a virulent type. a man may be following his ordinary occupation one day & be in a condition of general weakness from Berlin the next, but as a rule the onset is very gradual. Reehlbrinck & Winkler state that the initial period is characterised by a quantitative alteration evidenced by a diminution
in the susceptibility to the electric influence usually for both species of current, frequently led by a qualitative change in the electrical reaction of those muscles which determine the flexion of the foot; in addition there is an increase in the diameter of the circumferential measurement of the calf of the leg; occasionally pain or a feeling of heaviness in the legs or cardiac hyperexcitability accompanies these symptoms.

The face assumes a typical puffy appearance by which experienced medical men have recognized the onset of this malady long before other more positive phenomena had been demonstrated.

Rowe says that a white line at the commissures of the mouth is an almost invariable early sign.

While at Hong Kong, where the disease was at the time prevalent, an ex gunsase R.N., who was in charge of a Chinese revenue gunboat, informed me that he was in the habit, when he enlisted fresh hands (which was part of his duty) for the Chinese service of testing the amount of the candidate's knee jerk. This gentleman having no medical officer, finding that a large number of men who entered the service subsequently developed herpess.
became useless & had to be provided for by
the service, became inquiring as to a method
by which to put a stop to the entry of unsound
candidates. Eventually he told me was
enlightened by a Chinese servant. In beriberi
the knee jerk early disappears & unless a
Chinaman could show an average knee jerk
this officer refused to enlist him.

The course of the disease is usually very
chronic in places where it is endemic; at least
perhaps influences favorable to the developmet
of the virus, climatic or local, cause a sudden
increase in its potentiality & an epidemic
follows with its series of acute cases. Several
authors point out that a residence of a certain
lengthened duration in a locality in which
the disease is endemic is invariable before
manifestation of symptoms. Malacharion &
Peelharing mention 6-12 months.

The most quickly fatal cases are often such
as are accompanied by but slight visible out-
ward signs, & when the type of disease is of a
chronic nature exacerbations & remissions
are prone to occur. Speaking of beriberi among
the marines of the Indian Navy in "Palinurus"
"Narconda" Carter says "so constantly did the symptoms of the disease disappear altogether and reappear at intervals of uncertain duration that I feel sure, if a native Marowr be once affected he will, if he remain at sea eventually die of it; that indeed at short warning at a time when the return of the disease is least expected. But very usually the disease runs a steady course terminating after a duration of some months either in recovery or in death from exhaustion or asphyxia, or perhaps in the patient becoming a hopelessly bedridden and spherical paralytic. The disease may even last for years, with occasional remissions or exacerbations. Even when the characteristic symptoms are all gone the patient is left emaciated and emaciated and with feebleness which may persist for good.

Sensory disturbances may make their appearance either with before or after motor affection. Dr.made Simmons has found that in nearly all his cases anaesthesia is the first symptom, appearing over the anterior limb of the muscles of the fingers and around the mouth."
Along with a feeling of undue fatigue, first noticed in the lower extremities, there are various related subjective phenomena or hypesthesias.

The skin along the inner sides of the legs is found to be numb or very various sensations may be complained of, such as a feeling of heat or cold, prickling, tingling, formication, pins & needles, as the disease progresses these become intensified & deep seated pain may be present which is often referred to the bones.

In Wernicke's experience the disease is usually unattended either by actual pain or fever, but as a rule considerable discomfort is present; even in mild cases, in making deep seated pressure between the bones of the legs & forearms causes pain. Sensibility to touch is frequently diminished while pains are experienced in the same parts; a condition of anesthesia dolorosa.

Sensations of heat & cold, while the power of distinguishing between them as applied by the 'spoon test' is diminished, have also been noticed: they are frequently present over the trunk as well as the extremities & the patient are peculiarly susceptible to currents of air.
The muscular sense is much diminished and slight pressure is attended by pain.

These symptoms become more marked in cases that run a long course. Acute cases are usually attended with effusions which mask the sensory symptoms or replace them: commencing in the legs or feet, the dysaesthesia & anaesthesia spread as the disease progresses to the trunk & upper extremities. Occasionally are experienced in the lips: they are much increased by movement or exertion and followed in their course by paralysis. Malcolmson describes pain along the spine as being frequently present particularly over the lower lumbar vertebrae.

Palpitation of the heart, of a paroxysmal character, is often one of the distressing symptoms & accompanies effusion into the pericardium; it is probably due to implication of the Vagus which is frequently involved in the course of the disease.

Epigastric pain is also a common symptom & seems to depend on the presence of pericardial effusion. Surgeon W.G. Morris also describes having found 'girdle pain'.
Motor symptoms the earliest changes are to be found in a change in the electrical excitability of the peroneal nerves & the flexors of the ankle as already described on page 37-38.

Pekelharing states that a slight degree of the reaction of degeneration, quantitative & qualitative is always to be detected before there are any subjective phenomena; these observations have however not yet been confirmed so far as I am aware by other observers; the same writer is of opinion that the onset of the disease is always gradual & that the so-called acute attacks are but exacerbations occurring in the course of an otherwise chronic malady.

The Plantar & patellar reflexes disappear & may be found wanting in patients who have no other manifest signs of the disease, probably on account of the efferent muscle nerves being more susceptible than others to the disease.

The gait soon becomes affected, Pekelharing states that electrical alterations may occur without the disease progressing so far; the toes remain pointed to the ground, the anterior tibial muscles being unable to raise them & the heels are brought sharply to the ground; it does not resemble the gait of people affected with
Locomotor ataxy, though there is a degree of incoordination, from greater paresis of some muscles than others, but there is also a loss of power not present in locomotor ataxy. The gait more nearly resembles that of a child learning to walk; it is very commonly found without other such marked signs of the disease as ataxia; the amount of abnormality depends on the number of muscles implicated, for later the calf muscles also become implicated. The disease spreads to the extensors of the knee & thigh adductors; the wrist extensors & flexors may also become implicated & muscular hypertrophy is found between the radius & ulna & the tibia & fibula.

In some cases the patient loses the use of the limbs after a certain amount of indisposition quite suddenly.

In the trunk the muscles of the abdominal wall & intercostals suffer; the diaphragm & muscles of the larynx occasionally are affected. The skin reflexes are early lost, but the organic reflexes concerned in the functions of breathing, swallowing, defecation and micturition are never involved. — Wallace Taylor

States partial paralysis of the bladder occurs rarely. Jany 8th.
Adema in the lower extremities causes the swollen leg to be comparable in shape to a pear, the stalk being represented by the ankle.
Vasomotor impairment is probably a cause of the extensive oedema and anasarca so often present. Effusions are not constant, in many cases there are only faint indications of them, sometimes they appear at the very outset of the illness at other times they are a late addition to the group of symptoms: hence the clinical distinction that has been made of a dropsical form, a paralytic form, a mixed form.

Oedema first appears along the inner side of the tibia & dorsum of the feet. The face is very often affected & the appearance of the patient is then somewhat pathognomonic, though I believe the anasarca of anthrax is similar closely resembles it.

Effusion into the Pericardium was found in 67 percent of autopsies made by Scherle & is often the cause of death by hindering the action of the already weakened heart.

Hypermnoria is not found quite so frequently, it accompanies oedema of the lungs & the oppression which it gives rise to is a marked feature of the disease. Ascites is common but less so than the preceding two forms. Effusion into the cerebral & orbital arachnoid is occasionally found; it
was present in 1 to out of 14 autopsies made by D. F. M. Bentley; while oedema was only absent in one case.

Wernicke of Redde has described the appearance of "factitious urticaria" he says "a patient may be lying in bed feeling tolerably well in the morning but in the evening the appearance of oedema over circumscribed parts of the skin especially near the nape of the neck will indicate that he is in extreme danger & the next morning he may die suddenly without pain or perceptible collapse.

The subcutaneous functions of the Nervous system are not at first so much affected as are the other functions; bed sores are rare; the trophic condition of the skin remains unaltered & the muscular atrophy & subsequent contractions are only occasionally seen & follow only in cases of complete paralysis.

The cerebral & mental functions remain clear and are but little affected: anæsthesia is generally marked. Torpor or coma does not occur at all usually, I can find no reference to them in any of the recent authorities; the statements of J. Fagge & Horton that the disease is identical with the
"Sleeping sickness of Africa" must be based on misconception, for judging by Leye's description of lethargy, the two diseases would seem to have nothing in common.

