An Enquiry into the Etiology of Beri-Beri.

George Broadfoot Serle.
Beri-Beri, a disease occurring in many subtropical and tropical countries, is recognised to be a form of multiple neuritis, which at certain times is endemic in its area, or may break out in epidemic form. Its chief characteristics being general or local oedema, numbness of certain skin areas, especially over the pretibial region, dorsal aspect of the feet, over the fingers, tips, more or less paraplegia, more or less hyperaesthesia, atrophy of implicated muscles, e.g., calf muscles.

There is also a greater or lesser degree of disease of the heart and circulatory system in general, and a very great danger of sudden death from implication of the muscles of inspiration.

Since Malcolmson in 1835 described Beri-Beri so fully, many are the theories which have been put forward as to its cause. In this thesis, I propose to detail some of the enquiries I have myself made in
in this direction, and in this part of the world, viz. Johore. (See map) The southern part of the Malay Peninsula.

The area of this state is about nine thousand (9000) square miles, with a population of 200,000. Composed of forty Europeans, 150,000 Chinese 35,000 Malays, the remainder being Javanese, Tamils, & other nationalities.

As most of the interior of the country is still covered by forest and jungle, the greater part of this population is confined to districts lying along or near the banks of the rivers & the districts near Singapore on the one side, and the Malayean boundary on the other.

There are three tolerably large rivers, and numerous smaller ones, all tidal for a great part of their course, and it is along their banks that most native towns and villages are situated, and
and by their means that all
communication and trade between
coast and interior are carried on.
The country is less mount-
ainous than any other part of
the peninsula. Its hills are all
detached groups, or portions of two
interrupted chains, running
along the East & West sides
respectively. The highest points
are Mount Ophir 4400 feet, and
Bluemut 3300 feet.
Regarding minerals, the country
is rich in iron, but this is
never worked, tin mining is
carried on in several places,
and gold only in one district.
It is now generally recognised
that whatever be the nature
of the Beri Beri poison, there
are certain conditions, which
have more or less influence in
rendering a person more or
less susceptible to it, e.g. taking
sex as a predisposing cause,
there seems to be a greater
number
number of men affected than women, and this, though somewhat, cannot altogether be accounted for by differences in their numbers or mode of life, taking only into consideration those cases met with in private native houses, where the general daily routine is much the same in both, the disease seems preferentially to pick out the males, and in some houses, where the disease has obtained a footing, I have noticed first one man affected and in the course of some months another & even a third, although the proportion of female inmates was the larger, the exception to this rule being a very marked susceptibility to the disease amongst lying-in women amongst the poorer classes of Malays. This is extremely common, but not quite so common amongst the Chinese, it does not seem to be altogether because of any lowered vitality at this time, as
other diseases, such as Malaria, may lower the system a good deal more and no Beri-Beri follow, and then again it may come on in the next pregnancy to complicate labour and prolong convalescence, if not cause death, and certainly it is extremely rare to get a recovery after a third attack. out of thirteen cases, attended in parturient women, with a history of two previous attacks, there was only one recovery and that after six months.

Occupation, I do not think, of itself, is a very important factor in the causation, it is rather that the followers of certain occupations are housed in a more insanitary condition than these and wherever large bodies of men are lodged together in dampness, closeness, filth, improper ventilation, as in many coolie lines, the disease, if introduced, will play as.
as much havoc amongst them whether they be miners, estate coolies, or vendors.

Age The ages of all admitted into the General Hospital, for the four years ending in 1902, were taken, as well as those attended outside, and the youngest affected during that time, was fifteen years, the oldest 60 years. The following table gives the percentage at the different age periods.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-24</td>
<td>16%</td>
</tr>
<tr>
<td>25-34</td>
<td></td>
</tr>
<tr>
<td>35-44</td>
<td></td>
</tr>
<tr>
<td>45-54</td>
<td></td>
</tr>
<tr>
<td>Over 54</td>
<td></td>
</tr>
</tbody>
</table>

giving its favourite age period as from 25-34 years, or higher than what is generally thought if the age period, 21-30 years, is taken. It gives a less percentage, but only slightly, than from 15-34. The disease mentioned by Kiiota of Sokia, as occurring in children about
about one year old, suckled by Beri-Berio mothers, I have never once seen the symptoms were restlessness, gastric and cardiac distastances, cyanosis, oedema etc.

Nationality. The following table gives the nationality of those treated in hospital here, and the percentage of Beri-Beri cases amongst them for every hundred admissions from all causes, for the year 1899.

Chinese. B.B = 8%. of the admissions

Malay, Japanese. 5.7% from all causes

Portuguese. 2.4%

Benjautis.nil

Sikhs. nil

Arabs. nil

giving at first sight greater susceptibility of the Chinese over every other nation, but considering the mode of living of the ordinary Chinese man, i.e. their making up the greatest number of the coolie classes of this state, and therefore living in crowded club buildings
club buildings, and mostly not under very good sanitary conditions. (The rate is not nearly so high amongst the richer classes and the better housed.) also from the fact that they are the greatest patrons of the hospital, and seek advice more generally. and from the fact that, the Malays are very often treated outside in their own houses. I consider the latter to be just as susceptible, but it is different in this part of the world with Pamil, i.e. Indians from Madras. these often live in even worse quarters than the Chinese & where Ben Ben is life, yet how very seldom do they contract the disease, in fact the figure 2.4% is rather high, this experience is borne out by other doctors all over the Malay States.

Barry in the Indian Medical Gazette for September 1900, says that, in Rangoon the greatest sufferers are the Pamil coolies, who come over from Southern India, to work at the padi harvest.
harvest, and that they are attacked out of all proportion to the Bur-
mans and Mahomedans.
The conditions mainly responsible for the onset, he says, are clam,
isanitary houses & want of nutro-
genous food, the same class of
coolies, living in towns, do not suffer
to the same extent, because of
better sanitary conditions & ability
to procure meat. This is contrary
to experience here & Tertius
Clarke, speaking of the northern
part of the Malay Peninsula,
says, ‘Rare or very rare
amongst the Tamils of Lower Iraq.
I have only seen one case among
the Punjabiis & one in the Arabs
resident in Jofore.
Taking into consideration the
whole circumstances, and at the
same time, remembering mode
of life & surroundings, Tamils
especially, but also Arabs & Punjabiis
seem less prone to take the disease
than the Chinese and native Malaya.
Temperature and Rainfall

Taking the number of admissions for the year as 100, the percentage for the different months, with the mean temperature and rainfall for that month, are as follows (see also chart).

