NOTES ON BERIBERI IN THE MALAY PENINSULA AND ON CHRISTMAS ISLAND (INDIAN OCEAN).

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(With 1 Plate.)

SYNOPSIS.

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I. Introductory.

IN the following pages a number of notes of personal observations are given; with these are some records and observations which have been communicated to me by the kindness of the observers. The literature of beriberi is so great, and at the same time so divergent, and so contradictory are the views and records that have been given, that I have excluded it as far as possible from these notes. When the day of complete knowledge of the nature of beriberi shall arrive it must assuredly happen that many of the hitherto published records will disappear from their failure to mention the circumstances which attended the real cause. Until this time is reached, copious reference to these papers becomes mere picking and choosing what seems to agree plausibly with the writer's ideas, to the exclusion of other possibly most important material.

II. Conditions of Life affecting the Prevalence of Beriberi.

(1) *Insufficient, sufficient, and supersufficient Diet.*

Much has been written on diet in association with beriberi, and the nitrogen starvation theory is well known. Writers on these matters do not appear to have distinguished between the sufficient and the supersufficient in diet, and have confined their attention more to the insufficient and the sufficient. In and about the Federated Malay States one could not help being struck by the circumstance that the well-to-do escaped the disease whilst those that were not well favoured were stricken; and it was difficult not to believe that some constituent in food, when in sufficient amount, had a preventive action, or perhaps,

to put it more accurately, that the presence of an adequate amount of some constituent of the diet had a sheltering effect on the nervous system of the individual. On the one hand, beriberi may spread through a community which is taking a sufficiently nitrogenised diet, according to the physiological standard, as for instance among the long-sentenced prisoners in the gaols of the Federated Malay States. On the other hand, in an endemic focus of beriberi, the disease may be temporarily at a lull, although the diet for some months has been below the physiological nitrogenous standard, as was the case among the coolies on Christmas Island for a certain period[1]. These two contrasting circumstances must, however, be considered fortuitous; and they tend to show that beriberi is comparatively independent of relation to a diet which circles near about the physiological limits. When, however, persons are upon a supersufficient, in place of an insufficient or merely sufficient, diet there seems to be a marked resistance to the beriberic agency. Thus the well-to-do Chinese in the Malay States practically escape the disease entirely; in the tin-mining districts, where the deposits are rich, where the coolies earn much money, where the communications are good, and where the supplies of food are abundant, beriberi is a rare or absent disease.

i. *Christmas Island.*

A good instance is to be seen in the distribution of the disease amongst the Chinese on Christmas Island; they may be classified as follows:

(1) Coolies; (*a*) Sinkhehs or one-year contract men; these men have an advance money debt to work off, and consist of new men from China. They receive $12.00 per annum wages.

(*b*) Lowkhehs, men who have stopped on for a further period at increase pay ($7–8 per mensem), and no advance to work off.

(2) "Tukangs" or artificers (carpenters, blacksmiths, etc.), a superior class to the cooly and well paid. [They sometimes spent as much as $2.00 on a single fruit-pigeon.]

(3) Cooks.

(4) Mandors or gangers.

(5) Contractor's agent and head men.

(6) Hylam "boys" or servants of Europeans.

[1] I should add that the Company's officials have taken steps to prevent a recurrence of this, which was due to the dilatoriness of the Chinese contractor.

All of these with the exception of the last two classes (Class 3 have a house to themselves) live in similar houses (Plate X., Fig. 3), on a fine, dry, open site overlooking the ocean (Plate X., Fig. 1), so that there is no palpable difference in surrounding for the groups. Class (5) live on the same terrace but in slightly differently constructed houses, whilst Class (6) live in the back premises of the Europeans' houses in less healthy positions.

It is a striking fact that beriberi was practically limited to the coolies, who alone were subsistent upon the contract diet. The only other persons affected amongst the Chinese workers of the Company were a few of the tukangs. The following figures illustrate the relative escape of the latter; in the year 1901, whereas there were more than 260 *deaths* from beriberi amongst the coolies, only 6 *cases*, with one death, were recorded amongst the tukangs; in the following year up to October there were 82 deaths amongst the coolies, and only one, a fatal case, is recorded in the whole year amongst the tukangs. It should be noted that there were roughly about ten times as many coolies as tukangs (say 500 to 50).

The contrast is also illustrated by a collective investigation which I made in December 1901; out of 443 coolies (with a small number of mandors) not in hospital, 146 had diminished or absent knee-jerks, and 52 markedly increased knee-jerks, a total percentage of about 45; moreover, just on 10% showed oedema of the shins which was apparently independent of other causes than beriberi. On the other hand, out of the 55 tukangs, 4 only had increased, and 4 diminished or absent jerks, and one only had a suggestion of beriberic oedema of the shins. The tukangs obtain their own victuals, whilst the coolies live on the contractor's supply, which in the case of the lowkhehs may be added to by their private means (especially pork and pigeons).

ii. *Pudu Gaol.*

The following figures, from the records of the Selangor gaol at Pudu, suggest something more than a mere coincidence of low deathrate and of more liberal dietary in beriberi; they are the more worth quoting since some figures have already been published which give a very misleading idea of the prevalence of the disease during the year 1902. I am much indebted to Dr Travers and Mr Galloway for preparing these figures which give the monthly admissions to hospital and the deaths; the former appear too large in so far as admissions and readmissions are

not distinguished, but at any rate during the year 1902 they are too small, for there were numbers of undoubted cases of beriberi which were not admitted, indeed a very high proportion of the prisoners was at one time affected. Attention must be called to the period of eight months from May to December 1901, during which more or less at the suggestion of Dr Hamilton Wright the prisoners were given some 6 ounces of meat every day irrespective of grade; from enquiries that I instituted it appeared that no fish (dry or fresh) was used. It will be observed that during the period there was no alarming increase of admissions, moreover there were only two deaths, a figure which with one exception is the lowest on record in the history of the prison. On January 1st, 1902, the ordinary grade diets were resumed; unfortunately we have no reliable information as to whether any cases of beriberi really arose in the gaol during these eight months. Soon after the return to the old order of things there was an extraordinary rise in the number of admissions in the month of January. Mr Galloway kindly analysed these for me with the following result:—

	December 1901	January 1902
1st week	6 admissions	9 admissions
2nd ,,	4 ,,	12 ,,
3rd ,,	4 ,,	16 ,,
4th ,,	4 ,,	31 ,,
Total	18	68

There was then an alarming increase in beriberi which really began to assume serious proportions some three weeks after the resumption of the ordinary diet arrangements. As will be seen from the table the disease became more and more prevalent, but the figures would be still more striking if all the cases which were merely put on light labour were included. By the end of the year the deaths had exceeded all previous records. Towards the end of the year the disease began to disappear, so that at the end of the following February there were no cases in hospital. Dr Travers wrote to me privately on June 19th, 1903, that there had not been any further cases in the gaol, though there was no corresponding diminution of the disease outside.

At first sight it might appear that the reintroduction of the dried fish had something to do directly with the recrudescence of the disease. But this, as in the case of the Richmond Asylum, Dublin, does not appear to be the case, since a number of those who were stricken did not partake of it. Quite a number of short-sentenced prisoners were

Admissions and Deaths from Beriberi in Pudu Gaol from 1895 *onwards.*

	1895		1896		1897		1898		1899		1900		1901		1902		1903	
	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths	Admissions	Deaths
January	1	—	29	3	31	8	4	—	22	2	12	—	20	—	68	4	22	1
February	1	—	47	3	15	5	3	—	10	1	15	—	9	—	42	7		
March	1	—	43	—	24	3	2	—	9	—	15	1	49	1	62	3		
April	3	1	42	8	3	1	17	—	10	—	7	1	21	2	80	3		
May	7	2	36	5	10	3	1	—	1	—	5	—	11	—	93	6		
June	—	1	39	3	17	1	1	—	—	—	3	—	15	1	83	5		
July	1	—	22	1	12	1	5	—	—	—	11	—	5	—	67	7		
August	6	1	15	1	35	2	6	1	1	—	16	—	18	—	76	2		
September	51	14	50	8	35	6	1	—	2	—	20	2	12	—	131	7		
October	38	7	45	2	35	8	1	—	11	4	17	—	11	1	99	5		
November	21	6	65	3	51	16	10	—	3	—	21	1	16	—	35	2		
December	28	2	45	5	7	—	22	1	4	—	38	2	18	—	55	3		
Total	158	34	478	42	275	54	73	2	73	7	180	7	205	5	891	55		
Deathrate	21·5		8·75		19·5		2·75		9·5		3·9		2·5		6·25			

Feb. 24, 1903. No beriberi patients in hospital.

June 19. "There has not been a case of beriberi for nearly 4 months." Dr Travers.

Note I. From May to December 1901 all prisoners on special diet.

Note II. The figures for admissions in 1902, though including readmissions as in other years, do not represent the total number of beriberic prisoners, inasmuch as there were very many cases which were excused hard labour, etc. but who were not ill enough to be admitted to hospital.

Note III. During 1902 there was no special prevalence of the disease outside the gaol.

Note IV. Gaol population is about 400—500.

affected and the item is not on their dietary. The facts at disposal are not sufficient to adjudicate between coincidence and causative relation in the effect of the change from supersufficient to sufficient diet, they were however suggestive of the latter.