Sleep is generally much interfered with either by pain in the limbs, startings or twitchings, or on account of the cardiac or pulmonary conditions inducing dyspnea.

Locomotory system in the paralytic form the muscles at once loose their tone to become flabby; this is followed by atrophy, which is often extreme, & results in contracture, with the foot extended, the hand flexed. This contracture, at first due to the active or unopposed muscular action in the less affected group eventually becomes permanent from tissue change &formation of fibrous tissue.

In connection with the alimentary system

Dr. Bentley has found congestion & inflammation of the fauces, in a considerable number of cases, he does not attribute any particular importance to the condition, but it does not appear to have been noticed by other observers.

The appetite is generally unimpaired throughout in moderate cases, & in cases which ultimately terminate fatally, it is frequently unimpaired until
a very few days before death. Vomiting is not a common complication; if it sets in at all, the case will probably end fatally. Constipation occurs in a large majority of cases & is obstinate in character. Diarrhoea is a serious symptom, it is frequently explosive in character & seems oftener to occur in patients who have previously suffered.

It is common to find the conjunctivae have a slight yellow tinge.

The tongue in old cases is frequently furred & flabby.

The spleen & thyroid glands are usually normal.

Anaemia is not at first marked. Vernick & Fajer endeavoured to show that the disease was a kind of pernicious anaemia, but Bäde & Pekelharing have shown that this is by no means the case & that anaemia is by no means a constant nor even a common symptom. Pekelharing & Mülles state that beriberi has no dependence on anaemia for its symptoms.

Schenke & Bädez gave as their opinion "without examining the blood microscopically" but purely from clinical data that anaemia was not an important factor. See table over page.
<table>
<thead>
<tr>
<th>Name</th>
<th>Europeans</th>
<th>Natives</th>
<th>No. of red corpuscles</th>
<th>No. of Hemoglobin</th>
<th>Remarks</th>
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<td>D'Epstein</td>
<td>E</td>
<td></td>
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<td>D'Epstein considers his blood normal.</td>
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<td>D'Romanov</td>
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<td>10</td>
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<td>7,200,000</td>
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<td>Baddaung</td>
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<td>5,840,000</td>
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<td>Blood examined shortly after death.</td>
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<tr>
<td>Minem</td>
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<td>(native woman)</td>
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Corpuscles counted with the Malassez hemacytometer & the proportion of Hemoglobin estimated by the microcinnine method of Sr. Lagen. (From Rechinger & Windeler, Recherches).
Anemia. The investigations of Schneider have shown that the number of blood corpuscles may be increased as well as diminished.

Circulatory System. Pain in the region of the heart, palpitation, violent & paroxysmal, faintness & dyspnea are of frequent occurrence; they result from implication of the cardiac fibres of the Vagus. In mild cases may be absent.

The form & outline of the Pericardium may be altered by the effusion which takes place into the pericardium; the cardiac impulse is then feeble & diffuse & the area of cardiac dulness is increased either by hydropericardium or by dilatation of the heart itself. Blowing murmurs may be detected over the semilunar valves or apex & venous hum in the vessels of the neck is frequently present. In post mortem examinations the heart is generally found to be hypertrophied & dilated. Maga found the average weight of the normal heart among the natives was 237.5 gms., but the average weight of 25 hearts from people who had died of beriberi was 364 gms. Peterhans & Winkles found it to be 355 gms. See page 82.

The Pulse tends to increase in frequency & feebleness as the disease progresses.
Wallace Taylor states that sphygmographic tracings revealed a high & sudden upstroke with precipitous descent from the apex of the percussion wave & marked diastole.

The Respiratory System may be involved either by implication of the intercostal nerves causing paroxysms of the respiratory muscular mechanism or by dropping of the lungs & pleurae from vasomotor disease. Orthopnea is often seen in severe cases & asphyxia is frequently the immediate cause of death.

Urinary system: The secretion of urine is nearly always diminished. Malcolmson described it as being at first of a deep red colour without clod or sediment & says that in some old cases it becomes copious turbid & pale. Unless marked fever accompanies the attack the urine is not much altered; it is usually concentrated & diminished in quantity & albumen in generally not present: viz. 1020-1010; a critical increase in the secretion may mark the commencement of improved.

Fever, the temperature is not always found to vary from the normal, often in severe cases there is a slight evening rise, but occasionally
It falls much below normal (see charts pages 104 & 105) found of 5 cases that were treated in the United States Naval Hospital from the Brazilian cruiser "Guararara" that the temperature averaged 99° F. Wernicke does not consider fever a constant feature of the disease. In the case which occurred on board U.S.S. New York the temperature was 99° during the stay of the patient on board. But sometimes there are febrile attacks, particularly during the exacerbations which are so common, of which are probably often classed as "pneumonia" or "acute attack." Békélharing records such a case in the person of a European, a soldier, aged 30, in which the bifacial nerve was affected and the pulse rose to 103 1/2° F. Fever however is not a characteristic sign of the disease. I have said so much about it as Homan Clems, apparently mistaking the disease altogether, has stated his opinion that the complaint is an "electromotor" fever. Two epidemics resulted from this disease, one in 1882 and 1894. Droopy as already been referred to under vaso motor impairment (page 45). Its conspicuous presence or absence depends in great measure on the state of the heart-conditioned by that
of its nerves, partly also on the vascular impairment which results from the general peripheral neuritis partly on the state of the blood. The 'mild' or 'dyspeptic' cases are far more acute & fatal than the dry or atrophie. Dr. B. L. Coupland narrates an outbreak of the latter variety of the disease affecting the boys & girls schools at Sontow. The anaesthesia & paralyses were marked and attended by atrophy, but none of the cases proved fatal.

Case I. LING AH TAY, aged 26, stoker, was placed on the sick list of H. S. Mermaid at Hong Kong on 14th April 1887.

Patient was a well developed Foochow man who had been employed in the ship as stoker since our recommissioning in July 86. When I examined him I found fit for the service. He had been once before on the sick list with Chancroid which was well after 27 days. Latterly he had been going ashore every evening to sleep with his friends.

On inspection - patient presented himself with other sick people in the steerage, having
descended about a dozen steps - his condition was one of general edema, the face hands & feet particularly bloated, the lower extremities, abdomen thorax & forearms in a diarrheal condition. he is short of breath & the respirations are shallow and hurried; there is no jaundice or cyanosis, the gums are pale & firm, the tongue white & coated.

he complained of pain in the epigastric region & of sensation of pins & needles in his legs and hands which appears to be numb. he can stand easily & move about slowly. is unconscious to be allowed to go home. temp 94.2 f. resp 28 pulse 100, weak & regular.

he had never had any bad illness previously that the known of & said that he was quite well until two days ago; yesterday he was at his usual work but says he could hardly go on with it; he was noticed to be sitting about & not doing his work as usual but there was then no difficulty. he could have gone on the sick list earlier had he noticed without detriment to himself in any way.

severe symptoms - he had pain in the chest which he complained of most in the epigastre region, his legs also ached & he had tingling in his lower extremities & forearms, which were
also numb: his sensibility to heat & cold remained. The organic reflexes—swallowing, breathing, micturition & defecation were normal as also were the special senses. No knee jerk or plantar reflex could be obtained on either side. The gait was somewhat tottering & the lower limbs evidently were managed with difficulty: his speech was feeble.

The electrical irritability I was unable to investigate from want of a battery or cord.

The cerebral & mental functions appeared normal: he was perfectly intelligent but evidently in great anxiety: there was no sign of torpor or coma.

The cardiac impulse was weak but regular. The dulness somewhat increased to the right; on auscultation I found no murmur, but the first sound was muffled. The Respiration was 28: vocal resonance & pulmones were diminished posteriorly; the breath sounds were distant & fine crepitations were present with inspiration. I could not detect the presence of fluid in the peritoneal cavity; nor any enlargement of the liver or spleen. The bowels were constipated. Urine—about 300 cc were passed in the 24 hours, somewhat high coloured.
was a slight deposit of urates: 24 gr 1022, R.
acid: there was no albumen present.
The patient remained on board for the rest
of the day only: he was given a milky
soup which acted for food: he was given milk &
rice pudding which he took well. At his own
request he was allowed to go on shore to stay
with his friends as the ship was leaving
Hong Kong. I was unable to see him again.
I found on subsequent enquiry that he died
on 20th April - the ninth day of his being sick.