<table>
<thead>
<tr>
<th>Month</th>
<th>Admissions</th>
<th>Temperature</th>
<th>Rainfall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan 1899</td>
<td>127</td>
<td>76.6°F</td>
<td>16.6°F</td>
</tr>
<tr>
<td>Feb</td>
<td>109</td>
<td>78.3°F</td>
<td>2.83°F</td>
</tr>
<tr>
<td>March</td>
<td>187</td>
<td>75.6°F</td>
<td>12.12°F</td>
</tr>
<tr>
<td>April</td>
<td>105</td>
<td>72.9°F</td>
<td>5.95°F</td>
</tr>
<tr>
<td>May</td>
<td>77</td>
<td>82.2°F</td>
<td>4.67°F</td>
</tr>
<tr>
<td>June</td>
<td>51</td>
<td>79.9°F</td>
<td>3.39°F</td>
</tr>
<tr>
<td>July</td>
<td>31</td>
<td>80.3°F</td>
<td>4.74°F</td>
</tr>
<tr>
<td>August</td>
<td>41</td>
<td>79.9°F</td>
<td>4.88°F</td>
</tr>
<tr>
<td>Sept</td>
<td>61</td>
<td>79.8°F</td>
<td>9.42°F</td>
</tr>
<tr>
<td>Oct</td>
<td>81</td>
<td>79.5°F</td>
<td>10.11</td>
</tr>
<tr>
<td>Nov</td>
<td>71</td>
<td>78.5°F</td>
<td>11.11</td>
</tr>
<tr>
<td>Dec</td>
<td>107</td>
<td>77.8°F</td>
<td>13.70</td>
</tr>
</tbody>
</table>

The annexed chart shows how closely the curves of rainfall and admissions coincide with one another, a rise in the number of inches of rain for the month, accompanied or followed by a rise in the admissions.

In February the rainfall is low, and
the admissions also lower, but not to such a great extent, the reason being that, the influence of the nearly January rain is still being felt. Temperature being fairly equal all the year round, a lowering of it that occurs, is due to the rain, the influence is may have, cannot well be ascertained, the predominating influence is the rainfall and consequent dampness of the atmosphere and soil not only on the incidence of the disease but in the aggravation of its symptoms & the raising of its death-rate.

I have marked on the map certain places, where Ben Ben is exceedingly common, acute and fatal, at the same time almost point out, that for the most part, the reason is because these are mining settlements, where hundreds of newly arrived Chinese coolies are sent, to live in overcrowded lines, with no idea of sanitary matters either in their houses.
houses or immediate surroundings, sufficient reasons, of course, for many
diseases breaking out at those spots, and experienced teaches one, that,
localities in the immediate
neighbourhood & otherwise identical
but with no overcrowding etc., may
be perfectly free from the disease
again, the pulling down of con-
gested buildings, the replacing
them by roomier and better
ventilated ones, and improved
sanitary arrangements, have
stamped out the disease, in
places, where previously it was
a scourge. Hence, I think that
locality, per se, (unless it is a
low-lying tract of land, damp &
subject to floodings) has little influence
in determining the incidence of
Blue-Beit.

In the capital town of this
country Blue Beir is most prevalent,
has been for years, in the resi-
dential suburb, where the houses
are built on hills, are large and
airy
array, and occupied by better class government clerks, who live well. There is no overcrowding.

In the uncultivated valley at the foot of the hills, runs a stream, into which defecation is performed by all the inhabitants of this district, and it looks as if locality had something to do with the attacks of Beli Bebi here. But after careful investigation, I found that those attacked were employed in either the government printing or jail offices, which were undoubtedly the centers of the infection.

These then are certain facts concerning the conditions which have an influence in the production of the disease, in the southern part of the Malay Peninsula.

As regards the true cause of the disease, my first inquiries were to try and ascertain if there were anything in the dietary, such as deficiency of nitrogen, the quantity
quantity, the quality of rice, the mode of cooking, or the water and utensils used, the rice theory, at present, being very strongly upheld, experimental feeding with different kinds of rice was tried in some of the hospitals. The ordinary coolie has the following quantity of food supplied to him every day:

**Chinese**
- Siamese Rice, 1 3/4 lbs.
- Fresh fish, 6 ozs., with pork every other day.
- Dried salt fish, 4 ozs.
- Green & white vegetables, 6 ozs.
- Lard, 2 ozs.

This is always well cooked, eaten fresh and hot. Practically no alcoholic liquor, but condiments such as ketchup & vinegar are freely used.

**Malay**
- Rice, 1 3/4 lbs., generally Siamese, sometimes local.
- Fresh fish or fowl, 8 ozs.
- Dried fish, 4 ozs.
- Green & white vegetables & fruit,
Bread, made either of flour or ground rice about 4 ozs., a little tea, coffee & curry stuff, & a fair amount of coconut oil.

The vegetables are cooked in earthenware dishes, the others, generally in iron pots, the whole served out in ordinary English plates, all food is brought to the boiling point, & no alcohol is used.

Samils

Indian or Penang Rice. 1 1/2 lbs.
Wheat flour cakes. 3 or 4 ozs.
Fresh fish. 1 lb. 7 ozs. (sometimes mutton fish)
Dried fish or shrimps. 3 ozs.
Vegetables, e.g. peas & onions, 11 ozs.
Curry stuffs & coconut oil,

Sometimes a good part of this is cooked at one time, so that, some of their food is kept overnight & eaten cold, the rice they use is also sometimes of an inferior kind & may be sour before eaten, native whisky, gin, & rice liquors are largely consumed by many.

Arabs here live mostly like Malays.
Those called Ceylonese eat rice (Indian) only occasionally, their diet being made up of flour, vegetables, e.g., dal, chicken, fish, and a large quantity of fat, in the form of ghee. These quantities are supplied to cooks on estates, mines, etc., but at the same time, it must be admitted, that, many of the same class, not so fortunately situated, have a worse diet than the above, by a very long way.

Still, although, that quoted above shows a fair quantity of rice (except Cabulee) it cannot be said, that from the amount present, the nitrogen starvation theory holds in this part of the world, and the same may be said of the fat, and taking the most affected part, of the capital town of this country, where the houses are occupied by the better class government servants, the food is even richer and more varied, than, that stated in the Malay Table.
But there is another interesting question, which requires answering. That is, is there anything in, or connected with, the rice of kind of rice used by the different nationalities here, which causes the disease? It has been said that Beni Beni affects those eating Samoese rice to a far greater extent than those using the Indian variety, because the poison which is said to be the cause, is rendered inert during the preparation of the latter variety, and the Samoans are put forward as an argument in favour of this. Beni Beni rarely attacks them, the rice they eat is Indian, or native, prepared in the Indian manner.

It may be as well to explain the different varieties of the grain in use in Japan and their mode of preparation. They are:

1) Diamese
This, and much from Rangoon, is sun-dried paddy (rice in husk) threshed by machinery and bagged in large mills, it is eaten by Europeans, Chinese and Malays, in fact it is the same as that used at home, there are several qualities according to rice, and as it is brought into Singapore in large quantities, bought up by Chinese merchants, stored in not over-clean godowns, it is sometimes in bad condition when it reaches the consumer, being damp, dry, sour when cooked.

2. Indian, used by Tamils, the grain here is first soaked in water for a few hours, then steamed or boiled, dried in the sun for a day & the husk removed.

3. Penang, this may be grown in any part of the Malay Peninsula, as it is very cheap and because, in its preparation, it somewhat resembles Indian, it is largely used.
used by Samil coolies, and is often of very inferior quality.
It is first of all soaked for two or three days and slightly fermented
then steamed, dried, and husked.
Since the time Sakaki did so much to banish Beri Beri from
the Japanese navy, by the
introduction of an improved
dietary, rice has been over cooked
again, by different cooks, given
as the cause, either by excluding
nitrogenous elements from the food
by containing certain germs, or
being in bad condition.

Mr. Charles Hose of the Sarawak
Civil Service, says, Beri Beri is
caused by the consumption of
mouldy rice, in which, some
fungoid growth has grown.