(2) *Racial Distribution on Christmas Island.*

The class distribution on Christmas Island has already been mentioned, but there is a further aspect of the distribution in respect to race, which deserves attention both in this locality and in the Malay States.

The population on Christmas Island consists of Europeans (about 12), with a few Eurasians, Chinese (about 500—600, mostly Cantonese and Khehs, with a few Hokkiens and Hylams), Malays (about 20), and Indians, Sikh police (about 12), and Tamil coolies (who at the time of my first stay on the island were nearly 40 in number); there were also a few units, such as Bengali and Cingalese.

As has already been noted, the

1. *Chinese* suffered severely from beriberi.
2. *Tamils* also suffered from an epidemic.
3. *Malays* only give record of about half-a-dozen cases, of which two were fatal in the period from April 1901 to March 1903. I am indebted to Dr Giddy for these and other records of the island.
4. *Europeans* and *Eurasians* apparently escaped[1].
5. *Sikhs* did not suffer.

The *Bengali* was stated to have been attacked, and the *Cingalese* had a severe attack.

[1] As a personal experience, I may note that after about a month on the island numbness down the front of both shins occurred, about 3 weeks later this had extended to sides of calves, with quite marked, though slight oedema; about 3 weeks later still the numbness had gone, the oedema lasted some time longer, and disappeared about the time when the knee-jerks, which had been markedly exaggerated, became more normal; on the supposition that the collective examination of the coolies, which was done on five consecutive evenings, had any connexion with this, the "period to discovery" (*i.e.* incubation period + days of disease which elapsed before recognition of symptoms) would have been between 19 and 24 days. About the 6th week a short run gave an increase in pulse-rate of from 80 to 152, whilst two controls (European and Tamil) gave respectively 90 to 120 and 78 to 138.

(3) *Racial Distribution in the Federated Malay States; with general notes on the habits of the various races, especially of the Chinese.*

In the Federated Malay States the *race distribution* is on the whole similar. Beriberi is unknown or practically so amongst the *Europeans*[1]; it is not usual amongst the *Eurasians.*

I met with one case in an Eurasian of Dutch extraction and examined his wife and family, none of whom showed any suspicious sign, or admitted suspicious history.

Sikhs likewise escape the disease, both in their outside life and almost always when confined in gaols. Many of the native warders in the gaols are Sikh, and though exposed in day and night hours to hypothetical beriberic effluvia, they never contract the disease (the same is true of the Bengali and Sikh attendants at the Singapore Lunatic Asylum).

Sikhs are mostly employed as police, watchmen, and the like, there are also a few independent carters. Their food consists of wheaten flour, as chief staple, which they usually make up into a sort of damper or "chupati"; not much rice is taken and that generally as "canji," *i.e.* rice boiled in water to a thin porridge. Of animal food, pork, mutton, goat, and fresh fish; several men informed me that they do not like dried fish and practically never eat it. Vegetables and fruit complete the list. Tobacco is not used. Alcohol is often used to excess.

Pathans are mostly in similar circumstances as the Sikhs, they too do not appear to be liable to beriberi. I saw one case in gaol.

I have no exact notes on the habits of the Pathan, but in general he is less particular in his diet than the Sikh, for it is not regulated by the same religious scruples.

Tamils as has been pointed out are very rarely stricken with beriberi except in institutions.

[1] One casual observation may be made here since it is so strikingly in conformity with an observation I made at Parà (*Report of Yellow Fever Expedition to Parà*, p. 59, Liverpool, 1902). A well-to-do Chinaman told me that the Chinese did not care to stay more than two or three years in the hot moist climate of the Malayan Peninsula, and that they endeavoured to "cool down" in China at similar intervals. The ways of the Portuguese on the Amazon agree then with the ways of the Chinaman, and be it noted that a considerable proportion of the wealth in the Malayan Peninsula is in Chinese hands. The Briton stays out in these climates for much longer periods without a break, and I must admit to a sense of disappointment at the lack of enterprise and energy which appears to me to obtain in them. It seems that the "West Coast of Africa" is the only part of the equatorial belt (within 10° N. and S. of the equator) which we treat with a sage respect in this wise.

Tamil coolies come over to the Malay States in considerable numbers; they are a shifting population; immigration contractors are said to allow 25 % women, in order to induce the men to stay. They are employed on agricultural estates, and in road-making, &c.; but very few are employed at the tin mines. They come to hospital with readiness, so that as Dr Braddon has pointed out their ailments are well represented. Their food is largely vegetarian; the rice which forms the main staple is husked in a particular manner, for the paddy is steamed before the husking; this leaves the thin pericarp and testa (zilvervliesje of the Dutch) attached to the grain; this kind of rice is however not universally used, for on the score of cheapness a certain number of Tamils buy the ordinary Rangoon or Siam rice, which is milled without the preliminary heating. Before cooking the rice, Tamils were observed to remove dust, etc., by winnowing, sometimes without washing in water at all. Braddon has sought to find the escape of Tamils from beriberi in their usual habit of eating this kind of rice; Vorderman and others in Java, on different grounds, have sought to show that the "zilvervlies" has a protective effect, especially in the polyneuritis of fowls.

Currystuffs (tamarind, coco-nut, chillies, etc.) form an important item in the diet, which frequently contains dried fish, but rarely meat. The Tamil is not at all particular about the water he drinks; he is much given to alcoholic excess. He also smokes tobacco and chews "betel" (fresh leaves of the betel pepper, with scrapings of the areca, or "Pinang" palm-nut and gambir). His personal cleanliness is considerable, in conjunction with which he oils his skin. He generally lives in small or tiny huts on the ground, or in long, wooden sheds divided into small cubicles. Naturally the above notes do not apply altogether to the better class.

Tamils do not associate much either with Malays or Chinese, for there is mutual racial contempt between them.

Sewage disposal is casual.

Malays are occasionally subject to beriberi in their home life, but owing to their want of appreciation of European medicine they do not frequently come to hospital or consult European doctors, so that it is not possible to gauge accurately how widely spread the disease may be amongst them. It may be noted that they have a considerable contempt for the Chinese, and so do not associate with them. Besides Peninsular Malays there are a number of immigrant Malayan races, as from Sumatra, Java, and Borneo.

The staple diet of the Malay consists of rice and fish, the latter being usually dried, with currystuffs, vegetables, etc. Fowls, goat-flesh and beef are also eaten, but no pork, on Mohammedan principles. Curry[1] takes some work to prepare and several Malays living *en garçon* have told me that they rarely made curry, the fish is then either fried in coco oil, or merely scorched or toasted over embers.

[1] The stimulating effect of curry may perhaps be beneficial. I noted that the beriberic Malays, that I saw, gave histories of not having had much curry. The other curry-eaters, Tamils, likewise escape outside, but not in gaol. Both are "betel" chewers.

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Sewage disposal is casual.

The Malay is a strict teetotaller; he smokes tobacco in cigarette form and chews "betel." His house accommodation is generally small, either on the ground or raised on posts. A few renegade Malays take to opium, as also a few to drink.

Chinese may be classified in several ways for our purpose. It is amongst them that most of the beriberi occurs. Thus they may be divided into the *rich* and the *poor*, with an *intermediate group* (mostly artizans or small shopkeepers); it is amongst the poor that beriberi mainly occurs.

Again, they may be classified according to their length of residence into *Sinkhehs* (new-comers on one-year contracts from China, who are considered the chief sufferers from the disease), *Lowkhehs* or longer residents. Amongst the latter I have met with many cases of beriberi which gave a history of having been 10 and even 20 years in the States and often that they had never had it before. *Straits-born Chinese*[1]: these are much fewer in numbers and by repute beriberi is practically unknown amongst them (I have asked several Chinamen to what cause they attribute the escape of Straits-born Chinese, and they always replied that they are more careful in regard to what they eat, which perhaps is doubtful in the case of the poorer ones). And lastly *Towkays* or wealthy Chinese, amongst whom beriberi seems to be unknown.

When classified by *race* the most prominent point that is to be noted is that the *Hylams* are not attacked at all commonly; the majority of the Hylams are employed as domestic servants, there are also some engaged in fishing on the coast. I only met with one case in a Hylam "boy"; his master became ill (ague) and went away to recuperate; about 20 days after his departure the boy became ill with numbness and weakness, eventually he was unable to walk and had some attacks of dyspnoea, one of which was fatal. It may be added that three other servants were left in the house but none were affected so far as I could learn. Whether the absence of scraps from the master's table had anything to do with the illness it is of course impossible to say.

Anyhow the salient points in regard to Hylam "boys" are that they are in association with Europeans or better class Chinese, and have many potential possibilities in their dietary.

In race, the other Chinese are mainly *Cantonese*, *Khehs* and *Hokkiens*;

[1] The escape of Straits-born Chinese is very hard to explain, unless it is similar to the "escape" of natives in yellow fever zones; anyhow curry eating is not widely spread or constant enough to explain it.

but classified thus they do not give any sign of special distribution of the disease so far as I am aware.