The Chinsamen on board freely expressed their
opinion that Ah Fai was suffering from
"Kakhe" that he was "Plenty bad": they thought
that others in the ship would contract the
disease: there were however no other cases
though the disease was prevalent in the
neighbourhood at the time.

The following case is reported by F. E. Morehead
in Trans Med & Phys Soc, Bombay 1855
Vol ii. I insert it to illustrate the subject
having had no other cases under my care —
Case II. Puschatum Zeena, a third-class Kalasree of the ship "Fairie Album," a man of short frame was admitted into the Jamsetjee Jejeebhoy hospital at Bombay on 21 July 1853. He had been ill 16 days; his ship had been two months and 18 days at sea on passage from Singapore to Bombay in wet and equally weather. The crew were healthy up to 21 July, after that 35 men of the crew of 55 fell ill with symptoms similar to those of this patient. A death resulted in 10 cases — one, a Portuguese Sepoy, died after 3 days illness only; for the last month, the crew were on somewhat diminished rations; at no time did any corbelles form part of the diet, for the crew refused to take vinegar or pickles; they were exposed also to bad weather and fatigue.

On admission, patient had been ill 16 days the feet, legs, thighs were oedematosous. He walked with a waddling gait. The pulse is easily compressed. There is no abnormal dulness of the precordial region, and the sounds of the heart are normal. The bowels are rather confined, and the urine is scanty.

He complains of weakness at the epigastrium.
There is no vomiting, the tongue is not coated but is rather florid. There is no sponginess or discoloration of the gums. For some days after admission he complained of weakness, sense of weight at the epigastrium, there was abnormal dulness on percussion to within two inches of the umbilicus. The urine showed no traces of albumen. He was treated with occasional doses of compound powder of jalap & quinine. The ordinary antiseptic mixture of the hospital & a fresh vegetable diet. Under this treatment the constitutional symptoms & dulness at the epigastrium disappeared & he left the hospital quite well on 27 June.

Three other cases were admitted with the patient on the 7th June from the same ship, of the four cases two terminated fatally: General anaemia was present in all of them.

The two following cases are interesting showing the extreme depression of the temperature which sometimes occurs. I have abstracted accounts of them from Dr Babbage's thesis for 1884.
Case III. Lin Bune Sing, chimney, at 36, was admitted to the Tan Teck Seng hospital at Singapore on 7 Dec 86: he had previously been in hospital suffering from diarrhoea & was discharged cured 28 days ago. Two days after leaving hospital he ate a dish of pig's blood, which caused a relapse of the diarrhoea. A week after this his feet became numb & he was unable to walk.

On admission, he has numbness all over the feet & legs, to the hip joint, in the left hand; a feeling of constriction, & numbness, across the chest & abdomen, especially in the umbilical region; pain in the calves of the legs on pressure. He is able to walk a little but is afraid to do so as he feels that his legs are weak & soft & give way under him. His gait is unsteady: he attributes the complaint to his indulgence in the dish of pig's blood.

On Dec 12 complains of tightness & numbness in his chest & abdomen: the hands & legs are numb & painful: the feet are oedematous. Is thirsty, tongue red, appetite bad. Bowels moved three times, watery. Sleeps well at night.

Dec 13 th: had fever last evening which caused a sleepless night.

Dec 14 no change. Dec 15" then take this.
Morning per rectum (for 20 minutes) 96.6°F
Dec 18th is in a low condition. Temp 97°F tongue red dry & clammy, no appetite & has difficulty in breathing.
17th, complains of tightness numbness & pain in chest, numbness in abdomen, pain & numbness in hands & legs, also in his bones & lumbar vertebra.
Vomits, has no appetite; Bowels 3; could not sleep last night for pain in chest. Temp at 7 a.m. 95.4°F (per rectum 10 minutes) taken again at 9 a.m. found to be the same.
18th, pain in bones & body, numbness in chest, abdomen, hands, arms & legs; is in a very low state; tongue red & dry, no appetite, diarrhoea continues.
No sleep. Temp 95.8°F at 9.30 a.m.
19th, temp 95.6°F otherwise the same: on the 23rd the temp fell to 95°F, became delirious & died quietly at 8 a.m.

Case IV. Young Ah Lok, age 37, gambier coolie, ad 22. Dec 84 to the San Dank Sing hospital; had resided in Phohoe for about 2 months when he became sick with intermittent fever & in about five days time he noticed that his feet were getting numb & his legs weak; thirteen days afterwards
CLINICAL SHEET

Patients
Heng Ah Lock

Name

Age
37

Rating

Gender
Male

Date of Admission
22 Dec

Result
Died

Date
30 December

Disease
Beri Beri

Remarks
He remained for about a month, dyspepsia and jaundice, loss of appetite continued, fever & legs ulcers remained.

Temperature (Fahrenheit)

97 98 99 100 101 102 103 104 105 106 107

Time
24 25 26 27 28 29 30
he experienced similar sensations in his hands & had also pain in the pit of the stomach; about two days after his or attempting to get up from his bed he found that he was unable to walk, with a stick he could stand with some difficulty but fell on trying to walk. He has previously had intermittent fever & diarrhoea several times & two years ago while in Hwagang Lake his legs became weak but he got all right in 3 months without medicine.

On admission - is a spare built man, lies on his back with the legs drawn up, says they hurt when he stretches them; also says he is obliged to lie on his back on account of inability to turn on his side. There is numbness in the extremities to the hip & shoulder joints; a feeling of fulness & distress in the epigastrium; pain or pressure over the dorsum of the feet, calves of the legs & the whole of the inner side of the tibia & in the forearm between the radialis & ulna, also in the intercostal spaces; feels as if the tendons at the back of the legs are tense. Expression anxious & distressed, eyes dull, achenes normal, corners of mouth cracked, nose, gums firm, tongue coated with thick white fur in the centre, edges. F anxious inclined, a thick white coating covering the whole of the soft palate & fauces.
25th. Patient is continually moaning, complains of distress all over the body; is unable to get up in bed or move without help. Pulse 114, Resp. 27. Temp. 99°, bowels constipated, appetite bad.

26th. Did not sleep well from difficulty in breathing & sense of tightness in chest.

27th. Is much distressed, voice husky, complains of great trouble in the lower part of his body & of the numbness in his arms: respiration much, laboured, & the heart's action distinctly visible through the chest wall. His hands are semi-flexed & he cannot open them. Complains of pain over lumbar region. Pulse 120 at times almost imperceptible at the wrist. There is slight oedema over the chest wall. Heart's action distinct, the sounds are dull but there are no murmurs, he has no appetite, says that if he eats anything he gets pain in the stomach & a tight oppressed feeling in the epigastrium.

28th. Patient is very bad. Bowels move freely after an enema. Temperature 96° F. (thermometer kept in rectum for eight minutes)

Pulse almost imperceptible. His whole body is paralyzed. Respiration laboured. Died 2 P.M.
The following case presents the variety of the disease known as atrophic Beriberi, the patient was under the care of Drp Pekelhuizing at Batavia (loc. cit. 1847).

Case V 3. A native sailor from the Royal marine steamer "le Zeelee" at 25, had fever when a child & as a soldier 2½ years ago. A year & a half ago he contracted Beriberi at the Moluccas & was, on account of it, invalided out of the service in 1885.

He recovered, presented himself again for enlistment & passed into the service. Sent to Algehi he again took the sickness; after spending 40 days on the "Krispi" on the coast of d'Oeleh-leh he was sent to Padang, paralyzed; after a stay in Padang he arrived on 24th Nov. at Batavia.

State on 24th Nov. 1886: Patient of good frame; face a little puffy; pressure of finger leaves no puckering along crest of tibia nor round the ankles.

Patient experiences pain in arms & legs like pricks of a needle, is very sensitive to pressure on bones & muscles. No difficulty in urinating or defecation.

Eats, drinks, sleeps well. Pulse small & 29th
112 per minute. Respiration 32 per minute.