J. W. K. Hunter, in the Lancet (1898)
claimed, to have found the
staphylococcus of Pekelharing &
Winkles, in the cooked rice
provided for some lascars, suffering
from Beri Beri, on board a ship.
at Glasgow.
Working on the idea that, Buri
Buri is a rice disease. S.E.R.
R.M.S. (Indian Medical Service)
1910 examined the rice liquors,
consumed in Rangoon, by the lower
class and discovered an organism
in these, and this organism
he also found in damp rice &
in a fritter of yeast, which the
Lokhinese use to ferment rice water
which they obtain from Singapore.
after a time the same organisms
were noticed in the blood of
Buri Buries & in the cerebro-spinal
fluid & serous exudation of the
sciatic nerve & facial motam.
The organism could be cultivated on
rice broth, beef broth, blood, hydro-
thorase and ascitic fluid, the spores
were killed by a temperature of 120°F
after 9 hours. The organism itself
is a diplo-bacillus, and is generally
angular, the spores split into
two and grow out into rods.
The blood for examination was taken
by
by means of a capillary pipette from
veins & cerebrospinal fluid.
One cc. of the cultures on rice & beef
broths, was injected into the peritoneal
cavity of fowls, the temperature fell,
they got thin, unable to run, fell
over, missed their footing, had a
peculiar, uncertain walk, became
blue & died in about thirty days.
on post-mortem examination, the
organisms were found in the blood
there also were, increase of eosino-
phils, white cells, enlargement
of heart, cheesy material & organ-
isms in the kidneys.
Injections with cerebrospinal fluid
in heart blood from cases of Peri
Peri produced similar symptoms
death took place on 22nd or 23rd
day.
In the rice the disease appears
as a fibrous, clammy coating,
sticking the granules together.
and of pike birds fed on this
rice, all became blue in the
comb, weak, pale, listless, with
clammin
brooking wings and loss of feathers, 
had diarrhoea with some blood, 
angular organism was found in 
the blood, death took place on 
seventh day, and on post mortem 
examination, hyperaemia & petechiae 
of gastro-intestinal tract was found. 
If diseased day pears were taken, the 
symptoms were less acute, and 
death only after 50 to sixty days. 
The organism discovered, was the 
size of the Bacillus Tuberculosis 
and so distinguishable from 
Bacillus subtillis. 
The experiments carried out in 
pegs, produced similar symptoms 
and post-mortem changes. 
Bijkmann and Vorderman (vide 
Scheube's Diseases of Warm Countries) 
say that fowls fed on cooked rice 
develop polyneuritis, paralysis of 
the legs and wings, & respiratory 
muscles. Raw rice & starch, should 
have the same effect, but here the 
disease takes longer to set in, and
if the grain, whether cooked or uncooked, is covered with the husk, or if the bran from the inner husk is added to the rice or starch flour, there is no result, and if this food is given to sick fowls they die.

Hickman says the inner husk protects the fowls from the disease and cures it, he therefore believes Ben Ben is caused by a poison generated in the crops of fowls from starch, and the husk contains a material, which renders this poison harmless.

Voderman's experiments in the prisons of Java go to prove the same thing, where pelled rice was supplied to the prisoners, there was one case of Ben Ben to 39 prisoners, with half husked rice, the proportions were 1 to 10,000, and with a mixture of pelled and half shelled, 1 to 416.

In another page I have given tables of the diets supplied to different countries.
cookies, on the estates and mines of this country, and which is much the same food in quantity and quality, as given to convicts in John's jail, and inferior to what was the usual amongst better class natives previous to contracting Beni Beni, and certainly, it does not appear to bear out the nitrogen starvation theory, if this theory were true, then one would expect to see this disease playing havoc amongst the very poorest and beggars, whose diet may be very little else than rice alone, instead of finding it one of rather well fed coolies and servants, the fact is, the nitrogen starvation theory is quite untenable, and against the experiences of all medical men in this part of the world. And again, there are many spots in the tropics and even close to Beni Beni estates, where no Beni Beni prevails, although the diet is similar.
The proportion of nitrogen to carbon, then, may be physiologically correct, may even be above the normal, and yet of no influence in preventing either the spread, or incidence of the disease.

The Chinese diet already given, which contains a good amount of fat pork, does away with the idea that a deficiency of fat is a cause. Captain Barry of the Indian Medical Service, with a large experience in Rangoon, puts his faith in the Nitrigenous starvation theory, because there the disease attacks the Hindu labourers, who come over from India, to work at the paddy harvest, and whose diet does not contain much meat, but, because these men improved when meat was supplied to them is rather a poor argument, especially when at the same time they were taken out their damp, filthy hovels and placed in better surroundings.

In the same way one could argue that deficiency of nitrogen was the cause of
of Typhus Fever and other diseases. It is a very strange thing, that, through-out the whole of the alluvial Peninsula, the Tamils very seldom contract Beri-Beri, even on a diet inferior to that of the Chinese. The fact is, as has been pointed out by Manson and by Scheube, that when Sakizuki improved the diet in the Japanese Navy, he also improved the general hygiene and raised a healthier state of the every day life of the Japanese sailors and soldiers.

Many surgeons in the Dutch Navy, with large experience in the East Indies, have found, that there are no reasons for connecting Beri Beri decrease with improved dieting. Thos's theory of mouldy rice is, I think, out of the question, if this were the true cause. Beri-Beri would be very much less prevalent than it really is, in the territory of those one scarcely ever needs to eat mouldy rice; and certainly no
employees of labour here supplies any thing to his cookies. but the best.
Both Chinese and Malays are very
particular indeed, both as to the
quality supplied to them, if mouldy
rice is eaten by any one, it is only
by Chinese and Tamil beggars,
and certainly there are neither
numerous nor specially affected of
Bee Bee.
As I have already pointed out, Siamese
rice, similar to that eaten in Britain,
forms the staple food of both Chinese
and Malays, and as it is stored by
the rice merchants it may yet
damp and mouldy at the bottom
of the bag, but no employees of labour
serves this out, it is returned if it
is supplied to a government insti-
tution, and the average Malay is
so careful to see that he is not
cheated in any way, to buy it.
Granting that mouldy rice was
largely bought, the process of cooking
would kill such growths off, natives
as a rule, being very thorough in
their
their methods, but the less affected
Samils very often cook but once a day
and like their food sour & cold.
The method, amongst Chinese, is to
boil their rice with water, then steam
it for an hour or more, and to
prepare soft rice and rice water, the
boiling down of the rice takes about
two to three hours, processes which
are sufficient to kill moulds, bac-
teria and their spores.
If mouldy rice were the cause, then
the most affected would be the
Samils, who, after cooking, leave
it for some time and often eat
it when it is quite sour, yet they
very rarely develop Bui-Bui.
This is very noticeable all over the
Malay Peninsula.
The bacterial theory has had, and
has now many supporters, here I
shall describe work and experiments,
conducted by myself, as to the
presence of a specific bacterium
existing in different rices and in
rice liquors.
Many different samples and qualities of each of the three kinds of rice eaten here, were bought from different shops that supplied natives. These samples were carefully received into sterilised, stoppered bottles, specimens, examined microscopically, were therefore of the Siamese, Indian and Penang varieties.