Female Chinese. In regard to *sex*, there is but little to say; females are proportionately few to the males, but inasmuch as there are over 20,000 of them in the States (Census, 1901) there would be enough to yield cases of beriberi; but the Chinaman is shy, and generally speaking the illnesses of his women do not come within the ken of the white man; an exception must be made for a certain number, in that a considerable number of women live in brothels, and some of the keepers have a system of medical inspection. So far as could be gathered, beriberi is very far from common, notwithstanding that they live herded together; on the other hand their employment is fairly lucrative and they follow where money is plentiful; it may be supposed that they are not usually stinted in food.

Locality of Residence. A separation may be made by division into *town* and *out-station dwellers*, so far as the Malay States are concerned (*e.g.* not true of Singapore). It is most striking that beriberi is not rife in the towns; it is from among the mining camps that cases come, and ward after ward of beriberics may be examined without meeting with cases from the towns, all give a history of coming from some mining station.

It would appear that the history of many places is, that a settlement is started, soon after work has begun beriberi crops up, and increases to a dreadful extent; the communications and means of transport are improved, and beriberi ceases to be a terror. This may be applied to the States as a whole, where according to accounts the fatal importance of beriberi was much greater than it is nowadays. On a smaller scale the same may be said of Bentong (Pahang) which was ravaged, but now that better means of communication have been opened the disease is less; the same too may be said of Christmas Island. There is however at least one qualifying consideration, which is that in opening out new places the crowding and herding together may be excessive, the accommodation may be bad, the food may be deficient in quantity and in quality, but a *comparatively large number of Sinkhehs* (*new-comers to the country*) is introduced: the Sinkhehs both by repute and by apparent actuality are especially liable to contract the disease.

It may be noted here that all Sinkhehs pass a few days in depots in Singapore whilst their registration is being effected; and it is difficult not to believe, from an infective or contagious aspect of the disease, that Singapore is responsible for a more or less constant stream of

beriberi carriers[1], who afterwards may spread the disease locally. It is quite an interesting point that in the Malay States the disease should be so much associated with out-station life whilst in Singapore it is a town disease; especially when it is remembered that a majority of those that come up from the latter place to the former are distributed to the out-stations rather than to the towns.

Work and Trades. Another mode of division is by the nature of the work or trade; this is a large and not very fruitful mode. Two points only are mentioned here. One is that the cooks or those on the kitchen staff, even in gaols, habitually escape.

A somewhat interesting case on Christmas Island may be noted here; a cooly, who was a friend of the cooks, slept in their house but worked and ate with his fellow coolies; he became beriberic, none of the cooks did, the period covered being five months. The likelihood of an occasional nocturnal gamble in the other houses cannot be excluded.

The other is that of the coolies at the tin mines. The men spend all the day except for mealtimes in the open air; the "mining" consists in working alluvial deposits which is chiefly surface work; the men seem to be of cleanly personal habits and do much bathing. (Sinkhehs are sometimes driven regularly to bathe at 4 a.m. as a hardening process.) They may be crowded in numbers such as 60 to 80 in a single large shed, or three, four, and more may live together in smaller houses. Where accommodation is limited there may be two occupants to each of many of the bed spaces; on a single visit[2], some such houses I have seen looked very favourable for the dissemination of a contagious malady, but only one or two cases could be found, though a neighbouring more reasonably filled house abounded with beriberic cases. These "kongsi," as they are called, are built on the ground without flooring; the side walls are more or less permeable for air by gaps and interstices between the boards or palm leaves, with either or both of which the construction is made. The larger kongsi are divided up by partitions, which though not roof-high cause some stagnation of air. Bed platforms of planks some 3 feet above the floor are fixed across and along. The

[1] In the event of the importation of Chinese coolies to South Africa it might be well if the emigration agents avoided bringing the men through Singapore, or at any rate insisted on stringent measures being taken in respect to the condition and crowding of the transmigration depots in that town. The introduction of a number of beriberics amongst the African mines might prove as disastrous there as it appears to have been in the Malay Peninsula and on Christmas Island.

[2] Even repeated visits to a given locality will not help much in such cases, for the population may shift and become impossible to trace.

Chinese cooly almost invariably uses a mosquito net, and moreover seems to keep it in order. In a large kongsi, generally too in a small one, one corner is reserved as kitchen. The roof has a high pitch and is thatched with "atap," or other sort of palm thatch. With the reservation made above there is fairly free ventilation.

There is generally some accumulation of dust and rubbish about the floor, but hardly enough of such untidiness to merit the appellation filthy, which I have heard applied. Notwithstanding the amount of standing water about the kongsi the coolies seem to be remarkably free from malaria. Streams of water or pools from old diggings are near at hand for bathing; plenty of water is a necessity for washing out the tin sand, and up in the hills quite elaborate systems of conduits are to be seen to bring the water to the tin ore deposits.

The staple food of the Chinaman consists of rice, fish, pork, and vegetables. In the case of the Sinkheh and in the poorer districts, that is where worked-out or poor land is being "mined," the fish is practically always of the dried variety, and pork is generally given out once a month, rarely twice; the diet is supplemented with vegetables both fresh and preserved (salted, pickled or dried); the kapala or headman does not always get the best quality either of rice or of fish. In districts where there is more money to be obtained the worker is able to supplement the fare supplied to him. When working under contract to a kapala or when as in many places small sets of men agree to work together and share the results of their work every month, they live more or less independently and each may cook for himself or one may remain to take care of the house and see after the commissariat; this may be by rotation or one may be permanently the cook. In richer workings the men have pork every day as well as some dried fish, which is rather eaten in small quantities as a relish than as a real food: on festive or propitiatory occasions fowls, etc. are added to the menu. About the workings there is usually at least one patch of garden, where sweet potatoes and other vegetables are grown. From what I have seen in the Malay States the ordinary tale that a Chinaman will live and work on a "handful of rice" is by no means true. In one working I visited far away in the jungle the men were in the habit of getting three days' supply of pork and parboiling it at once in order to make it keep.

Many other foods might be mentioned in connexion with the diet of the Chinese in the Malay States, such as peas, beans, a sort of vermicelli, sauces, etc. Certain kinds of beans are made into "beancake," a sort of jellylike mass; beans are also allowed to sprout before they are cooked by boiling ("tow-gay"). The Chinaman is fond of sugar and sweet things.

In regard to cooking by the Chinese, the dried fish is usually fried in fat or oil directly or after washing with water; occasionally with certain kinds it may undergo a preliminary boiling in water, but this does not appear to be a common practice. In the Perak State gaol (Taiping) by routine the dried fish is always steamed before being fried: this was a difference between this and the Selangor gaol, which seemed to be a circumstance connecting the prevalence of beriberi

at the latter place and its absence at the former with the mode of cooking this article. Pork is boiled. Green vegetables are more or less fried in fat or oil. Rice is mainly cooked with the water which does not drain off after washing three times with water; in the form of "canji" or "bobor" it is boiled with water.

The Chinaman is very abstemious from alcoholic drinks; he chiefly drinks boiled water in the form of tea.

He smokes tobacco in pipe, cigarette or cheroot (some of this tobacco is stated to be arsenicated in the curing process, the smoke of Chinese tobacco usually has a peculiar, somewhat garlicky odour). Opium smoking is commonly practised, occasionally to excess; opium can also be obtained in "tabloid form" of Chinese manufacture; some of such preparations are vaunted remedies for curing the opium habit. A Chinese interpreter informed me that for a few cents hypodermic injections may be obtained in certain places in Pinang and Singapore.

Sewage disposal consists in collecting the material and using it in crude form on kitchen gardens. That this is not contributory to beriberi is shown by the fact that the Christmas Island coolies had practically no fresh vegetables, and also by the fact that the "refractory" races eat these vegetables also.

He does not chew betel. Only a few Chinamen who have long resided in the States, with whom may be included some of the Straits-born, eat curry.

Japanese. Quite a number of Japanese women and girls reside in the Malay States: they are employed in brothels. Their employment is lucrative, for they follow to districts where much money is being made; on the other hand they leave a district as soon as the tin ore becomes scanty. The number of Japanese in a place gives some criterion of the value of the deposits which are being worked. It may be inferred that their dietary is not stinted.

Whilst beriberi does occur amongst them, it appears to be quite uncommon, though Japan is quite one of the centres of beriberi and though they are associated with the Chinese in the Malay States. One girl with well marked beriberi was seen in hospital at Ipoh, the history given was suggestive of the disease having been acquired in Hongkong or in Singapore. Another patient, not beriberic, gave a history of having been laid up with beriberi in Hongkong. Three brothel mistresses of three, four and ten years' residence in the Malay States said respectively that they had met with one, one and three cases of "kak-kè" (beriberi) respectively during the time. Dr Travers informed me that though he had seen cases amongst these women, it was not common. It may be added that when ill they consult European doctors, so that if the disease were at all prevalent amongst them it would be known. Some enquiries concerning diet did not elicit much information. Whether their escape from beriberi is due to the relative infrequency of the disease amongst females, which is asserted by most authorities, but denied by Grimm, is a question which cannot be answered here.