The right masslabial fold hangs a little lower & is flatter than the left; the right angle of the mouth droops; patient cannot blow out his cheeks when
the lips are closed & cannot whistle. When he shows the teeth the right angle of the mouth droops still more & the commissure of the mouth is more marred to the left than to the right. Type of Respiration is exclusively costal & the diaphragm does not descend even in deep inspirations: the least pressure made with the hand on the belly is painful.

When the right hand hangs freely it presents the following aspect, bent on the forearm in a position of slight flexion, the proximal phalanges flexed on the metacarpals, the middle phalanges flexed on the proximal & the distal on the median: the thumb is flexed & adducted. Patient cannot open the fingers, he can flex them well into the hand, but the power of active extension of the fingers & hand is suppressed. Dynamometer marks 2° in the hand (extreme weakness). Flexion & extension of the forearm at the elbow is possible but is performed with very little power. Movements at the shoulder joint are extremely weak & small in extent. All the muscles are atrophied, the least so are the biceps, deltoid & the pectorals.

Left upper extremity is like the right; the which presents the same aspect cannot be extended.
Thumb adducted cannot be abducted or put into opposition. Dynamometer marks 0. There is marked general muscular atrophy.

Right:  
Left:  

Measurements, upper extremity, upper extremity.
Round the hand over the head 19.2 cm 18.8 cm.
Round the wrist 15.7 15.4
Greatest circumference forearm 24 23.5
Circumference below belly of biceps 24.6 23.5

The inferior extremitiés, of which the muscles are also greatly atrophied, are extended and turned inwards: the foot is somewhat extended, there is a knock knee'd aspect; he cannot move the feet. At the knee the only voluntary movement left is very slight flexion; at the hip the patient cannot however extend the limb when it has been flexed.

Right:  
Left:  

Round the foot over head 23.5 cm 22.5 cm.
metatarsal bones  
Round leg above malleoli 20.7 21.0
Greatest circumference of calf 32.0 31.6
"  of thigh 42.5 42.2
He cannot raise himself to a sitting attitude or when put there he cannot lay himself down.

In a word nearly all the muscles are atrophied movements of the eye are good, the tongue can be moved in all directions; he speaks & swallows without difficulty. All the reflexes are absent except that of the pupils which are equal & reach rapidly & evenly.

Sensibility - the least touch is felt on the face & head & in the mouth; on the chest salicyl etheria is complete. Superiorly, the anaesthetic zone is distinctly marked by a line on a level with the 2nd rib which passes over the right shoulder & turning round the hollow of the axilla passes downwards, in the line of the anterior axillary border, separating the region of the back, where the patient perceives tactile impressions, from the anaesthetic space of the chest & abdomen:

on the left side this line descends immediately along the ant. axillary line without turning round the shoulder. Apart from the back the anaesthetic area extends outwards over the flanks for about 10 centimetres in all directions.

Right arm, sense of touch has almost disappeared except on the back of the thumb, the thenar eminence
In the palm of the hand. Left arm-perception of touch remains only in centre of palm.
In the lower extremities absence of touch is much diminished: patient perceives a strong touch in two regions of the right limb: one is shaped like the sole of boot, extends along the outer side of the calf beginning 10 centimetres above the external malleolus & reaching the ham; the other zone is bounded below by a line 15 centimetres below the groin & extending round the thigh while above it reaches the anaesthetic zone of the belly & is bounded by a line which passes downwards from the sternum to the apex of the groin or then turns upwards to the senssite area of the back.

The sense of touch in the groin is accompanied by pain, probably from affection of the crural nerve.

In the anaesthetic zones patient does not perceive sensations of heat or cold: one can prick him without causing pain, if one takes care not to press the muscles: a strong faradic current is not felt. None of the following muscles are ever reaect to the induced current even though a strong one

- N. radial
- M. ext. pollicis
- M. indic. tensor
Reaction to induced current has diminished on both sides in the following muscles

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flex. radialis dorsi</td>
<td>100 +80</td>
<td>100</td>
</tr>
<tr>
<td>Palmaris longus</td>
<td>+80</td>
<td>+50</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td>+10</td>
<td>+60</td>
</tr>
<tr>
<td>Flexor carpi radialis</td>
<td>+10</td>
<td>+60</td>
</tr>
</tbody>
</table>

Contractions are very slow & persistent.
The sensibility of the ant cranial nerve to the continuous current determined it to be the source of the pain felt in the groin.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>N peroneal not irritable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. tibialis anticus 15 million/sec, E.C.C. Am. 16 million/sec E.C.C.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N radialis not irritable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M. ext. comm. dorsi 20 mA. A.C.C.</td>
<td>15 mA. A.C.C.</td>
<td></td>
</tr>
<tr>
<td>M. ext. comm. dorsi 18 mA. A.C.C.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This suffices to complete reaction of degeneration while in a large number of the other muscles there was partial reaction of degeneration.
In the zygomatic branches of the Facial Nerve & in the muscles round the mouth there was a partial reaction of degeneration. Here, as well as in a certain number of the muscles of the arm, the direct phrenic excitability was augmented.

The organs of Respiration presented nothing abnormal. The Cardiac outline was extended both to right & left; the apex beat, in the xiphoid interspace, was strong; diastole sound double second pulmonary sound accentuated.

The Temperature rose a little towards evening for example 9th Dec, 97.8 97.8 100.4 Fahl.

10th Dec, 97.8 99.0 100.4 Fahl.

No albumen in the urine.
16th Dec, 1200 grams, density 1015; heavy; reaction neutral
17th Dec, 1000 " ; 1020; " ; slightly alkaline.
18th Dec, 820 " ; 1020; " ; slightly alkaline.

Patient succumbed to failure of Respiration without having later presented any great change.

We have here a type of atrophic kerikeri; we remark that the heart was hypertrophied, the congestion dusty, & that there were extensive sensory affections.

In the hydroptic form, on the other hand, instead of being diminished in size the muscles are swollen by hydroptic effusion; moreover the adhesions are more widely spread.
not confining themselves to the crest of the tibia & face they extend to all the cavities of the body.

In the 'mixed form' electric alterations may be very diverse; they may consist in a simple diminution of irritability of nerves & muscles or in an atypical partial reaction of degeneration, though partial typical reaction is not wanting while in 'atrophic herileri' a typical partial or complete reaction of degeneration is the rule.

In 65 cases observed by Herkelots,

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paralysis</td>
<td>60</td>
</tr>
<tr>
<td>Numbness, &amp; feel or hands</td>
<td>57</td>
</tr>
<tr>
<td>Pain &amp; soreness, &amp; feel or hands</td>
<td>48</td>
</tr>
<tr>
<td>L'sema</td>
<td>40</td>
</tr>
<tr>
<td>Spasms</td>
<td>33</td>
</tr>
<tr>
<td>Sait of sheep</td>
<td>11</td>
</tr>
<tr>
<td>Stuttering when walking</td>
<td>12</td>
</tr>
<tr>
<td>Sense of weight in the limbs or thorax as if increased, or had a weight attached</td>
<td>24</td>
</tr>
<tr>
<td>Sensation in extremities of coldness</td>
<td>5</td>
</tr>
<tr>
<td>&quot; hating parts or pneumonia</td>
<td>3</td>
</tr>
<tr>
<td>&quot; tingling</td>
<td>10</td>
</tr>
<tr>
<td>&quot; feel being covered with clay</td>
<td>4</td>
</tr>
<tr>
<td>Copious sweats (3 cases) one general, one over the lumbar &amp; buttock &amp; one in patches here &amp; there everywhere except over the lumbar &amp; buttock</td>
<td>3</td>
</tr>
</tbody>
</table>
D. Bristow in "Diseases of Nervous system" elicits details of a case of Kukke which was under his care in St. Thomas' Hospital, London. The case appears to have presented all the clinical features of Kukke, but there is no reference as to how or where the disease could have originated. It occurred in the person of a female at 30, terminated after some weeks in recovery.