A small quantity of each sample was taken and powdered down in a sterilised mortar and then mixed with sterilised water, and from each were made (1) two unstained specimens hanging drops, two stained with watery iodine-blue, two with carbol puchain (Bueh–Kuban & Wolffer) and two by Gram, all were examined with a Beck microscope using 1/2 inch immersion lens.

After repeated examinations, the only objects, that could be detemined, were larger and smaller spores, round and oval, and in the specimens stained with carbol puchain & ethylhylene blue, what appeared to be micrococci.
came into view, as well as Bacilli, these latter were very distinctly brought out in the Carbol Fuchsin specimen, and especially, here also, were the smaller sized spores.

In detail. The microcoeci were arranged in groups, but were not often seen. The smaller sized spores were very abundant in some of the specimens and the different characters they presented, were curious.

The bacilli were large and spores were seen within them as well as free, the bacilli themselves were either straight or slightly curved, had unstained transverse septa and these were sometimes plainly seen, that, the bacilli had the appearance of two different elements with a clear jelly between, or a chain of elements in the same substance,

Another peculiar condition seen, was, that, they had all degrees of curvature, so that, some appeared only
only slightly curved, others seemed to have one half at right angles to the other half, and between these two extremes, all conditions occurred, there were also dumb bell shapes. Sometimes two appeared precedent to end like diplobacillus, they retained the stain after Gram, produced spores. These latter sometimes narrower than the former, but sometimes swollen up & broader.

These appearances then, coincide very closely indeed with what was seen by Kost in Rangoon: ny: "A diplobacillus, generally seen as an angular organism, which develops by spores, these split into two and grow out into rods." Between the starch cells in rice, may be seen micrococci-like bodies, which are really the spores, the organism is very active and appears also as double and single dumbbells. "The organism is the same size as the B. Tuberculosis."

With this latter statement, I do not agree.
Those seen by me were much larger, otherwise the appearances were much the same, and all the samples examined contained them.

In order to differentiate the above objects at the same time, repeating the experiments of Post, I made the following cultivations.

The three aforesaid pieces were broken down into a coarse powder, in a sterile mortar, a layer of each spread over the bottom of a Petri's capsule, which had been previously sterilised, over these layers some sterile distilled water was sprinkled, and the whole covered.

On all the specimens, there developed a copious growth of a dirty looking fungus with a greenish hue, matting all the particles of rice together, growing into their substance and giving them a dirty grey appearance.

Microscopic examination disclosed long septate filaments, branching in all directions, at the termination of
of some of these there were bulb-shaped swellings & spores. These latter sticking on the end of a stalk-like structure which, in its turn, was fixed to the bulb. There were also many free spores, which were identical with the larger spores found in the films already described.

From the appearance, filamentous structure and the mode of spore formation, I came to the conclusion that the fungus in question was one of the Aspergilli, most probably Aspergillus Nigres.

Distilled water was mixed with other samples and after standing to allow the rice to settle to the bottom, the supernatant water was poured into sterilised flasks plugged with wool. Other samples were boiled for ten minutes in distilled water, so as to make rice broth, this poured into sterile culture tubes, to some of which were added a few grains of rice, while others were left without any such addition.
on every one of them a growth took place, which, on being examined microscopically, stained and un-
stained, was found to consist of spores and bacilli, similar to those seen in the original films and again staining well in carbol-fuchsin. The growth consisted of a white
scum on the surface of the water and after a few days could be caught up on the point of a platinum
needle. The longer these cultures were kept, the more distorted did the bacilli become, producing the many varied forms already described, and noted by Post in his experiments.

From them, growths were got on gelatine, (liquid in this climate), on both, prepared, according to the
recipe of Daniels, from Bovril, (Laboratory Studies in Tropical Medicines page 279) a scum formed on the
surface, leaving the medium otherwise clear, if kept for several days the scum sank to the bottom of the
lake.
but, as another formed on the top, on agar, sloped and in Petri's plates, a dry growth developed on the surface, on potatoes a pinkish colour was produced, and an all, if old enough, the same peculiar irregular forms, already described, could be well detected, but especially after six or seven days.

The conclusion, after a very full examination, I came to, was, that the organism Post experimented with was this same organism and that it is the Bacillus Subtilis, in spite of the fact, that he says it was smaller and about the size of the Bacillus Tuberculosis, and that on rice there are always present a mould, this bacillus and their spores and these latter are not killed by simple boiling for ten minutes, that they were present on all the three different varieties of rice at equal numbers, but mostly present on those of an inferior quality. They were also found in panic, i.e. the husn-decorrelated...
non-decorticated rice grain.
I was never able to grow, on any medium
the staphylococci of Pekelharing &
Winkler, as was done by W.K. Shuler.
From these different cultures as well
as from fresh preparations, made
inoculations into fowls and three
cocanut monkeys, not that I thought
that the mould or bacilli would
cause Ben Beru, but especially for
the purpose of following out to the
last degree, experiments made by
others and deemed by them very
important and at the same time
carry on an investigation on feeding
with inferior and mouldy rices.
In all, controls were kept.
Materials used were:
sterile water, shaken up with different
rices and filtered through a thin
layer of cotton wool.
1. A little of the growths, obtained
on gelatine, beef or meat extract
rubbed up with distilled water.
The hypodermic syringe was of
the usual pattern and was in every
case.
case boiled for a quarter of an hour before use.

The above materials were injected (a) subcutaneously and (b) intraperitoneally. Part chosen was the back for (a) and the lower part of the abdomen for (b).

In the fowls, these pieces were pickled and washed well with corrosive sublimate 1:1000, after further, the wound was sealed with collodion. The monkeys were kept in the open air near others, which I had kept for some time in perfect health. The fowls were in coops, with a small dry run. They were kept some little distance from one another and were scrubbed with jeyes fluid twice weekly.

Regarding the feeding experiments, it was difficult to get the third monkey and the fowls to eat mouldy rice, still, what they did get always contained the mould and the Bacillus, and to ensure success, small quantities of the culture...
cultures, about forty-eight hours old, were mixed with the foods: e.g., smeared on mangoes for the monkeys, and mixed with dry rice for the fowls.

As far as possible, with the exception of food, the conditions of those experimented on, were similar to the controls.