(4) *Review of the Habits of the Natives in these Parts.*

In this short survey of the ways and habits of the different classes of the population in the regions visited, two main distinctions may be made according to diet; namely, the WHEAT eaters and the RICE eaters, that is to say those whose farinaceous mainstay consists respectively of these cereals. To the former belong the *Europeans*, the *Sikhs* and *some Eurasians*, and amongst them the incidence of beriberi is *extremely low*. To the latter belong the *Chinese*, the *Tamils*, the *Malays* and some *Eurasians*, and it is in and amongst these races that the incidence of beriberi is *high*; but even here the disease is not uniformly distributed, for whilst the Chinese and Malays are affected in their natural mode of life in the place, the Tamils practically only suffer when confined in gaols and the like. Though it is to be admitted that the Tamils, for the most part, consume a rice which is prepared in a different way to that which is eaten by the Chinese, the assumption of a rice theory to account for the disease does not remove the following obstacle, namely, that many of the Chinese likewise escape though constantly consuming the so-called dangerous kind of rice. Thus the town-dwellers and the better-off classes are not afflicted, whilst those who are in less good circumstances or in more inaccessible regions are not so lucky. (I only heard of one case in a "datoh" or leading *Malay*, but unfortunately I was not able to investigate it.)

(5) *Differences in the amount of Phosphorus in the Foods.*

When looked at from the broad point of view it is difficult, if not impossible, to renounce all idea that food and beriberi may be linked together somehow, and many of the published records are in favour of some connexion, though as a rule the writers are adherents of one or other theory which incriminates some particular article.

The records of the Pudu gaol show that the old theory of *nitrogen starvation* cannot be upheld, and the temporary lull in sickness in the endemic focus at Christmas Island during a period when insufficient nitrogen was being given is a further item against such a theory; though many of the coolies actually had beriberi at this time, it did not become severe enough to incapacitate them from work.

There is another constituent of food which has not, I think, been referred to in connexion with beriberi, but it may be observed that the

wheat eaters and the well-to-do obtain more *phosphorised* matters in their food than do the poorer rice eaters. The suggestion may be made that a sufficient amount of assimilable phosphorus in the diet has some power of fending off the essential beriberi poison or of protecting the nervous system from its action.

It is known that the addition of fats to the diet is not without importance in beriberi, but I have not been able to discover whether the presence of these bodies has any direct influence on the absorption of phosphorised compounds; it is however perhaps suggestive that the carnivorous animal passes ingested or injected inorganic phosphorus compounds through its system by the kidney, whilst the herbivorous animal passes it almost entirely by the faeces; the same is true of the glycerophosphoric acid (see Bergmann).

The statistics of the phosphorus content of different foodstuffs are mainly those in which the estimation was made by incineration, a method which would be calculated to leave much organically combined phosphorus out of account; possibly these are the more important, *e.g.* lecithins. I made a single estimation from some of the dried fish (one of the Scombridae) by oxidising with nitric and sulphuric acids, with addition of potassic chlorate (Halliburton); eventually iron was eliminated by means of citric acid and the phosphoric acid weighed as magnesic pyrophosphate; Mr Anderson, analyst to the Christmas Island Phosphate Company, gave me his kind help in this.

20·2 grams lost 7·8 grams on desiccation at 100° or 38·7 % moisture, after treatment as above 0·3674 g. of $Mg_2P_2O_7$ were obtained, which gives a percentage of 1·15 of P_2O_5 in the moist fish freed from bones or 1·88 % when desiccated. (Another sample of 55 grams gave a moisture percentage of 40·0.)

Though this is but a single analysis, I feel justified in recording it, as Katz has obtained comparable figures in his estimations of total phosphorus in various meats. Thus he gives

Dry pike flesh with fat	2·35 %	reckoned as	P_2O_5
Dry eel flesh „ „	1·09 %	„	„
Dry pork with fat	1·79 %	„	„
Dry beef „ „	1·60 %	„	„

These figures are naturally much higher than those obtained by the incineration method; the following of which may be quoted as showing considerable differences in the phosphorus contents of the various substances.

Thus the ash method gives about 0·8 % in wheat, 0·8—0·9 % in peas and beans against 0·19 % in rice, all reckoned as P_2O_5; also beef yields 0·285 %; mutton 0·425 %, and pork 0·16 % (it may be noted that the pork eaten by the Chinese is mainly fat). Probably no observer who has had to deal with beriberi cases will deny the good influence of adding peas, beans, milk, eggs and the like to the diet of patients.

Apart from the general deficiency of metabolism in beriberi, to which I have recently recalled attention (*Brit. Med. Journ.* Vol. I. 1904), it should be noted that the appetite in the disease may be very bad. Visits to beriberic wards at mealtimes always showed many unemptied plates. Mr Galloway of the Pudu gaol pointed out to me that as soon as matters mended in the epidemic of 1902 there was a marked diminution in the amount of waste rice which is disposed of day by day.

III. Experiments on Animals.

The following are notes of a number of experiments which I made in the hope of inducing beriberi in animals. Monkeys of the genus Macacus[1], small coconut monkeys or "brok" in Malay (*M. nemestrinus*), and the longtailed monkeys (*Macacus cynomolgus* and another species) or "kĕra" in Malay, were used. A few rabbits and guinea-pigs were also tried. It must be admitted that at the present time we do not know that any of these animals are capable of contracting the human disease, beriberi; several authors have claimed to have reproduced the disease in animals but it appears that the cases claimed are merely of the nature of septic neuritis.

Recently Hamilton Wright has claimed to have induced beriberi in monkeys, but whilst the looseness of his report may be sufficient indication of the looseness of the observations upon which it is founded, it may be well to add a note concerning these monkeys. For instance there is no reason to suppose that his "monkey 27" was anything but a case of neuritis due to septic absorption from an old standing chronic deep ulcerated cut in its neck, which was caused by the wire collar whereby it was chained. Two monkeys from the same batch, from the same place, which were under my observation may be regarded as controls, especially one which became emaciated and weak through an ulcerated cut round the abdomen caused by a retaining band; owing to want of space this monkey had been chained outside a cage; it had been regarded as stock, but was killed at once when my attention was drawn to its condition; its liver was full of abscesses, but as it was not anticipated that there would be any special interest about it no cultures were made; the nerves,

[1] The anthropoid Gibbons would perhaps have been better for such purposes.

however, were prepared with Marchi's method and were found to be in an advanced state of degenerative change: this was most marked in the anterior tibial, but present in other limb nerves as well as in the phrenic. The second monkey had no external lesion, it became rather suddenly ill with marked weakness, the knee-jerks exaggerated and intensified response to blow on muscles. At the post-mortem the apices of both lungs were solid, with the exception of a cavitation of the left; very scanty tubercle bacilli and abundance of staphylococci were found; the nerves examined showed many extensively degenerated fibres. Neither of these monkeys had been exposed to any presumable beriberic influence. Both of these as others in the same batch were found to be infested with a malaria-like parasite in their red blood corpuscles, and the temperatures of five out of the batch were all very irregular, *e.g.* rising to 103°, 104° and the like (the statement in regard to "monkey 27," that its temperature did not exceed 100·5° till July 18th, is perhaps explicable by the desire to please, or by the gratitude of the small responsible native assistant for having escaped conviction on a charge of perjury, though he soon after was imprisoned for two years for theft and forgery[1]).

Owing to these findings the nerves of every animal that came to post-mortem subsequently were examined, but it was only in these two obviously septic and possibly "malarial" cases, and the one noted in text, that degeneration was found.

It is also to be regretted that the suggestion of Drs Gimlette and Travers to expose animals to the atmosphere of the cells of the gaol was not arranged in a more satisfactory manner; thus the cells by direction were allowed to be in a nauseatingly filthy condition, well calculated to make any free living beast fresh from the jungle ill. But it would occupy too much space were I to go further into the statements recorded in this report.

As will be seen below only one monkey gave a suggestion of possibly artificially induced beriberi, and attempts to repeat the experiment proved without satisfactory result; although this monkey did not apparently suffer from any ordinary septic influence, in the light of the two controls mentioned on p. 128 I am not disposed to assert positively that it had a beriberic neuritis.

(1) *Feeding Experiments (Dried Fish and Rice) on Monkeys.*

First, having been impressed by the apparent connexion between dried fish and beriberi, some monkeys were given portions of dried fish; one control received nothing but boiled rice, and one monkey received fish and rice only, the remainder had fruit, &c. as well. None of these showed any really suspicious symptoms. The rice-and-fish monkey after three months on fish and three months on rice and fish died about a month after resumption of fruit diet; nothing was found to account for death, but its nerves were healthy.

[1] See *Malay Mail*, June 13, 1903.

The fish used included a sample which had been implicated on Christmas Island, as well as a number of samples which were bought in the open market and kept aerobically, anaerobically, or infected with the above-mentioned suspected fish.

So far as they go these experiments were negative.

(2) *Do Bed-bugs carry Beriberi?*

Two monkeys were subjected every few days to the bites of a number of bed-bugs which had been captured from time to time about the bed-boards occupied by beriberic patients at the Lunatic Asylum and General Hospital (gaol ward) at Kuala Lumpur.

These experiments proved negative. The bugs refused to bite a rabbit.

It may be noted that it was possible that the bed-bug might be a carrier of beriberi from some points of view; but whilst these insects are common in Tamil home quarters, during the gaol epidemic I failed to find any about the cells on two or three occasions. I did not succeed in finding any in the coolie houses on Christmas Island. In a house at Tras (Pahang) (*v.* p. 137) where a number of men had been affected with beriberi (*vide infra*) bugs were found abundantly in an old piece of sacking; some were collected for experimental application, but all died in transit from the breakage of a formalin bottle.