D. Oliver of Newcastle on Tyne reports in the Lancet, 23 July 89, an outbreak which occurred on board the Chinese transport "Soo Nan": the "Soo Nan" left Korea for this country, with a complement of 2640. It was supposed the disease was contracted there: one man died in the Red Sea, one at Suez, three shortly after their arrival at Newcastle on Tyne, but no specific aetiology was allowed, though were still suffering. The disease in this case was of the "wet variety": jaundice, anaemia, numbness, pain, lassitude, loss of muscular power, etc.; a feeling of malaise which lasted for several days, the sailors noticed that their feet and ankles were swollen as also were their faces; there was no anaesthesia nor from fever; it lasted 6 days; the disease progressed into paralysis. Great pain and stiffness was complained of in the hamstring tendons and the muscles behind the knee joint. Great lypogia but for them was much
then to rise from a kneeling to a standing position. Such exercise was always with great difficulty accomplished; in the case of some was so severe that fainting was nearly induced. Breathlessness on the least exertion was a noticeable feature; in all the pulse was small & feeble, the usual average being 72 to 80. Tongue white & coated; appetite good; no brain symptoms. In all cases distinct evidence of dilatation of ventricular cavities was present. All the men attacked were South Chinese.

Dr. Putnam in *Journal of Nervous and Mental Diseases* Aug. 1890 (New York) records a case from a fishing vessel which returned from the Grand Banks off Newfoundland. The patient was a man aged 39 & the symptoms were weakness of both arms & legs, trembling gait, numb & prickly sensations up to umbilicus, arms the same but to a less degree; legs swelled, tender & fitting on pressure. Nine of ten other men who were on the same boat were similarly affected. Putnam sent round circulars to physicians at sea, first towns & they reported that they had met with cases similar to those described. Hearsay by saying that it would seem that in 1887 & in 1889 there were
Epidemics of this infective disease occurring among the crews of vessels fishing along the northern shores of N. America & that sporadic cases have occurred during the interval.


In October 1890 20 cases were landed from vessels which had returned from fishing. Eleven of these were from one vessel which had a complete crew of 13. Of these two died within twenty-eight hours, another died later of hydrophobia & the rest recovered. One had died on the voyage home. The symptoms in all the cases were similar general edema, weakness, numbness of limbs, shuffling gait, inability to bend the knee. Before the symptoms manifested themselves there were prodromata in the form of depression, weakness & disinclination to work.


Pathology. The nature of Beriberi has not
been at all generally recognised
under that name there has been a remarkable
confusion of distinct diseases. In 1882 the
results of a prolonged investigation of the disease
in Japan were published by Professors Baëtz
& Selenge, & in 1889 Professor Pechevsky and
Secteur D'Inkler of the University of Utrecht
published the result of their researches in the
Dutch East Indies, whether they were sent by
the Netherlands government to investigate this
disease. The conclusions arrived at by these
four savants are in complete accord & have
established the fact—suspected so long ago as
1835 by D. John Grant Macklison—that the
disease consists in a multiple peripheral
aneurism, & also that its production is due to the
action of a specific organism. The confusion
which has surrounded the pathology of the
disease is not however completely cleared up
for in 1887 in a report called "The acute
ascenemia of Ceylon or Beriberi" by W. R. Kynezy
we find that a disease characterized by the
occurrence of acute ascenemia & dropsy & produced
as D'Inkler says by the presence of Amylostomata
in the intestines, a disease which in fact is a Helminthic disease, is also the Beriberi of the Straits Settlements and the Karake of Japan; and, in Para 9 of the Report, they (Dr. Kynsey & Macdonald) state that they consider they have identified the two diseases as one. In 1885 Dr. U. C. de B. R. (reviewed Lancet 1886 p. 545) having found amebiasis present in the intestines of people who had died of Beriberi advanced the theory that the disease was due to them, but it has since been clearly demonstrated that amebiasis are extremely common, that they prevail in countries where beriberi is unknown—for instance in Italy, see D. Bosirio Lancet 1890 p. 435—& that when they give rise to disease, paralytic is not one of the symptoms. In 1890 T. P. S. Gill of the Indian Med. Staff published a 'Report on the Nalaazar or Case or Beriberi,' he had been sent to Assam with the special duty of investigating the disease known by the above name, he found that a great many different diseases were brought to him as 'Beriberi & Nalaazar' but he came to the conclusion that the epidemic which was then so prevalent in Assam was identical with Anti-Septic inoculation, or Negi's Beriberi and that it was
an entirely different disease to the Berilere of
Bacilz, Schenke, Petlekhering, Wrinkler, &c.
During his stay in the country, he came across no
cases of true Berilere.

From the fact that Kypros's Report was in English
it has so doubtless been more widely read in this country
than those of Bacilz, Schenke in German or that
in French by Petlekhering & Wrinkler, consequently
a considerable number of medical men are under
the impression that true Berilere or Nalke is
caused by Leptospiroa: thus in Brit. med. jour.
5th Dec 1884 Walker describes "two cases of
Berilere associated with Distoma crusium
and Protospiroa duodenal & other parasites", the symptoms
being anaemia, with dyspepsia in one case, diarrhoea
in the other. So also Surgeon W. F. Thomas writing
in Ind. med. Gaz April 1884 on Berilere remarks
that the only appropriate treatment consists in
the expulsion of the parasites from the intestines.
He evidently refers to leptospirosis. Kyproz
describes the symptoms of this disease under
three headings: 1. Dyspepsie, 2. Anemia,
3. Diarrhoeal, but is will already have
appeared from what I have written so far
in Nalke that the first two of these classes are
conspicuous by their absence. In the Annual Sanitary Report for 1882, Dr. Douglas Clarke epitomizes the account received from various sources by describing the disease as commencing with frequent attacks, ushered in with inquisitive flushed by omitting frequently, ceasing, accompanied by pains in joints, a continued headache resulting in a condition of general anemia with spleen & hepatic enlargement, this is followed by extreme weariness of the mental mental characters affecting the face eyelid, abdomen, feel occasionally by melanoma epistaxis diarrhoea melena aphonia symptoms of large scale bronchial cataract. The disease generally terminates by death from exhaustion... Nearly all the symptoms seem to depend on the intense anemia which shows itself at an early period continues to increase in intensity as the disease progresses.

Comparing this last passage with the results of the examination of the blood of tubercle patients, such as that given in the Table page 48, 49 or those of Wallace Taylor in The Journal of the American Medical Association, 1886, 65-70, 115-119, the differences between the two diseases as far as the state of the blood is concerned will be very apparent.
But if proof were wanting to show that Ankylostomiasis & Kahke were distinct diseases a report will be found in Brit. Med. Jour. 1889 pg 193 by Van Greggel of 5 autopsies on Beriberi subjects in which 20 ankylostomata were found; Beriberi (Kahke) was prevalent in the gols of Burmah 1886-1890, many autopsies were made & a special search was made for intestinal parasites, though they were often found they were always insufficient often to prove that the disease could not be attributable to them. - Surgeon Leslie "On Beriberi & Expres Since 1891.

Dr. Leslie worked out in detail the life history of the parasite which is the cause of Ankylostomiasis. He calls it "Ascaris duodenale," it belongs to the order Nemathelminthes which with the Acanthoccephala make up the order class Nemathelminthes. The worms vary in length according to sex & maturity from 6-15 cm. Many hundreds are often found in the intestines post mortem. The parasites & their eggs are excreted in the stools in large quantities, so means of diagnosis is thus afforded. Leslie also gives in detail the symptoms of Ankylostomiasis, shows how in many ways the parasites get introduced to the intestinal canal & points out the value...
distinctions between that disease & Beriberi.

In the 2nd Med. Gaz. 1 Jan. 79, Dr. F. W. Bruce describes 12 cases of "acute dropsy," which were under his care as civil surgeon at Shillong; the disease he described had spread very rapidly and constituted what has been known as the Calcutta epidemic of 1777-80; according to W. B. Scott (Ind. Med. Gaz. 2 Feb. 80) the disease was known as 'beriberi'; it usually commenced with fever, swelling of the legs, dyspepsia, purpura or petechial spots were common, sometimes redness of the skin; the nature of this disease was repeatedly discussed by the Calcutta Medical Society, but the members could not arrive at a conclusion as to whether the disease was beriberi or not; several medical men who had seen what had been called beriberi disasters were persuaded of the identity of the present epidemic, others who had seen what was probably true Beriberi in Madras (where it is endemic) said the two diseases were entirely different. The epidemic was known to the natives as "parapha" (paraphala, Swelled feet).