The results obtained by feeding, were very similar, but not at all characteristic of Ber-Ber, the fowls suffered from diarrhoea, stools yellow or green, and after a few days contained slime and blood, the appetite failed, consequently they got much thinner, weaker and unable to walk, feathers got ruffled, wings drooped, combs got nearly white and in some cases exsanguination could not stand on their feet but lay on their breasts, death took place in some cases in a week, others lasted three or four weeks. Provided this feeding was stopped soon enough, they recovered when given
given proper food.
In one set of experiments, healthy
fowls, although not given anything
but rice with husk, placed in the
same coop as those already diseased,
and allowed to move about in the
sun, i.e. under conditions similar
to those affected but fed on unhusked
rice, instead of smeared mouldy,
developed the same symptoms
but of a much less acute type.
In no case was there any peculiar
quit but what could be explained
by weakness, certainly, there was
nothing to point to the disease
having any connection with
Pen Khi, and the post-mortem
examinations bore this out.
There was general wasting, the
stomach and intestine were hyper-
-aemic surnflamed, there were no
organisms in the blood, cerebro-
-spinal fluid, brain, nerves, or
kidneys.
To me the above appearances pointed
to gastro-enteritis from bad feeding,
a kind of fowl cholera. The monkey, fed on the same rice and on mangosteens and bananas, smeared with the cultures, for a long time did not seem to be affected but on increasing the quantities given diarrhoea and oedema of the feet developed, but nothing like Rolt's organism could be found in the blood or nerves. The injection experiments on fowls were very difficult to carry out, in the first place, their skin was difficult to sterilise, a pusulite often developed at the site of puncture, in spite of all cleansing, and this difficulty over, it was a long time ere the right quantity of the culture was found, to inject, minute quantities used and carried on for about fifty days, brought out the following symptoms, gradual wasting and thinning with feverishness in the case of fowls, they got less inclined to move, had no inclination for food and finally settled down in one position to die.
if disturbed they made a few efforts
at running, a half fly and a half jump,
but there was no gradual develop-
ment of paralysis, nor any bacteria
found in the body fluids, and a
"failure to cultivate either the
mould or Bacillus on rice broth.

Of the two monkeys, one died of
peritonitis, and the other injected
with 1 cc of the
culture in distilled sterile water,
developed intermittent fever, loss of
appetite, debility, and died in 33 days,
again nothing was found in the
body fluids.

The symptoms, given above, are not
those of Bub Bub, and in none
of the cases, was there anything
to show, that the animals exel-
fermented on, suffered from
menitis, and certainly at no time
could Bub's bacillus be found or
cultivated after death!

On the other hand, the post-mortem
appearances could all be found in
fowl cholera, or any severe diarrhoea,
a very common complaint in fowls in this part of the world. The fact of the matter is, I think, that Pet was all the time experimentating with the Bacillus Subtilis, that the staggering gait seen by him in his fowls, was simply due to weakness, and certainly because a fowl staggered, there was no reason to suppose it was Ben Ben. The theory of Bickmann and Vodmann, I think, also fails to explain this disease. The Chinese and Malays near fowls in very large numbers, not only for their own use but for sale, the food is always cooked, rice, either what is left over from the owners meals or collected by them from cooks families or institutions. The inmates of White Government Hospital, must eat hundreds during the year, and in all that time, I never saw a case of polyneuritis in fowls. Such an obdurate man as the Chinaman, would have soon found out anything amiss, if such feeding...
affected the health of his forces. That Beri Beri, then, is due to rice, or rice infected with moulds or microorganisms, I think cannot now be accepted, nothing in the way of a specific germ has yet been cultivated, but apart from bacteriological evidence, there are other circumstances, amounting in my opinion to actual proof, that the above article of diet is not the cause. I Beri Beri exists where there is no rice eaten, e.g. in certain islands of the Malay Archipelago, where sago takes its place, and yet Beri Beri is present. It is absent in some places, where rice is largely eaten by all classes, e.g. the greater part of Bengal seven in certain districts of that territory with food similar in every respect to that used by the inhabitants of the most infected areas, in fact a horse may make all the difference. The frequency with which the disease attacks previously healthy Chinese
Japanese, &c, always, when imported into a locality where it is prevalent, the mode of living & surrounding conditions remain the same as before, and conversely, certain houses, institutions & areas of country, which have always enjoyed perfect freedom, may become endemic foci, partly the introduction of a few infective individuals, and again no change of circumstances otherwise.

In the Jh gone government Hospital, the Beri-Beri cases were previously kept at one end of one of the very large wards, the other part being occupied by patients suffering from general diseases, and many of these latter contracted it, but when all the Beri-Beri cases were transferred to a ward of their own and the general ward cleaned & refloored there was no more infection of the other patients. It must never be forgotten, either when considering food, especially rice as a factor, in the causation, that the manner of cooking, the very
thorough boiling it generally gets would kill off all organisms, moulds and their spores. Their \cite{Ritchie1884} say, that "the boiling of any fluid, for one and a-half hours, will ensure sterilisation under almost any circumstances." The Chinese man cooks his rice for at least two hours.

Such, then, is the conclusion I came to regarding rice, and the natural question then arose, was there anything else in the dietary or in the cooking utensils, that could cause it, or was it a form of malaria poisoning?

As I have already pointed out, the inhabitants of Johore, cannot be accused of eating any kind of food in a high, decomposed, or putrid condition, the only exceptions being the Famlies occasionally, and sometimes the Malays eat what is known as "Blachang" composed of decomposed or rather, putrid gruins. But one can leave these out of account, as neither
sour nor decomposed foods are at all generally eaten, they are not in continual use, but only on occasional diets. I have already given, may be therefore taken as in a good and fresh condition.

Regarding the cooking utensils, the commoners class of people eat out of earthenware bowls & plates of a coarse variety, manufactured largely in England & Germany. The better class use English dishes. Cooking is done in iron pots and pans imported from Europe, the ingredients mixed in stone dishes or in cocoanut shells, and the native made brass pots strays only act as carriess of the plates bowls, in none of all these is there any arsenic or anything to cause metallic poisoning except the brass trays, which however never come actually in contact with the food. Fruits, such as bananas, pineapples, oranges, limes, etc. of course are eaten raw, only occasionally are
Singapore or European sweets eaten, but I think, it may be taken, that these having have nothing to do with the incidence of the disease, they are eaten by natives and Europeans and to a much greater extent by the latter.

As regards those articles forming the staple food of all classes, I may say, that in every case after the cooking process was over, they were, as far as could be made out by the ordinary processes of bacteriology, sterile.

I have spoken of meats, such as beef, mutton, pork, fish, fowl, supplied by miners and planters to their cooks, in hospitals to patients, and used by all classes of the general community, were always found by me on inspection, to be in good condition and without a taint of decomposition, and it must be remembered the Malays and Hindus, from religious scruples, and the Chinese
Chinese from choice, especially those of the latter, who have their food supplied by their masters, are very particular indeed in this. The mode of cooking meat is much the same with all, that is, put into an open pan, mixed with varying quantities of water or oil, or first water and then oil (such as coconut oil made by the dhobies and pig fat by Chinese), the whole is brought to the boil and kept at this for at least half an hour, stirring or turning over all the time.

A process of thorough boiling in water is even gone through with the dried and salted fish, and in spite of different German and Dutch theories of dry, salted, insufficiently cooked or infected fish being the cause of Beri-Beri I must say that here experience is against them. I have never seen fish eaten raw or insufficiently cooked and it is a difficult matter to
to see how they can remain infected by either organism or parasite, in an active state, when the cooking process is over.

The disease is found chiefly amongst minkers, who live, in many cases, scores of miles from the sea coast, instead of as should be the case, amongst the thousands of Malayan and Chinese engaged in fishing, curing and salting fish around the coast.

I have visited towns of several thousand inhabitants each, both in Johore and eastern Sumatra, where the sole occupation is fishing & fish drying & curing. I have scarcely seen a case of Blue Line amongst them.

Vegetables such as yams, beans, cucumber, peas, potatoes etc are eaten by all classes, but again are all well cooked in water or oil or both.