(3) *Effect of Injection of Serum of Beriberics into Animals.*

Serum. The fresh blood sera of persons dead of beriberi and of persons suffering from the disease were injected hypodermically in quantities of 10 to 30 c.c. into monkeys and in less quantity into a guinea-pig and rabbit; beyond a passing local oedema in the guinea-pig and a superficial necrosis of skin in the rabbit there was no result to record. This negative result agrees with those of other authors.

(4) *Administration of contents of Gastro-intestinal tract of Beriberics by the mouth to Monkeys.*

A series of monkeys was given by the mouth quantities of the gastro-intestinal contents obtained at fresh post-mortem examinations of beriberi cases. About 10 c.c. were swallowed or given by a catheter passed into the stomach. The doses were repeated once. The matters thus tested were the contents of the stomach, duodenum, jejunum, ileum and colon. In no case did any suggestive result accrue. Cultures on

agar plates were made at the same time: the small translucent colonies which were sometimes abundant all proved to be ordinary streptococci. Otherwise there is nothing worthy of record.

(5) *Administration of Dust to Monkeys from "infected" Localities.*

One monkey had its throat rubbed with dust obtained from crevices in a bed-board of a cell in Pudu gaol; two months later it weighed 1114 grams. At this time it unfortunately received an injection of 10 c.c. of beriberic serum[1]. 113 days after the dust application (55 after injection) it was rather ill (weight 940 grms.), and had an erythematous patch in the groin and about the feet. Solution of sulphide of soda was applied but it proved caustic and caused a slight superficial ulceration two days later. The knee-jerks were markedly exaggerated. Two more days later the knee-jerks could hardly be elicited, there was no oedema. Next day it died, viz. 118 days after commencement. At autopsy the liver was very pale and fatty; there was very little fat about the body; the suprarenals were somewhat gelatinous in appearance (a condition I have noted at some post-mortem examinations of men dead of beriberi), and there was a small superficial ulcer in the left groin (from the sulphide). Otherwise the organs were merely anaemic. The limb nerves and the phrenic showed much degeneration by Marchi's method; whether this degeneration was due to beriberi is a question that cannot be answered.

Naturally I instituted other experiments with dust applications and also confined two monkeys in a packing-case with some mats which had been in use by coolies who had died of beriberi, but none of these gave definite results.

(6) *Throat to Throat Infection Experiments on Monkeys.*

Since the administration of the contents of the stomach and bowel had been negative, trial of application of mucus from the throat of a patient directly to the throat of a monkey was made.

A monkey was first swabbed from the throat of the above-mentioned monkey [Section III (5)]. Two months later its throat was swabbed with mucous matter from the throat of a beriberic case; two and a half months later it had somewhat exaggerated knee-jerks and the direct muscle reflex was well marked; but it was not killed for nerve examination, so that the result was not satisfactory.

The other monkeys did not show any symptoms.

[1] There was a lack of animals.

IV. Observations on Man.

(1) *Clinical observation of the Throats of Beriberic Patients.*

Clinical observation of the throats in two places so distant as Gopeng (Perak) and Christmas Island (more than 1000 miles apart) as well as in Kuala Lumpur seemed to indicate that in early cases and those which had recently come to hospital, there was a marked faucial redness. This condition was not associated with tenderness or swelling of the lymphatic glands.

Sterilised cottonwool swabs mounted on wires were smeared on the throats of a number of patients and then on the surface of agar medium in Petri dishes. After 24 hours, but better after 48 hours, remarkable numbers of small low translucent grayish colonies appeared on the plates; after 96 hours they measured only about 1 mm. in diameter. Under a low power of the microscope they showed a well-marked distinguishing feature in that there were tiny loops of projecting organisms around the periphery. Some plates were crowded with these "*small looped*" colonies and this almost or quite to the exclusion of other kinds of colonies. Morphologically, they assumed a somewhat streptococcal appearance in short chains, but there was a great tendency to the formation of involution forms, of a swollen irregular or rod-like character; no motility could be detected. The organisms retained the stain after treatment by the Gram-Weigert method and showed some capsular material. A considerable number of plate cultivations were made from gaol dust and dust obtained from suspected sleeping mats, but none of these revealed similar colonies. The same may be said of the cultures which were made from the intestinal tracts[1] of beriberic corpses.

It would appear that some constituent of the mucous material in the mouth is necessary for the development of the organisms described above (cp. *Bacillus influenzae*), for subcultures all failed to grow on being transplanted on to fresh agar or into broth. Original cultures which were brought back all died out on voyage.

The following are notes of the last 5 cases examined:

(1) Lim Chin gives two months' history; can just walk; knee-jerks absent; direct muscle-reflex markedly increased with fibrillary wheal. Fauces and pharynx very red.

[1] Dr Mott, I think, was the first to observe the reddened condition of the duodenum in acute cases. The redness may extend a considerable distance along the small gut; it would appear to be a secondary phenomenon.

Throat examination: *microscopically*, chiefly micrococci with some Gram-Weigert staining bacilli.

Plates from the throat:

24 hrs. Very abundant small translucent colonies.

48 hrs. Very marked abundance of small "looped" colonies.

96 hrs. On one plate only five, on second only one other than small translucent "looped" colonies.

(2) Leong Suk gives history of ten days; knee-jerks absent; direct muscle-reflex markedly increased; can hardly walk. Throat reddened.

Plates from the throat:

24 hrs. Very abundant small translucent colonies with looped edges, with a few large white opaque colonies.

96 hrs. One streak consists entirely of numbers of small looped colonies.

(3) Theng Yoon gives history of 4 months and has been 13 years away from China. Knee-jerks absent; direct muscle-reflex in leg rather lively, in arm markedly exaggerated; slight oedema of shins.

Throat: *microscopically*, cocci, diplococci and a few rods.

Plates, 48 hrs, very abundant growth, nearly all small looped colonies.

(4) Vong Heng gives history of $2\frac{1}{2}$ months' illness and has been 7 months away from China. Calves very flabby and tender; knee-jerks absent; no oedema; cannot walk. Throat not reddened. *Microscopically* few cocci and few bacilli.

Plates, 48 hrs, not many colonies, but several of them are of the small looped variety.

(5) Lim Chee gives history of 2 months' illness and left China 22 years ago. Can walk in the "typical" beriberic manner; knee-jerks absent; no oedema. Throat slightly red; *microscopically* many cocci with some club-shaped bacilli.

Plates, 48 hrs, abundant colonies, nearly all of the small looped variety.

The above are samples out of the examinations which were made. It was obviously important to see whether this type of organism was a common inhabitant of the throat, or whether it was solely to be found in connexion with beriberi. Unfortunately before proper control observations had been established the local heads of the Government became so discourteous that it was not possible to consent to remain working in their sphere, where one had to be more or less in the position of a guest of Government. Unluckily, too, there did not appear to be a favourable locality for prosecuting the matter at the time, so that I reluctantly returned home with this portion of the investigation uncompleted.

(2) *Notes on some clinical aspects of the disease.*

Knee-jerks. Too little notice is I think taken in most books of the preliminary exaggeration of the knee-jerk, which so commonly precedes the loss thereof. Whether the knee-jerk is ever lost without a previous

stage of increase I am not able to say, but observations on a number of patients showed that, given the increase, it may remain and never disappear before the patient's apparent recovery, or it may slowly decrease to absolute loss, or it may disappear rapidly in a few days, giving way to complete absence. Recovery of the knee-jerk after loss seems to be a very slow process, but that it is eventually regained there can be no doubt if the history of having had the disease in several prisoners and others is to be believed. Increased response to a blow on the muscles seems to obtain generally after the diminution of knee-jerk has occurred.

Tenderness of calf muscles. It appears that rather too much prominence has been laid upon this symptom, so far as my experience goes. For though I have squeezed the calves of many hundred beriberics, I cannot remember having caused flinching or wincing in more than half-a-dozen cases. Nor taking cases in all stages of the disease do they often complain of pain having been caused.

Variations in prominence of symptoms. Several authors have already insisted upon the variable prominence of certain symptoms in different epidemics, and this is a matter which my own observation confirms. But I do not think with some of these writers that this is to be regarded as indicative of a different breed of beriberic noxa. Thus the last little epidemic that I saw on Christmas Island was characterized by very marked affection of the heart, with very slight or absent oedema and much exaggeration of knee-jerks; still at the same time there were a few cases of severe "wet" beriberi. Indeed wherever there were sufficient cases, one could see examples of all the different varieties as a fairly complete clinical picture of a single disease.

Hoarseness of the voice is a symptom which seems to be more frequent in some epidemics than in others.

At my first visit to Christmas Island there was a great deal of stomatitis (swollen sore gums), but judging from the complete absence of this at a later period and in other places I hardly think that it can have any causal connexion with beriberi.

V. Remarks on the Epidemiology of Beriberi.

It would seem from the literature of beriberi that none of the theories which have been advanced will explain satisfactorily all the accounts which have been given. Often these accounts are wanting in sufficient completeness for pronouncing an opinion. An instance of the

unsatisfactory nature of the literature may be cited in the oft-quoted outbreak in and about New Caledonia, the account of which has led to its being used to support the dried fish theory, the rice theory, the emanation theory, and also the infective theory. It is clear that if one of these theories be true the others must be essentially wrong.