Dr. Lorrain, after careful investigation, reported that there was nothing special or peculiar in this epidemic, that its onset was intermittent, fever with profuse sweating, exhaustion, oedema, general
Mr. W. Chambers read a paper on the disease calling it the "Epidermic Fever (acute edema)". Medical opinion as to its identity seems to have been pretty equally divided, but looking at it after this lapse of time it seems to me the disease cannot have been beriberi. It was a fever, it attacked women and children, young and old; it never occasioned paralysis. Sir J. F. Jasper took the epidemic to be that of beriberi in his article on the disease in Quain's dictionary of medicine. He states that the disease is "the sleeping sickness of Africa" (also stated by Morton in his "Life of Clive") which is certainly an error. I presume to say that in Europe parasitic edema is possibly the same disease, (this was also previously suggested by Briers & Verriest) whereas it seems that in some respects the "disease resembles scurvy, it may probably also be the consequence of the cachexia that so often results from long residence in a malarious climate, especially when that has been accompanied by exposure to privation or excessive exhaustion of the vital powers . . . . the resulting partial starvation of the cerebro spinal 'nervous centres & the serous effusion into amongst them sufficiently account for the paralysis which" in these cases, characterizes this disease." I think there must have been some confusion of diseases in
the author's mind; if the disease is "a consequence of the cachexia that so often results from long continued residence in a malious climate" why does it suddenly burst out in an epidemic, as at Singapore, say, during one year & disappear during the following year, & if starvation of the cerebro spinal meninges centres on serous effusion is the cause of paralysis when paralysis occurs, as it undoubtedly does, when acute paralytic crisis occurs, & acute crisis is serous effusion in brain. Case II 1762.

In 1871 & 1874 an epidemic occurred amongst the Indians & other foreigners in Mauritius which was also put down as Beriléri; the disease was an acute febrile one frequently attended by a tubercular eruption & acute chlorosis; it much resembled the Calcutta epidemic disease. Dr. Lovell, the chief medical officer of Mauritius, reporting on the disease (see verbalit. med. fed. laz. Dec 81) concludes that it was even more & not identical with Beriléri; thinks, however, it was the same disease as that prevalent in Calcutta, which he did not consider to be true Beriléri epidemic in Mauritius. The Calcutta epidemic is quoted by Hirsch as an outbreak of true Beriléri; the mortality was small 2 1/3 per cent. I believe he also includes the Mauritius outbreak amongst the occurrences of Beriléri.
Dr. Norman Cheeseman apparently formed his opinion of the disease from descriptions of the two previously mentioned epidemics, for in the weekly *Gazette* of 1884 he is reported as stating that "Bucheri is an "exanthematous" fever, hitherto least known by its "deceptae - acute general appearance & frequently but "not invariably" associated with jaundice & paraplegia."

He accuses Buchanan of having overlooked the occurrence of jaundice in the exanthem!

In an abstract made by Buchanan of 19 cases of patients admitted to hospital with diseases other than Bucheri, but who shortly after admission were found to be suffering from that disease, he states that 5 were admitted for "Fever", 2 for "Phlegmasia", 2 for "Rheumatism", 1 for "Dysuria", 3 for "Jaundice". He thinks that probably all had commenced B.B. when they were admitted.

Post-mortem appearances extensive areas of effusions are present in nearly every case. In one case out of 19 autopsies made by Buchanan there was dropsy about: it was general throughout the muscular connective tissue, particularly at the back of the skin. The case was in the skin connective tissue along the limbs & in the face. Hydropericardium is found in about 67% of cases, hydrothorax in 23% of cases, & ascites in 14%.

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centripetal effusion also takes place into the spinal \*\*\* cerebral meninges & into the joints.

The right side of the heart is usually hypertrophied & dilated, the left side may be also, but not in as great a degree as the right. Dr. Pelchharing & Winkler in 64 autopsies found the weight of the heart to be in 5 cases 8 to 9.6 ozs.

| 42 | 9.6 to 11.20 ozs |
| 10 | 11.20 to 12.8 ozs |
| 7  | 12.8 to 14.40 ozs |

Edema of the lungs is not found to occur alone but is present in some cases with dilatation of the heart.

Small hemorrhages are sometimes found in the heart between the muscles which have also been found in the sheaths of nerves, especially that of the sciatic. Atrophy of the voluntary muscles is usually present to a greater or less extent; the atrophied muscles are of a yellowish brown colour.

Slight hyperemia of the spinal cord has frequently been noticed but Dr. Pelchharing & Winkler did not consider it to be more than was accounted for by venous stasis. The blood dark red in colour congeals readily; clots were occasionally found in the right side of the heart & pulmonary artery.
As far as I have been able to ascertain, the only authors who describe the changes in the peripheral nervous system are Pekelharing and Winkel in their "Recherches," and Gowers in his "Dis. of Nervous System." The morbidity anatomy is referred to also by Ziegler sect. 669.

The changes in the peripheral nerves are indistinct to the naked eye; their existence was proved in 1881 by Baclay Schenke. To whom Pekelharing and Winkel give the credit of the discovery but their work on the subject has not been rendered in English.

Pathological Anatomy. The affected nerves were dissected out, cleaned, examined, and stained with congo red and a carmine; on examining longitudinal sections of them under the microscope, a certain number of fibres were found to be degenerated. The following appearances: the degenerated fibres are slightly thickened, the nuclei of the neurilemma are increased in size, the amount of protoplasm about them is increased and paler; in places the protoplasm has invaded the medullary sheath, meeting here and there across the segment, completely separating the myelinic axis cylinders into globules which stain less deeply with congo red than normal medullary substance.
J. Blood corpuscles with bacteria & micrococci.
A.B. Longitudinal sections of nerve fibres cleared & stained: protoplasm increased & separating nuclei. Basal cylinders.
C.D. Atrophied fibres.
E. Proliferating nuclei & protoplasm.
F.G. Fibres in various stages of degeneration.
H.I. Bundles of stripped fibres; myelin has almost entirely disappeared.

From Phillips & Winkler.
By detrise the myelin is in small fragments, the nuclei cease to proliferate. On the removal of the products of degeneration the sheath shrinks & looks empty in places, but here & there it is enlarged by the nuclei, protoplasm, & a few remaining myelin globules. In transverse section many shrunk sheaths are seen with a few of larger size where they have been cut across at these swellings. The changes are less intense in proportion to the proximity to the central nervous system. Evidence of regeneration is sometimes present.

Changes present themselves also in the muscular fibers; they are irregular in size; large rounded fibers which have lost the appearance of striation lie side by side with normal hexagonal fibers from which the transverse markings have not disappeared.

Except in one or two doubtful cases, Weilharian or Winkler found no signs of disease in the spinal cord. In the doubtful cases there appeared to be a slight vacuolation in some of the nerve cells of the anterior cornua. Dr. Schloovsky, who investigated the disease with Provovovskaya in the University Laboratory of St. Petersburg, found that atrophy in the anterior horns of the gray matter of the spinal cord was sometimes present, the cells being fatty degenerated without my processes.
Transverse sections of fasciculi from Peroneal nerve: degenerating fibres, colored yellow
from Pfeilhauer & Weissler
In malignant cases of the disease are accompanied by degenerative lesions of the Vagus—"heri heri goudrozante" of Francois. Any of the cerebral nerves may be attacked; the facial is oftenest the hypostosed but rarely.

The theory that the disease is caused by an organismal virus now finds general acceptance (Lacerda, Gomers, Schloeszky.)

The following are some of the results of the researches of Pechharing & Winkler at Alten:

1. Microorganisms were present in the blood of all who had passed a considerable time at Alten.
2. These organisms were cultivated in Jepsonized agar-agar, platinized serum, etc., or various media.
3. From about 25 cases, successful cultivations were obtained in 15 instances; of these, 12 consisted of micrococci and 3 of bacilli—2 of each differed from each other and importance is attached to them.
4. Of the 12 successful cultivations, 2 were yellow in colour and 10 white; of these 10, one consisted of a single colony of the members of which, of various sizes, could not be further cultivated.
5. The other nine cultivations grew in agar-agar.
in a thick milky white layer like pyrogenic Staphylococcus albus; they stain with alkaline aniline dyes; grow well in alkaline bouillon at Temp. of the body; resist drying strongly; generally liquefy gelatine - but a cultivation brought to liquefy failed to do so.