She illay, as found in Johore, is a person, who seems to think his
his duty in life is to do as little in the way of work as possible. Consequently, with the exception of some living in isolated villages they depend on their supply of vegetables from the Chinese, who are the great gardeners all over the Malay Peninsula and in Singapore, the consequence is that all classes eat vegetables more or less from the same source, and of all the articles of diet, these, in my opinion, seem to be of far greater potentiality in the causation of Beli Beu, than any other. Because if the disease is caused by a germ, vegetables have every chance of being contaminated with that germ. From the manner of manuring them with manure, faces urine and from the many localities from which they are collected. The gardeners collect the human excreta every morning from every house and institution in the neighbourhood, then store it
it up, it may be for weeks, and apply it to the vegetables as required, but in spite of all this, as I have mentioned, there are the preliminary washing and thorough cooking processes to be born in mind; there is the non-infection of Tamils, huge consumers of the products of Chinese gardens. There is the absence of disease in the very men who grow the vegetables, and there is the fact to be remembered, that pathogene
-ic micro-organisms find it difficult to live in crude sewage and more difficult still, in sewage, that has been decomposed owing to their being crowded out by the stronger saprophytes, which are the agents of the decomposition.

Fruits, as previously mentioned, are eaten by all, and most of all by Europeans, who never contract the disease.

Curry ingredients are never eaten by the race most easily affected, namely, the newly arrived Chinese in the country.
Waters, judging from the public supplies of the two chief towns of the country, has no influence as a causative factor, the disease is not general over these two towns, but simply in well defined foci, and where well waters or river waters is drunk by whole villages in common, the same remarks apply.

Alcohol is never taken in any form, native or European, by the Moham-
edan Malays, and the same may be said regarding the Chinese, very few of these latter drink spirits, unless on rare special occasions. The families are sometimes rather intemperate, gin, whiskey, brandy and arrack being their favourites and the tendency they have to take certain meals cold and sour, does not seem to do them any harm, this does not seem to fit in with the fermented rice longer than they, as their. They should be the most affected race here, instead of the least.
From the above it will be seen, that, I do not think any article of food in itself is or can be the cause of Bari Bari, it is difficult to conceive, in a country like this, where population is made up of several nationalities and that under similar conditions of living, food could be the cause in one localised spot and not over the whole. On the other hand, there is just the possibility that, though those food articles in themselves may be free from all blame, they may be infected just previous to their being eaten, either from the future patient or from the soil etc. Of this I shall have more to say afterwards. Just as there is a danger of organic small contamination, so is there a danger of something inorganic being added and it has been propounded, that many cases of Bari Bari are simply cases of copper metallic poisoning.

In the Liverpool epidemic of 1849.
Peripheral neuritis, where the cases were due to arsenical poisoning, Ronald Ross, after careful study, said, "That many of them, had they occurred in India, would be classified as Ben Ben."

In the alluvial States, around the tin mining centres, it is common to hear tin blamed, and it is true, that metallic poisoning fits in very well with many of the phenomena of this disease. E.g. it could be put down as very like a chronic poisoning, caused by minute doses of the poison absorbed during a long period of time, just as one sees in the clinical history of many cases of lead poisoning, but yet it must be confessed, at the very outset, that again, one is confronted with the most important fact, that metallic poisoning will not explain the peculiar distribution of the disease, and why, in some places with similar surroundings, results are nil.
The following metals are found in the State of Johore.

Gold, tin, (these being the only two that are worked, tin especially), copper, antimony, bismuth (very scarce) and arsenic.

These latter may be found in the soil or in stones lying free on the surface in certain parts of the territory, and are by no means general. They are not put to any use by the natives.

Gold has been worked in one or two spots, for probably hundreds of years, by the Malay, at a later date by the Chinese now only in one place either by Europeans with native labour.

Tin is the principal product, but the country is not so rich in this as the States farther north, as Pahang, Negri Sembilan, Selangor & Perak. The three principal places at present worked, have marked on the map viz. Kota Tinggi, near Muar & near Batu Pahat.
The owners are English but the labourers are practically all Chinese. The mines in Jhorne are situated near the base of, or along the valleys between, mountains, and are all surface, i.e. the soil on top is removed only to a depth of a few feet, say 3 to 20, when the layer containing the tin is reached. This is sandy or clayey, and is simply washed, in long boxes, with water.

Now Bup Bupi is generally rampant of the most delicate type, amongst the workers in mines. This fact is probably the chief reason that Bup Bupi has been described as a form of metallic poisoning.

Of the metals mentioned, arsenic is very often found associated with tin, so is iron, but at Kota Linggi, without doubt, the most affected spot in the kingdom, with these two exceptions, none or very little of the others are found, so that, they cannot
cannot be the cause of Beri Beri in this notorious locality.
As it was possible then, that there might be something in the arsenic and metallic theory and as tin could not very well explain these cases happening in other places, I conducted the following experiments:

I Examination of soils,
   (a) at and around the mines
   (b) in Beri Beri districts, where there were no mines.
   (c) in localities generally healthy.

II Examination of waters in the same localities

III Examination of different foods, sweets, utensils used in their preparation and manufacture,

IV Examination of the urine of Beri Beri patients, at all stages and both in hospital and on the spot.

The metals tested for were tin, lead, arsenic, antimony, copper, zinc, iron & gold.

As previously mentioned, all the above are found in different localities in
in the territory, special attention was given to endemic areas in testing for them, but I could never find any constancy of any one, although the areas were equally notorious, sometimes arsenic would be found, sometimes lead and sometimes antimony. (Iron is generally present).

In endemic areas, but where there was no mining, the result was. in a way somewhat similar, as each one could be detected but owing to the less disturbed state of the soil, the quantities were much smaller.

When it comes to an examination of the potable waters such as wells, one is confronted with a very strong argument against metallic poisoning, whether in town or country and whatever the source, on the whole the water drunk by all classes is wholesome and pure, this is certainly rather surprising considering the habits of Orientals, and
as regards such substances as zinc, lead, arsenic, etc., they were conspicuous by their absence, but iron was detected in some.

Food stuffs, including rice, vegetables, curry-stuffs, meats and even sweets eaten occasionally by natives, in fact all the materials entering into their dietary, were found free of any metallic impurity, looking utensils and dishes actually coming in contact with the food could not be blamed either. On this score, so that it is difficult to see why there should be metallic poisoning in some parts and not in others, and why here any more than at home, and why it should take the form of such a specific disease as Beri Beri in certain countries and not in others.

As Medical Officer, having much inspection of native bazaars, shops and stores, I took every opportunity of examining food material of all
all kinds and in all conditions, times and places and so never found the least evidence of Beri Beri being a form of metallic poisoning. The examination of the urine bore this out, the only changes found after a daily analysis of that, of nearly two hundred cases, were diminution in quantity as a general rule, and occasionally Albumen in bad and prolonged cases, just as one would expect in ordinary cardiac disease. No trace of metals.

Although Ross said that cases in the Manchester epidemic would have been taken for Beri Beri, had they occurred in the tropics, one must not forget that there were points of difference, e.g., conjunctivitis running at the nose, pigmentation of skin, skin rash, vomiting and diarrhoea as well as neuritis, symptoms not often met with in Beri Beri, let alone the fact of absence of profound circulatory disturbance.
The prominent symptoms of Pumbean Dyscrasie, Simplexism and Anti-
- moralism are conspicuous by their absence, and neither will any of them explain well founded
facts, such as, the distribution of the disease not coinciding with the
distribution of any particular metal, nor, the introduction of a few
affected prisoners into a healthy jail or a few convalescent healthy
lines, causing an acute outbreak to take place.