During the eighteen months that I spent in the Malayan region I made a considerable number of visits and inspections with the specific object of obtaining information concerning beriberi. I feel, on looking through the notes made at the time, that the greater the amount of facts that I was able to discover, the less could the possibility of some direct or indirect contact be eliminated. Naturally much has to be discounted from the accounts given by natives however well meaning; but occasionally I met, almost accidentally, with information from independent sources which tended to show that possibility of contact had occurred through the introduction of persons suffering from beriberi.

Scheube considers that the contagiousness of beriberi can be excluded, and I have met with cases where contact was present and the disease was not acquired (see p. 119, case of Eurasian and wife, and p. 123, case, living with cooks on Christmas Island). But on any theory such cases must be explained by the mysterious variations in susceptibility towards the noxa of beriberi, whatever that may prove to be. It seems probable that the food, or absence of a sufficiency of certain requisites in the food, bears some intimate relationship with the proneness to acquire the disease.

Further, on the assumption that beriberi is due to an infection of the throat region, the preexisting bacterial flora will have an effect upon the variability of and poison production by the effective microorganism. In the growth of the diphtheria bacillus we know that the presence of certain sugars in the medium, while leading to the production of acid, may more or less completely inhibit the production of toxin. Just as it is possible that the presence of other organisms capable of producing acid from the pabula in the mouth may alter the nature of the attack, so in beriberi it may be that some more or less fortuitous condition of flora or constitution of food remnants may have their due effect. There is also the question of an acquisition of immunity in beriberi (*e.g.* by slight attacks), but whilst there seem to be indications that such may exist actually, it will be better to defer consideration of this point until the presence of specific "antibodies" can be demonstrated.

(1) *Outbreaks in Isolated Places.*

That the cause of beriberi is to be found in and about the person or the personal apparel and personal furniture is suggested by the way in which the disease has disappeared from regions with the removal of the persons who brought it. Just as in Fiji the disease ceased when the Japanese left, so on Christmas Island a party of Klings who lived together in certain houses (one a new house) far removed from the Chinese quarters were severely affected during my stay there; but when they and their small amount of baggage left the island, there was no further trouble in that house during the next 12 months.

That the noxa of beriberi is carried in close relation with the person is also suggested by an outbreak which occurred amongst a party of Chinese carpenters whom I saw and examined on their return voyage to Singapore. They had been for a few months on the previously uninhabited Direction Island of the Cocos-Keeling group.

Out of the 12 men 2 died (one was seen to die, and undoubtedly did so of beriberi), 3 had severe beriberi, 2 had slight beriberi, 3 seemed quite normal, 2 were not examined.

It seems clear that the beriberi noxa must have been taken down to the island somehow in some vehicle or other. The vehicles which are apparently possible are (1) the one or more of the persons themselves, (2) some part of the scanty baggage with which this class travel (sleeping mat, blanket, a change or so of clothes and box of tools), (3) the food taken. Though I believe that food or some constituent of food may have a distinct influence upon the susceptibility towards the disease, since I have not investigated any outbreak in which every possible factor save food can be definitely excluded, I am inclined to disbelieve that the food can have done the mischief. (4) The men may have acquired the disease from the sailors or the vessel upon which they reached the island (the voyage would have been of about 5 days' duration); upon this I am unable to give any opinion as I have no knowledge of the vessel. Unfortunately I have no reliable date to fix the first commencement of the outbreak, but since the men were on the island from Sept. 1901 to Jan. 6th, 1902, it is possible that the disease was spread on the island. Perhaps one of the men who was found to be normal on the return voyage was the refractory or immunised carrier of the noxa.

The following case of the occurrence of beriberi may be mentioned here, since personal contact appears to have shown some influence.

A party of picked Malays (South Borneo) was brought over to work in the jungle in Pahang. The men suffered severely from beriberi soon afterwards. Their employer, Mr Ponsford, was certain that they had acquired the disease from eating dried fish away in the jungle. For a short while they were accommodated in a house in the village of Tras, which had previously been occupied by Bengali bakers. With one exception they remained but a short time in this house. The exception was a man who injured his foot by a falling log, he was laid up and remained in the house; of the whole party *he was the most severely affected with beriberi.* There could be no doubt about the symptoms in any of the men. It is, then, very improbable that the other men acquired the disease in the jungle, in a manner independent of their injured mate. It may be concluded that all caught the disease in the house. The presence of abundant bed-bugs has been mentioned on p. 130. It happened that I met a Malay "Haji" several miles away through the jungle, who was suffering from severe beriberi, accompanied by dyspnoeal attacks. He gave a most circumstantial account of having been in the habit of sleeping with the South Borneo men in the house. He obtained his meals at the house of the local "Datoh" (district head Malay). He also said that another man suffering with the same malady had been of the company. There was no reason to suppose that he was not telling the truth, his statement was voluntary, and he had no reason to fear. I carefully abstained from any leading questions. The house was an old wooden shanty on the ground, dark inside, but with a fair amount of ventilation; the weather was wet but the interior seemed to be dry.

This case is illustrative of some others, which might be quoted as instances where though it might be supposed that the disease had been derived from the infected condition of a house, yet, even so, the possibility of actual contact or close neighbourhood of persons suffering from beriberi could be entirely excluded.

(2) *Alleged House and Ship Infection.*

The question of infection of houses and ships has been given some prominence in the literature, thus Rees speaks of certain ships in a fleet as "beriberi ships," and he draws attention to the continuance of the disease on these ships. If, however, his tabulated statement be examined, it will be seen that the so-called infection of the ship was not continuous through the history given, but that on a given ship the disease disappeared to reappear again some years later. To such interruptions no attention was drawn and no explanation was offered, but it is not inconceivable that the disease was not directly acquired from the ship itself, but from some item of the crew; and then no difficulty is to be found in understanding the circumstances. It must be remembered that it is not always or only those suffering from pronounced infective disease of any sort that are necessarily the most

dangerous spreaders of the disease. Not infrequently it may happen that the unrecognised spreader does incalculably more harm. But in beriberi, as yet, there is no established criterion of recognition such as we have in the case of the diphtheria bacillus.

Another point to which reference may be made is the poor result which is obtained by house disinfection; thus the curves given by Pekelharing and Winkler do not give a noted effect of sublimate disinfection. This was very strikingly the case on Christmas Island, for during a period of about a year the houses (*i.e.* floor and bed platform) were swabbed out once a week with 1 : 4000 corrosive sublimate, later this was done once a fortnight, since the men complained of the dampness, which together with personal observation of the process shows that disinfection was fairly thoroughly carried out. Now this disinfection was only in force for the houses occupied by the coolies; the precisely similar houses occupied by the artizans *were not interfered with.* It has already been mentioned that the artizans practically escaped the disease whilst it still continued to ravage the coolies. It must be clear that if the causative agent lurks in the sleeping mats, clothes &c., or actually about the persons of the coolies, the house disinfection is not calculated to be of much service.

(3) *Alleged Importance of Removal of Patients from a Locality.*

Many writers tend to lay great stress upon the importance of removing patients from the locality in which they were taken ill. Whilst I do not desire to combat the belief that a change of air and still more of food is beneficial, yet it seems to me that removal does not benefit the patient suffering from beriberi to a greater extent than the patient suffering from many other diseases. It need hardly be asserted that with malaria, and almost any other infective as well as some noninfective diseases, removal does good which can hardly be obtained by any other means. Beriberic patients can and do get well locally; and though they stay in or about a focus of the disease they do not necessarily suffer thereby to the extent which might be imagined. In my own case, if indeed the condition, which did not consist entirely of subjective symptoms, was really of the nature of beriberi, I may mention that I continued to occupy the same rooms, and very damp, nay moist were they, where if some miasm inspired with each breath had been the cause of the complaint I should hardly have survived to tell the tale.

Again, the cooly who is discharged from hospital is not often really

cured but rather merely relieved, yet for instance on Christmas Island though he returns to his old quarters, to his old work and companions, yet he does not as a rule become progressively worse, though in cases he may again seek hospital care.

Dr Travers called my attention to an interesting point in that, during the extensive outbreak in 1902 at the Pudu gaol, owing to the large number affected, all could not be given accommodation at the hospital within the gaol compound, which indeed was much overcrowded with patients at times. A number of the patients were sent more than a mile away to the district hospital. A considerable amount of selection had to be made of the cases to which hospital accommodation could be given, so that it was only the more serious cases that actually were admitted or admitted and transferred.

As shown by the deathrate the patients did much better within the precincts of the gaol than they did away at the district hospital.

It is striking too, in the case of some of the outlying district hospitals, in the Malay States, how different the beriberi deathrates may be. Certain of these hospitals are in charge of native dressers; but so far as I have seen, although there may be some erroneous diagnoses, and some deaths may be due to intercurrent circumstances, yet these seem to be comparatively few. Particularly worth mentioning is the case of Jugra hospital, in which, as Dr Watson pointed out to me, the beriberic death-rate was very high, whilst the place itself hardly contributes to the beriberi cases; these are brought from a place many miles away.

Table to show difference of Deathrate from Beriberi in various Hospitals in Selangor State.

(The figures are for the first half year of 1902.)