6. When animals were subjected to a single inoculation the result was always negative but when subjected to repeated inoculations a nervous degeneration was produced as demonstrated by microscopical examination.

7. From the blood of rabbits so infected cultures were made while an inoculation produced the disease.

Of inoculations so made into 7 rabbits & 2 dogs disease resulted in 6 rabbits & one dog.

A rabbit confined in a heated atmosphere which was kept charged with the micrococci took the disease.

A Water filtrate having been made of 580 cubic ft air believed to contain the virus 35 cc were injected into a rabbit at regular intervals (little by little) the rabbit developed the disease & died.
It was found that dilute hydrochloric acid in 1000 rendered the cultivation in Bouillon sterile. Philharing & Winkler concluded that if the microcosms found its way into the human stomach, it would probably be destroyed there by the gastric juice. The poison exists in the atmosphere as in probably brought into the system through the lungs. "En effet, le microcosme trouvé dans les gaz des lépreux et des nons cultivables, produit chez les animaux une révélation lépreuse multiple."

But it was insufficient to merely show anatomically that nerve degeneration existed in the disease for it might be a result of the malady as it sometimes is in Typhoid Fever or Scoliosis. Philharing & Winkler detected changes in the electrical excitability of the nerves actually before symptoms of the disease appeared & thus demonstrated that the disease is a result of nerve affection.

These conclusions have since been verified by Prof. Ogata of the Imp. University Tokyo.

In the Berl. Klin. Fick 25 Jan 92 reo B. D. F. 6 Feb 92 is an account of the experiments of Moreo & Morelli in the bacteriological laboratory of Monte Video: they also have cultivated the microcosms & have produced general degenerative phenomena.
in animals by subcutaneous injection. In 30 animals the adenomatous form of the disease was produced with dilatation of the heart. Colonies of micrococci were found in the epicardio-myocardium as well as marked changes in the muscula, the musculovascular being destroyed and the myxoma appearing as an empty sheath. The multiple nuclei described by Baclig Schede were found in all cases, namely, a disappearance of the axis cylinders, breaking up of the nuclei in a round cell infiltration in the sheath of Schwann. Regenerative processes were seen side by side with the pathological changes. The peripheral nervous system was affected by preference, but in advanced cases the disease had spread into the posterior columns of the spinal cord with symptoms like those of locomotor ataxy. In most instances however the cord was intact, only in very advanced cases were the anterior horns affected. Shchepkoff experimented in the laboratories of St Petersburg University, and he also a supporter of the parasitic theory, he concluded that the changes in the cord are accidental or consecutive, of very subordinate importance.
Mortality during the cruise of the "Palmaria" brig from Bombay, which lasted for eight months off the S.E. coast of Arabia, then off Maccabees. Nineteen Maries died, and were discharged to the shore not expected to live; it appears that with the disease breaks out at sea it will prove fatal to all affected with it if they are not at once sent ashore. From more than 50 cases there was not a single one in which the leucophraeal countenance and edema of the legs ever entirely disappeared after these symptoms had once been observed; even during the intervals in which the patient felt themselves well enough to return to their duties often before being reported sick they bore a suspicious cast of countenance which marked them out among the rest as affected with a disease from which it soon became the general impression of the crew that no one ever recovered." - W. S. Carter.

Instances of the case of a ship arriving in Japan from New Zealand after a voyage of 272 days: the disease spread so rapidly that 169 cases occurred with 25 deaths. The "Parmelee" in 1861-62 carried cooies from Martinique to Pondicherry, & after a voyage of 5 months she landed 281 alive out of 401. - Borchardt 1878.
Mortality in 1878 was less than 38% of the entire Japanese Army were affected by the disease, as quoted by Hirach.

<table>
<thead>
<tr>
<th>Year</th>
<th>Patients</th>
<th>Cured</th>
<th>Died</th>
<th>Invalided</th>
<th>Remaining</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td>500</td>
<td>356</td>
<td>82</td>
<td>3</td>
<td>59</td>
</tr>
<tr>
<td>1879</td>
<td>590</td>
<td>517</td>
<td>49</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>1880</td>
<td>386</td>
<td>209</td>
<td>144</td>
<td>3</td>
<td>40</td>
</tr>
<tr>
<td>1881</td>
<td>260</td>
<td>212</td>
<td>21</td>
<td>8</td>
<td>19</td>
</tr>
<tr>
<td>1882</td>
<td>338</td>
<td>225</td>
<td>114</td>
<td>6</td>
<td>63</td>
</tr>
<tr>
<td>1883</td>
<td>497</td>
<td>390</td>
<td>37</td>
<td>24</td>
<td>46</td>
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<tr>
<td>1884</td>
<td>455</td>
<td>368</td>
<td>29</td>
<td>32</td>
<td>26</td>
</tr>
<tr>
<td>1885</td>
<td>33</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>1886</td>
<td>9</td>
<td>8</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1887</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

By R. Horinuki, M.B.  
Rei-Kuni 9th. Dec. 1887

In 1881 out of a strength of 28,583 active troops belonging to the Madras command, there were 20 deaths from variolation of 0.7 per thousand of the mean strength. In 1883 the disease broke out in the
9th native infantry regiment; there were 31 cases of which 14 were fatal (Dandham-Goggi Pathel).

It is stated that in India in 1873 the mortality was from 60-75 percent of cases.

Balfour in "Edin. Med. Surg. Journ." 1847 p. 492 says that the natives have suffered to a considerable extent while Europeans have been almost exempt. 2 cases only, having occurred out of the whole force. It has been empirically almost entirely in the stations on the coast explain between 16° and latitude 17°.

Exceptions this rule will be found in the occurrence of twenty-four cases at Salem of which seventeen died; 17 of 31 cases at Secunderabad whereas 5 were fatal of 22 at Hampstead of which died. Several of these however were in men recently arrived from the coast.

<table>
<thead>
<tr>
<th>Admitted</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masulipatam</td>
<td>130</td>
</tr>
<tr>
<td>Vizianagaram</td>
<td>21</td>
</tr>
<tr>
<td>Chicacoole</td>
<td>243</td>
</tr>
<tr>
<td>Rajahmundry</td>
<td>38</td>
</tr>
<tr>
<td>Samuleottah</td>
<td>81</td>
</tr>
<tr>
<td>Vizianagaram</td>
<td>418</td>
</tr>
<tr>
<td>Bokampore</td>
<td>92</td>
</tr>
<tr>
<td>Russelondah</td>
<td>16</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>589</strong></td>
</tr>
</tbody>
</table>
Table of Kakke cases in Imperial Japanese Navy: Table I

<table>
<thead>
<tr>
<th>Year</th>
<th>Force</th>
<th>Cases</th>
<th>Ratio of Cases per 100 Size Force</th>
<th>Deaths</th>
<th>Ratio of Deaths per 100 Size Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>1878</td>
<td>4528</td>
<td>1485</td>
<td>32.79</td>
<td>32</td>
<td>2.15</td>
</tr>
<tr>
<td>1879</td>
<td>5081</td>
<td>1978</td>
<td>38.92</td>
<td>57</td>
<td>2.88</td>
</tr>
<tr>
<td>1880</td>
<td>4956</td>
<td>1725</td>
<td>34.81</td>
<td>27</td>
<td>1.57</td>
</tr>
<tr>
<td>1881</td>
<td>4641</td>
<td>1163</td>
<td>25.06</td>
<td>30</td>
<td>2.58</td>
</tr>
<tr>
<td>1882</td>
<td>4769</td>
<td>1929</td>
<td>110.45</td>
<td>51</td>
<td>2.64</td>
</tr>
<tr>
<td>1883</td>
<td>5326</td>
<td>1236</td>
<td>23.12</td>
<td>40</td>
<td>3.46</td>
</tr>
<tr>
<td>1884</td>
<td>5638</td>
<td>718</td>
<td>12.74</td>
<td>8</td>
<td>1.11</td>
</tr>
<tr>
<td>1885</td>
<td>6918</td>
<td>41</td>
<td>0.59</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1886</td>
<td>8475</td>
<td>3</td>
<td>0.04</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>1887</td>
<td>9106</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