**Microbic Theory**

This I believe to be the correct one, as I think it is the only one
that will explain all the circumstances of the case.

Although the discovery of the specific germ, has yet to be made,
there are so many points in the course and behaviour of an
attack of Pumbean, and its mode of spreading, that one seems to be
dubb to come to no other conclusion.

The following are the different workers.
workers in this direction, and a very brief account of their discoveries. Firstly, come Be kelharing and Winkler, who discovered in the blood of their patients, both micrococci and bacilli, the former of two kinds, viz. white and yellow, some liquefying gelatine and some not. As mentioned before, Houston of Glasgow claims to have found these same staphylococci in the blood of mice eaten by the patients at the time. Different coecci and bacilli have been brought forward from time to time by many other different observers, in Japan and the Dutch East Indies, and often there are the malaria-like bodies of bloognes, found by him in the spleen. Lastly, the haematozoa of Borthiis. Still in spite of all these observations and the work done in carrying out many experiments, no two writers seem to agree, each one having his own particular organ-
I, during the last eighteen months of my residence at a government hospital, made a systematic examination of nearly every patient admitted, I also, so as to fulfill all the conditions necessary for detecting the germ, as mentioned in Pittard and Winkler's book, conducted many blood examinations and made cultures as well, in the very centres of the most affected part of the country. In mentioning these, the usual course of research and as given by Muir and Ritchie in their Manual of Bacteriology, at the same time I may say, that I took care, as far as possible, to cover the same ground as the above two authors. Their work on the subject being more exhaustive than any of the others.

1. Microscopic examination of the blood of patients in hospital, in all stages of the disease, and blood of acute cases, in the endemic area.
area.

2. Microscopic examination of the fluid in brain, spinal cord and nerves.

3. Microscopic examination of the soil of infected floors, and scrapings from the planks of patients' beds.

4. Inoculation of agar and gelatine culture tubes with the same material, smears on agar plates, and plates of agar and blood by Wertheim's Method.

5. Inoculation of monkeys with Ben Benc blood.

In all the utmost care was taken to ensure sterility. The blood was taken either from the ear or the finger only after repeated washing with warm water and soft soap and soda, followed by corrosive sublimate solution.

Stains used were thioflavine Blue, Ziehl Nielsen, Gram, and carbolic Fuchsin. All apparatus was sterilised.

If these precautions be thoroughly taken, the blood of ben bences at
at all times, and whether in hospital or in the endemic area, is sterile. The only abnormal thing, ever seen, was the malarial parasite; the same can be said of the cerebro-spinal fluid, if the patient has died of pure Beri-Beri, but washings of the soil, bed-plank scrapings (beds here are simply trestles with half and 3/4-in. planks placed over them and the whole covered with a mat, rug or blanket) contained fungi, spores, bacteria and micrococci.

Inoculation of the tubes and flasks, in the case of blood, practically always remained sterile, if they did not do so, it was always proved to be due to some contamination, which is very difficult to avoid here, this could be proved by a repetition of the experiment.

The organisms present in soil and bed-plank scrapings, as far as I could see and elucidate, did
did not differ from those cultivated
in any part of the hospital,
sometimes a fungus would grow,
sometimes the Bacillus subtilis
and very often the organisms
of suppuration, Staphyloceccus
Pyogenes Armanas + Albiz. These

certainly could be easily obtained
in the surgical ward as well,
and of course with better methods
and in better hands, more
organisms could be differentiated.
The results of inoculation were
rather varied. There was practically
never, if care was taken, any result
from the inoculation of blood,
even that of the most promising
cases that could be got. But inoc-
ulation of the washings, in des-
tilled water, of plank scrapings
nearly always gave some result,
when injected subcutaneously.
there was either a slight, simple
local inflammation, or a pustule
very rarely were the results more
serious,
The cultures had the same action. As repeated subperitoneal injections of the washings caused death from peritonitis, I diluted them very much and injected in a minute quantity so as to see the effects of a chronic poisoning and if any effect could be produced in locomotion or in the nerves, but without success. I have come to the conclusion, that up to the present, no specific organism has ever been discovered, those brought forward by the different authorities, including Pekelharing and Winkler, I think to be ordinary organisms, either contaminating the skin, or surroundings, from their description and inoculation results. I think Pekelharing and Winkler were really experimenting with Staphylococcus Pyogenes Aureus, Albus or Citrus, but still in spite of the non-discovery of any organism up till now, which one could accept as the
the cause of Bari Bari, I believe an
organisnal theory, the only one
that can fully explain all the
facts of the case, and altho the
bacteriological proof is wanting in
this disease, still there is just as
much reason for accepting it as
most people accepting the germ
theory of say Scarlet Fever on please
where the evidence is equally want-
ing.

1) Bari Bari is communicable, there
is plenty evidence to bear this out;
it can be carried from place to place,
even across the sea in ships, and
if the place to which it is carried
is a suitable medium, it can spread
amongst the inhabitants, one bari
arriving in a suitable
town can infect others and cause
the disease to break out in that
town.

2) Its rapid spread in civilized lines
and amongst people housed together,
even though they may be living
in good hygienic surroundings, as
in government jails, it can break out as an epidemic, more severe at one time than another. (3) It has developed in localities, such as aboard ship, where there could be no tethmic influence. (4) Its development and spread occur best under conditions of high temperature, moisture, close-ness and dirt, thus resembling other infectious diseases and the growth of most germs in the laboratory.

These views are now being held by an increasing number of observers, however much they may differ as to the actual germ itself and its mode of action.

For my own part, I believe in the theory first propounded by Hamilton Wright, that the germ is taken into the mouth with soiled food, Manson says the germ does not live as a parasite in the human body, but in the soil or the surroundings of the patient.
patient a toxin is generated there and this volatile poison, rising up and being absorbed constantly during the day, gives rise to the disease, hence if the patient leaves this area and ceases to absorb this poison a gradual recovery takes place. But against this volatile poison theory are the facts that a change of air and surroundings do not always, even in slight cases, produce this happy result, and even tho' the place gone to is not an endemic area; and secondly, if it were due to a volatile poison, how can the peculiar localisation, seen in the disease, be explained. Medical men here know, and I have seen it, actually confined to a single or couple of cells in an otherwise healthy child. Bases from the earth diffuse a great deal more than only to the extent of a small part of the surface on which a building is erected, the mere fact that nurses and attend-
ants and other patients do not take the disease, is not an argument in favour of the absence of a germ in the human body, it may simply mean, that the disease is not directly contagious, how many cases of Syphoid have been nursed in the general medical wards of our hospitals without spreading to other patients or nurses as long as they take the ordinary simple precautions of cleanliness and care of soiled clothing and bedding.

The fact, that some patients do mind after a change of locality, is a very common occurrence in almost all diseases.

Blood observations dispel at once the idea of anaemia or filariosis being responsible for its incidence.

Repeated microscopic examinations of the stools of all classes of natives, and post-mortems conducted with every care, in nearly every case of death, at a large Government Hospital, force one to admit that
neither the enterocholema, tricophth-
alis dispar nor any other of the
larger intestinal parasites can explain
the very least in the etiology of
Blue Blut.