	Cases	Deaths	Death rate %
Kajang	103	nil	0
Kuala Kubu	69	19	28·8
Serendah	113	6	5·3
Kuala Langat (Jugra)	10	6	60·0
Kuala Selangoi	nil	nil	—

Note. For these figures I am indebted to Dr Travers. We paid a special visit to Kajang to look up records and examine cases for control of current diagnoses.

Table to show high mortality from Beriberi in Kuala Langat (Jugra) Hospital.

(The figures are for 1902 and are analysed to show length of stay in hospital.)

No.	Patient's statement of duration of disease on admission	Where acquired	Date of admission	Date of death	No. of days in Hospital
1	45 days	Sepang	12.1.02	21.1.02	9
2	20 ,,	,,	16.1.02	17.1.02	1
3	15 ,,	,,	22.1.02	14.2.02	23
4	10 months	,,	22.1.02	1.3.02	38
5	25 days	,,	28.1.02	18.2.02	21
6	10 ,,	,,	28.1.02	—	120*
7	30 ,,	,,	11.2.02	—	90*
8	30 ,,	,,	26.2.02	—	13
9	30 ,,	Jugra	4.3.02	—	7
10	3 months	Sepang	20.3.02	3.4.02	14
11	16 days	,,	8.10.02	1.11.02	24
12	2 months	,,	14.10.02	24.11.02	31
13	1 month	,,	12.11.02	22.11.02	10
14	3 months	,,	10.12.02	29.12.02	19

Total cases admitted ... 14
Total deaths 10
Mortality ... ... 71 %.

* Approximately.

Note. I am indebted to Dr Watson for these figures. He points out that all the cases except one were removed about 40 miles from the place where the disease was contracted. It will be observed that many of the cases were not fatal very soon after admission. I made other similar analyses of records but the one given will suffice to illustrate the point.

(4) *Length of Exposure of Persons before Acquisition of Beriberi.*

Some writers have insisted that there must be a prolonged exposure (*e.g.* years) before the disease is contracted. Observations on the incubation period are difficult inasmuch as the time of real onset of the disease is so ill-marked[1].

On Christmas Island I examined a number of beriberics who certainly had not been more than three months on the island. The men had passed a routine medical examination in Singapore which was designed to detect and exclude beriberics before they are shipped to the island. A batch of men who had been 16 days on the island was examined by me on the 9th of December, and found healthy except two with slightly

[1] The same may be said of the conclusion of the disease with recovery, so that such terms as "beriberic residual paralysis" or "post-beriberic paralysis" are at present without meaning.

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increased knee-jerk. On the 22nd of January one of these men died of beriberi. On February 4th, of nine of these men who were gathered together for examination, only two could be passed as free from suspicion of beriberi. Supposing the fatal case was not incubating the disease on his arrival he passed from inception through incubation to the fatal ending in less than 70 days. These men were not "Sinkhehs" (see p. 121).

The following figures show the length of residence of one hundred consecutive fatal cases of beriberi on the island. I am indebted for these figures to the District Officer and to Dr Giddy.

Length of residence	No. of deaths	
2 months	11	
3 ,, ...	21	
4 ,, ...	6	
5 ,, ...	12	
6 ,, ...	7	Total up to 6 months, 57
7 ,, ...	4	Total 6—12 months, 21
8 ,, ...	2	
9 ,, ...	3	
10 ,, ...	2	
11 ,, ...	8	
12 ,, ...	2	
1—1½ year	20	
1½—2 years	1	
over 2 ,, ...	1	

These figures tend to show that the greatest beriberi mortality occurs during the first 6 months of residence. It might be expected however that the numbers for the later months should be larger by virtue of invalids who had been sent back to Singapore and who might otherwise have added to the deathroll. But the contract period of time is twelve months, and comparatively few are thus disposed of, so far as I was able to learn, before their contracts mature.

The shortest period of residence on Christmas Island with a fatal termination through uncomplicated beriberi that I met with was in a cooly in Government employ (who had not been passed as medically fit before coming and may have been already suffering when he arrived). Signs of the disease were already well marked on the 16th day and he died on the 38th day after arrival.

(5) *Seasonal Variations in Prevalence.*

The places which I have visited, all within the equatorial zone, do not appear to show that there is any definite seasonal prevalence of

beriberi in this fairly uniform warm, damp climate. Dr Ellis has shown by his curves that the number of cases may reach its maximum in almost any month in the lunatic asylum at Singapore. (Compare table of admissions to Pudu gaol on p. 117.) On Christmas Island it had been supposed that the disease prevailed in the wetter months, but a very distinct outbreak occurred within what were the driest consecutive three months that had been recorded. In the converse sense, at the end of another small epidemic, the rainfall increased as beriberi diminished.

One fact which probably has no meteorological significance owing to the distances of the several places, is that in and about January, 1903, there was an almost simultaneous "disappearance" of beriberi in the Pudu gaol at Kuala Lumpur, in the gaol and lunatic asylum at Singapore, and on Christmas Island. It is hardly possible to say whether this could be more than a mere coincidence.

VI. Current theories of the Epidemiology of Beriberi.

(1) Food.

i. *Physiological.*

Diet as such, or *physiological or specific starvation*, has already been dealt with sufficiently in the foregoing pages. This aspect of diet has not always been clearly separated from the possible effects of unsound food.

ii. *Unsound Food.*

(*a*) *Exclusion of all articles except Rice and Dried Fish.*

Unsound food considered as a cause of beriberi has had many supporters. Looking at the racial distribution of the disease in the regions visited, it may justly be argued that the kind of food which forms the vehicle of beriberi must be one common to all the affected races. This practically excludes all foods with the exception of dried fish and rice. The Malays do not eat pork, though the Chinese do so. The Chinese do not eat curry. The other races do not eat Chinese sauces or preserved vegetables. Fresh fish, fresh vegetables and fruit, can likewise be excluded, because the Chinese coolies on Christmas Island did not obtain any of these articles except on quite isolated occasions.

(*b*) *Rice.*

The rice is practically all imported either from Siam or Burmah; it is milled without the preliminary steaming process already mentioned (see p. 120, Tamils). So far as I was able to learn the rice is distributed and consumed almost as fast as it is imported, so that it is not stored long locally. It is forwarded by merchants in Pinang and Singapore. Many enquiries were made by me to ascertain whether any additions are made to enhance its keeping qualities. Mr Cowan, Protector of Chinese, Perak, kindly gave me much help in this connexion. It appears that lime is often added in the husking process, partly to help the decortication, and partly to whiten the grain. We could not get any evidence that arsenic is purposefully added, and the evidence of the report on the samples of dust from the rice which I submitted to the Royal Commission on Arsenical Poisoning, show that it can hardly be supposed that arsenic had been added, inasmuch as the findings were extremely minute. Another point is that many of the bad samples of rice which I have inspected were full of weevils, so that even if arsenic had been added with intention, not enough had been put to prevent the development of these destructive agents.

When the distribution of the disease throughout the world is included, rice has also to be put on one side; thus in the outbreak at Richmond Asylum, Dublin, as on many Norwegian ships, it is stated that no rice had been consumed. It would be necessary therefore to bring such outbreaks into line with the rice theory either by supposing that it was not the same disease, or else that other cereals may be similarly infected with the causative agent; that is to say we reach the region of pure speculation without direct facts in support.

Two points which impressed me with regard to rice were that on *Christmas Island* all the rice-eaters obtained rice of the same quality from the same store (with a temporary exception of some men in Government employ). Yet, as has already been stated, it was the cooly who suffered. During one of my visits to the island, there was a severe outbreak of the disease amongst a party of Tamils; this was not coincident with any recrudescence amongst the Chinese coolies who lived about a mile away. These Tamils ate in company with the Malays, the accessory foods were dealt with by each but *the rice was taken actually out of the same pot of cooked rice.* Why then should not the Malays also have suffered if the rice were at fault? Again, in the *Pudu gaol*, patients in the gaol hospital were recovering, whilst about

the cells or work-places their mates were being invalided day by day. *They ate of the same rice, which was all cooked together.*

Whilst no doubt mouldy rice has often been consumed in beriberi epidemics, this does not appear to be a constant circumstance. Since some writers advocate this idea, and compare beriberi with pellagra, ergotism, lathyrism, &c., it may be worth while pointing out that these diseases are accompanied by severe central as well as peripheral changes in the nervous system; whilst such are absent in beriberi.

(*c*) *Dried Fish.*

Dried fish of one sort or another is on the whole a more widely spread foodstuff than rice, and it appears to fit in as a food origin of beriberi in a more worldwide manner than rice. It is a staple of diet throughout the East, it is eaten on the Amazon, on Norwegian ships, &c. &c. Curiously enough the Dublin outbreak was preceded by the introduction of dried ling as a novelty on the diet sheet, but it is stated that those who suffered particularly did not eat any of it, so also in Pudu gaol many short-sentenced prisoners were affected yet dried fish is not in their fare.

Up in the hills in Pahang, I found that the Chinese, who were comparatively free from beriberi, were eating expensive tinned fish, as they said that the ordinary dried fish went bad. The Malays, who ate ordinary dried fish ("ikan kring") suffered much more from beriberi, so I was told, and certainly I saw more cases of beriberi amongst them than amongst the Chinese. I may add that I spent a week walking about the jungle from one working to another; men we met were examined and catechised on the spot; others were examined in their houses, or huts which were mere frames enclosed with atap palm thatch.