"This improvement, I have no hesitation in believing, has been the result of that improvement in the scale of diet introduced into the Navy, in accordance with my opinion. But some say that the decrease of Kakke cases is not only in the Navy but in every part of the country. Happily there is a weekly death Report for Tokyo from which I have taken out the Kakke cases as made Table II" (second page).
<table>
<thead>
<tr>
<th>Year</th>
<th>1884</th>
<th>1885</th>
<th>1885</th>
<th>1886</th>
<th>1886</th>
<th>1887</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>last-half</td>
<td>first-half</td>
<td>last-half</td>
<td>first-half</td>
<td>last-half</td>
<td>first-half</td>
</tr>
<tr>
<td>Cases of Kakke</td>
<td>758</td>
<td>137</td>
<td>726</td>
<td>202</td>
<td>1272</td>
<td>319</td>
</tr>
<tr>
<td>Total</td>
<td>895</td>
<td></td>
<td>927</td>
<td></td>
<td></td>
<td>1591</td>
</tr>
</tbody>
</table>

This Table shows that while the disease was decreasing in the Japanese Navy, it was increasing in Tokio. Dr. Takaki considered that the decrease of disease in the Navy was due to the changes of diet introduced.

From Report on Health of the Navy by
K. Takaki F.R.C.S. Eng.
Sei-i-Kwan Pechpora Dec 1888
Aug 1889.
Table showing the Admissions & Deaths from Bencoolen for the year 1875 classified according to Nationalities:

<table>
<thead>
<tr>
<th>Nationalities</th>
<th>Years</th>
<th>1875</th>
<th>1876</th>
<th>1877</th>
<th>1878</th>
<th>1879</th>
<th>1880</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Admitted</td>
<td>Died</td>
<td>Admitted</td>
<td>Died</td>
<td>Admitted</td>
<td>Died</td>
<td>Admitted</td>
</tr>
<tr>
<td>Chinese</td>
<td>3-8</td>
<td>5</td>
<td>45</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>22</td>
</tr>
<tr>
<td>Malays</td>
<td>61</td>
<td>9</td>
<td>34</td>
<td>11</td>
<td>1</td>
<td>1</td>
<td>81</td>
</tr>
<tr>
<td>Indians</td>
<td>8</td>
<td>1</td>
<td>7</td>
<td></td>
<td></td>
<td></td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>127</td>
<td>15</td>
<td>86</td>
<td>14</td>
<td>2</td>
<td>2</td>
<td>142</td>
</tr>
</tbody>
</table>

From T. Irvine, Rowell's Report on Bencoolen in the civil jail at Singapore.
Prof. Travers quoting from Kolnische Zeitung says that in the 4 years 1879-1883 there were 17,722 soldiers of the Dutch Army in the East Indies under treatment for beriberi & the casual loss from death & invalidism amounted to 1100 while more than half the force was unfit for active service from the effects of the disease.

The Official Report for 1885 states that during that year 5,388 persons in the colony were treated for the disease of whom 235 died & 847 were incapacitated for further duty.

On 17 Sept 1885 four hundred invalids were shipped from Batavia; during the few days in port before starting the number fell to 364; others were taken daily from Hospital to fill up the gaps. The night before the vessel left eleven died & four being past recovery were left behind. Of the convicts at Pulau Bras 17 out of 20 took the disease. At the Hospital during the first six months of the year there were 1500 deaths & 170 during August. The strongest men in the most temperate in the use of alcohol fell as readily victims as the weak & intemperate.


Prof. Reibelherr & Dr. Trinkle arrived at Batavia on 19 Feb 87; there were there 340 auxiliary troops.
who arrived from Fradoura, where beriberi does not exist on the 12th jan of the same year. they were not in good condition but on the 14th Feb of then were ill - it was said, but during the last week in Feb beriberi broke out amongst them. eleven had to be sent away from Aljeh 10 were dead; in the following week 73 had to be sent away to their homes & 14 others were dead; a week later the remainder were sent away. During that time soldiers who had made good shooting in the morning were dead at night. (From Peterson & Winkler's Researches ch.2)

Surgeon Leslie (or herein) gives the following returns of the disease in Burma; in Toungoo gaol during the rainy season of 1878 there were 53 cases of which 11 were fatal & 11 were released by order of government. In that gaol it was epidemic in 1881 there were 103 cases with 57 deaths & the sickness was not checked till the gaol was converted. At Mandalay the gaol was a new one it was occupied by prisoners in Feb whilst still being constructed; the disease broke out in July & seizures were of daily occurrence until at the end of Sept the population was reduced by the liberation of 87 convicts & the transfer of 301 others to Zoulmaim.

Dr. Wallace Taylor finds that women generally have the disease in a much milder form than men, and many of them affected with rabies have it so mildly that they do not appeal for medical aid. "Of the number of cases recorded in my work both over 16 per cent have been women and an estimate of 10-15 per cent would be a close approach to the ratio of female cases occurring in rabies: over 36 per cent of them have been juveniles. It may occur during lactation but is not then attended with special mortality. Of cases shortly before or after childbirth over 65 per cent have been fatal."

Diagnosis - from other forms of symmetrical peripheral neuritis, namely those following fevers and metallic poisoning, must be distinguished by the history of the case and from the fact of its usually being endemic. Alcoholic neuritis usually occurs in females, hemianesthesia in males; alcoholic neuritis is preceded by marked tremor & is apt to attack the upper extremities rather than the lower. From arthritides by the control over the bladder & rectum being unimpaired. By the absence of tendency to formation of ulcers & by the usual absence of anemia & weakness.
From Acholostomiasis by the absence of profound acedia with enlargement of spleen & liver by the presence of parasites in pulp not arising from the mechanical muscular obstruction of drooping; by the diminished excitability of the affected muscles fibres or reaction of degeneration & by the absence (occasional only) of archiprostomata & their own in the stools.

From Lathyrism or poisoning by Lathyrus sativus in which spastic paraplegia occurs, with ankle clonus & relaxation or incontinence of urine.

From cases of Primary Heart Disease by the sensory & motor affection which usually appear in the extremities before the heart is involved, by the absence of electrical alterations, & by the absence of history or signs of primary vascular disease.

From chronic Rheumatism or rheumatic arthritis by the symmetry of the affection, by the presence of peculiar distribution of edema & by the absence of tendon reflexes.

From Typhoid fever in which swelling of face, eyelids & hands occurs, by the absence of high fever, or by pain being more referable to the course of nerves & less localized in the muscles & by the greater affection of the flexor muscles in Typhoid fever.
With Locomotor ataxia the disease is hardly likely to be confounded: the absence of affection of the rectum & bladder of the muscles of the eye & of the Asyll Robertson papilla, of marked crises and inco-ordination will sufficiently distinguish the disease.

Treatment, a patient's life is not safe while the remains in an affected locality; when the disease has manifested itself once in the constitution it may return on any fresh exposure to infection & prove fatal when least expected, even though perfect recovery had apparently taken place: Patients should therefore leave infected places once for all & take up their residence in inland elevated places as far removed as possible from the sea breezes which apparently suit the disease so well.

The administration of quinine effectually is attended with good results. D'Robertson of the Imperial Chinese Navy has great faith in the free administration of quinine & this is also testified to by Mr. Pearce of Fockow in their Med. Res. Imp. Maritime customs U.P. Sept. 8th 1859.

D'Robertson informed me that it was not particularly the wet or tropical variety of the disease in which this treatment was safe: he is convinced of its utility.
in the early stages of all forms. When the heart is affected a becoming dilated probably no medical remedies are of much value, but in the early stage of the disease probably small doses of strophanthin would be of value.

Pain must be allayed & for this strophanthin seems to be the remedy most generally found useful. The affected parts may also be protected with cotton wool & contractions must be prevented as far as possible by mechanical means.

In the Dutch East Indies prophylactic measures against the spread of the disease have been attended with great success; old wooden buildings particularly seemed to harbour the disease & as could not be burnt were found to be disinfected by sulphur fumigation & rigorous cleansing with acid solution of chloride of mercury. Since these measures have been carried in the barracks & yards of the country the disease has not occurred in any formidable character there.

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