When one has resided in many of the fishing villages around the
southern extremity of the Ilala
Peninsula and many of those
further north and in Sumatra,
where in some the inhabitants
are almost wholly Ilala, in others
almost wholly Chinese, where the
houses are of the dirtiest and in
the most overcrowded condition,
built on sand, mud or right out
on stakes over the sea and
surrounded by acres and acres
of gutted fish in all stages of
drying, black with flies and
covered with maggots, where the
food is for a great part taken
from the sea and later fresh,
salted or in the above condition
and where decomposed prawns
are esteemed a luxury, without
seeing
seeing a case of Beri Beri, he became fairly well convinced that there cannot be much in either Belcher's or Allen's fish theories.

As I have before stated, the experiments carried out by Dr. Hamilton Wright, at Kuala Lumpur, Malaya Peninsula, and the results arrived at can certainly explain most of what is doubtful in the actual cause of this disease.

After very much elaborate and carefully conducted work, he came to the conclusion that although the food delivered to those experimented on, was free from any known germ, yet there was nothing to hinder it becoming contaminated after delivery and before being eaten. He says the first symptom complained of, is that the patients cannot eat and a feeling of oppression in the epigastrium, the germ gains entrance by the mouth, produces a
a toxin in the pyloric end of the stomach and duodenum and giving rise to an angry inflammation and haemorr-
aphagic injection of the mucosa there, the toxin on being absorbed acts aphrophically on the peripheral terminations of the affrent and efferent nerves, the organism escapes in the facies and lodges in confined spaces through accident or care-
lessness and in the presence of congenial meteorological, climatic and artificial conditions of close association from overcrowd-
ing, the organism becomes virulent and gaining entrance to the healthy body in food contaminated by it, gives rise to an attack of the disease. He says the germ remains focal because he believes it to be destroyed by the action of direct sunlight, or that the presence of CO₂ or other gas is necessary for its
its virile development.
In Kuala Lumpur jail, and this is the rule all through, the prisoners dejecte very often during the night into faeces, which remain in the cells till next morning and the first meal is eaten with the fingers before the daily bath is taken. The experiments carried out by him were as follows.
Monkeys were placed in cells which were supposed to contain the Beri-Beri poison, and fed on bananas, sugar cane and sweet potatoes, these, just previous to being eaten, were rubbed on the floor to pick up the supposed germ. Successful results were obtained, a disease with the symptoms, post-mortem appearances and pathological changes in the nerves etc. of Beri-Beri.
There is no doubt in my opinion, but that Hamilton Wright is on the proper track, one thing about
about his experiments is that they can be easily repeated by others.

The unpleasant epigastic pain mentioned above is in my experience very often complained of by those seeking admission to hospital, and I have noticed the congested condition of the mucosa of the stomach and duodenum in many post-mortems but especially in the early deaths of acute cases and whenever this condition was found there was always accompanying degeneration of nerve fibres and trunks.

I had a very favourable opportunity of carrying out experiments on monkeys near the general building of Johnstone Government Hospital. Here there was a small isolated one roomed house, with a six foot verandah in front, the only light for this room was a baliwick
latticed window, small and situated near the atop covered roof. Just previous to the experiments it had been used for dangerous patients such as major criminals or lunatics suffering from Beri Beri. There was practically no sunlight and the floors were very often damp and the ventilation anything but perfect. Immediately after its closure as a place of human abode, I introduced four monkeys into it. Two others I kept about five yards away in a wooden box house and six feet from the ground but with a chain long enough to allow them to reach the ground on which they took their food, and another pair were kept in two different cages hung high up on the outside wall of the house on each side of the small windows yet about six feet from one another so that there could be no comminu
unequal between the two. Every one of them was quite healthy at the commencement of the experiments. I fed them all on bananas, mangosteens, other fruits and a little rice occasionally, whatever was given was of the same quality from the same source in all cases.

The monkeys outside ate their fruit on the ground about five yards away from the building, one on the outside of the wall, high up, was given good food without contamination. The other about six feet from it was given the same food, but previously having been rubbed on the floor, so that it practically received just the same as the four living in the ward, the only difference being that it was kept high up, just under the eaves.

The results were as follows.
In the case of the two monkeys kept at the five yards distance, even though they always ate their food on the ground, they were quite as lively and active at the end of four months as at the beginning. The one kept outside on the wall and which received good uncontaminated food also escaped. In all three therefore there were no symptoms in life and no morbid changes observable after death.

All the others, that is, the four living inside and the one outside which received the infected food, developed symptoms and either died, or were killed when dying, with an average duration of sickness of three months. The symptoms in life being in all cases the same, firstly it became difficult to get them to take their food, they looked sick and refused to play and became weak and thin, their limbs wasted, were anaesthetic and
and gradually got paralysed so that they were only able to huddle themselves up and sit; this became so extreme that, at last, they could only lie on their sides. Their knee jerks were absent; the heart's action most irregular and just near the end they go so powerless that they could do nothing; they could not take a grip of their fingers nor hold their food and if their arms were lifted up they simply let them fall with a flap. The legs and feet were exactly similar and in each case sensation was also abolished; they felt nothing, the other reflexes as well as the knee-reflex were abolished. In most cases the form of the disease seemed to be of the atrophic variety but in two there was distinct oedema present in face, legs and slightly in the hands. the heart was weak, very irregular and enlarged. The final stage of the disease was paralysis.
paralysis of the trunk muscles.
The post mortem examinations disclosed the following changes:
- enlarged heart with a greater or less amount of clotting on right side, fluid in the pericardial sacs in two, congestion of the lungs, and congestion of mucosa of stomach and duodenum.

The brain substance was normal but in two of the cases the membranes were injected.
In different parts of the cord there was atrophy and signs of degeneration, and these two changes were very prominent in the nerves of the arms and legs, as well as being found in the intercostals and vagi.
Bacteriological examination of the blood, cerebrospinal fluid gave negative results, as tested on agar. Shovel smeared agar & rice broth.

Here then is a disease in monkeys.
monkeys, artificially inoculated, which
is similar in every way to a
disease in man, viz: Bur-Bur
not brought about by a volatile
poison, (as advocated by Blanqua)
as these experiments seem to show,
not seemingly conveyed by flies
or other insects, and not even
brought about by food per se
but only when contaminated
with matter in the immediate
vicinity of persons infected,
matter, which, under certain
conditions tends to be active, such
as dark dwellings and in moist
and close atmosphere and yet
rendered more or less inert where
ever direct sunlight strikes.
When every thing in connection
with its behaviour and symptoms
is taken into consideration, this
'something' the real cause of Bur
Bur seems to be of the nature
of a germ, and one which is
not breathed into the system
but carried in on the food, and
as Hamilton Wright says, produces a toxin in the stomach and duodenum which produces the general symptoms. I think it is on this line that the most successful future work in connection with Beri Beri will be done. Altho the specific agent has not yet been discovered, still the experiments and results described above, viz. the artificial production of Beri-Beri, can be equally well accomplished by any one interested and qualified to carry them out. Results, which to all appearances are positive, will be got, and working on this foundation and at the same time remembering the bacteriological aspect chiefly, will all the mystery of the etiology of Beri Beri be solved.