A good deal of the fish is preserved in fishing villages along the coast of the peninsula, much is also imported. As seen in some fishing villages the process of preserving merely consists in sun-drying, the larger fishes having been split open, the smaller not; they may also be parboiled by dousing in a large pot of boiling water before they are exposed on bamboo frames to the sun. When dry they are either done up in bundles or packed in tubs with coarse salt. On the whole the dried varieties are less sodden and decomposed than the salted ones.

The proximity of stores of dried fish is apparent by the smell. The consumer and small restaurant keeper often exposes his supply of fish to the sun for further drying, and when it is thus well dried it hardly

has any smell. Fish as obtained wholesale from the merchant, however, is often mushy and offensive. One kind of bacterial growth occasionally gives trouble and is mentioned by Vorderman, it consists in the formation of a brickred film on the surface of the fish. Dr M. J. Wright, State Surgeon, Perak, kindly obtained some of this reddened fish for me. The chromogenic bacteria found in it were a pink-red Sarcina and a brilliant yellow bacillus. Another sort of fish, stated to come from China (somewhat like our red mullet in size and colour), was found to be affected with a yellow bacillus which apparently tends to grow or produce its colour along the nerves, thus the lateral line stands out as a lemon-yellow streak. The liability of dried fish to certain bacterial infections was of interest from the possible origin of beriberi from this cause. It may be noted that the merchant from whom the red infected fish was obtained had given up trading in it on account of the way in which it went bad in his store (which included a privy). It has already been pointed out, however, that there are reasons for exculpating this article in beriberi.

(2) ARSENIC.

Associated with food theories is the suggestion that *arsenic* is the cause of beriberi. Manson has pointed out that clinically beriberi and chronic arsenical poisoning appear to be distinct from one another. It has also been suggested that some other mineral substance or inorganic poison may be the cause, but Manson has, I think, rightly insisted that the evidence of power of multiplication of the cause of beriberi is strong and controverts the probability of any inorganic source. Ross, who has advocated an arsenical origin to beriberi, has recorded the finding of arsenic in the hair of beriberic patients; but the results of the analyses he gives are really opposed to his hypothesis in as much as only traces of the metal were discovered.

I made some attempt to determine to what extent arsenic is used by the Chinese. Mr Cowan very kindly gave me much help in gathering information on the question. It appears that "white arsenic" can be bought for about 20 cents a kati (say 5 pence for 21 oz.), and that it is used for the destruction of the white ant. In a list of Chinese drugs "arsenic," "white arsenic," and "red arsenic" were included; we made enquiries at two druggist's shops, but we could not get any admission that any of these were given as medicine. So far as we were able to determine it would only be as an impurity of other drugs that arsenic would be taken in medicine.

I may here interpolate a case of "burning palms and soles," the only one I met with in a Chinaman. Though he was somewhat like a beriberic in other symptoms it seemed possible that he was an arsenical, or a combined arsenical and beriberic case. Samples of his hair were sent to the Royal Commission, but they were not fortunate enough to reach the analyst. He gave a very distinct history of having been for six months under a Chinese doctor whose medicines caused a tendency to diarrhoea and intense griping; the burning in the palms and soles began three months after this treatment began. It may be added that several other cases of "*burning feet*," all in Indians, were seen, but it was not worth sending samples of their hair for they were all having arsenic administered therapeutically; the results did not seem to lead to cures or improvement. Enquiries as to cosmetic preparations failed to elicit any information as to the addition of arsenic.

Out of many hundred beriberics that passed through my hands I only once saw a case of *herpes*; this I think may fairly be put down as incidental.

I went to some trouble to determine whether *pigmentation* occurs in beriberi. The Chinamen vary very much in depth of colour—a certain small proportion are distinctly and markedly bronzed, and some of these individuals may have an arsenical coloration (see analysis of tow chang, p. 147). Personally I believe that the beriberic has a rather greater tendency to a deepening of colour on exposed parts, especially the legs; this may be due to the vascular changes which give rise to the oedema.

Enquiry of several "towkays" led to the presumption that arsenic is not intentionally added to rice for preservative or other purposes; quicklime is sometimes used in husking (see above, p. 143).

In order to see whether arsenic were added to rice, quantities of rice which was being consumed in Pudu gaol were sifted through fine wire-gauze sieves; the resulting dust was tested by the analyst of the Royal Commission (see Report, Appendix 31, p. 341).

Sample A was taken during a lull in the beriberi, July 28th, 1902. Amount of rice sifted = 200 lb., or about $\frac{1}{3}$ of a day's supply: total dust = 46·9 grms.; amount of arsenious acid = 3·2 parts per million of dust or $\frac{1}{46}$ grain per pound of the dust.

Sample B similar, but taken during height of fresh outburst of beriberi: total dust = 65·3 grms.; amount of arsenious acid = 2·0 per million or $\frac{1}{70}$ grain per lb. of the dust.

Sample C. Dust (Jan. 8, 1903) from whole week's supply of rice, *i.e.* about 4500 lb.; arsenious acid found = 5·0 per million[1].

Sample D similar to *C*, collected Jan. 19th; arsenious acid found = 5·8 per million[1].

[1] Total weight of dust not recorded.

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In the appendix of the Report of the Royal Commission it will be seen that several samples of whole rice were submitted to analysis with the result that traces of arsenic were found in some samples and none at all in others.

In regard to fish (Norwegian cod and "Bombay duck") small quantities of arsenic were found by the Commission: since these did not exceed $\frac{1}{80}$ grain per lb., and if the eastern native's diet may be compared only about $\frac{1}{4}$ lb. of fish is taken a day, on this estimate there would not be a dangerous quantity of arsenic in his diet.

I made a few examinations of Chinese and Malay dried and salted fishes and of the salt with which the latter were covered and also of one sample of the phosphate rock, which is worked on Christmas Island. Owing to the acid having become arsenicated from the bottles and the Marsh apparatus which was sent out having been defective and also broken on the passage, measured quantities of hydrochloric acid were boiled for some hours with successive pieces of pure copper foil until little or no visible stain was caused; the matters to be tested were then introduced. Finally a drop of diluted Fowler's solution was added to see that a marked stain was capable of being produced and compared with the test piece of copper. Seeing that the results obtained from the objects mentioned were all negative, I think that they may be fairly considered to show that arsenic was not present in dangerous amounts. Some hair and nails were also examined, but the results were not sufficiently reassuring for inclusion here.

The Royal Commission kindly undertook the analysis of a number of hair and nail samples which were sent. In Appendix 31, page 344,

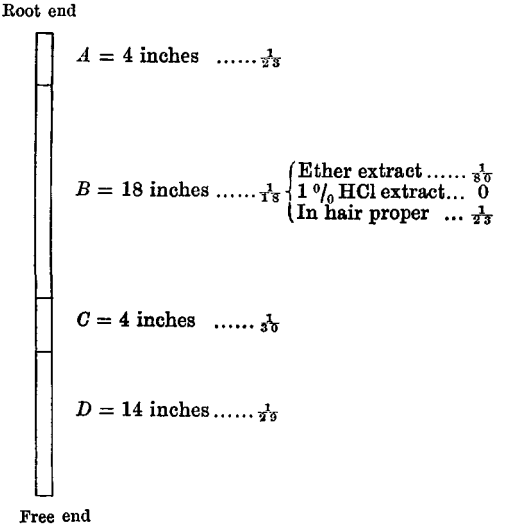

Diagram of "tow chang" to show distribution of Arsenic.
As_4O_6 in grains per lb. (found by Mr McGowan, Analyst to the Royal Commission).

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of the Report of the Royal Commission on Arsenical Poisoning will be found the analytical record of two "tow changs" or pigtails from Chinamen recently dead of quite recent acute beriberi; these were examined in sections, of which the one contained $\frac{1}{15}$ and $\frac{1}{16}$ grain arsenious acid per lb. and the other contained up to an $\frac{1}{18}$ grain per lb. (see diagram). The arsenic, though in notable quantity, from being distributed all along the hair shows that the patients must have been in the habit of taking arsenic somehow for a long period. If an allowance of $\frac{1}{4}$ inch growth per week be made, *i.e.* very much in excess of the probable real rate of growth, the end of one "tow chang," which was about 26 inches long, would have been some two years old. The quantities stand out in contrast to Ross's own cases and also some other beriberi cases which were tested for the Commission. We can say then that though it is clear that arsenic must be partaken of by many beriberics (as also by normal individuals), the analyses which have been made are against the supposition that there is a connexion between these two clinically more or less distinct conditions.

In the same report is included an observation of Dr McClosky of Kuala Lumpur upon the therapeutical effect of arsenic given to beriberi patients. In his wards at the District Hospital, cases without selection of any kind were put alternately on doses of Liquor Hydrargyri Perchloridi, Liquor Arsenicalis and Mistura Chlori (the latter I should mention consisted of Burney Yeo's mixture [see Squire's *Companion to the Brit. Pharm.*], without quinine: this I suggested as worth a trial, and, although not curative, it has been extensively used, since it gives as good results as any other medicine[1]). The Liquor Arsenicalis was

[1] I may add that a small experiment to see whether Chlorine mixture would have a prophylactic effect, was made on the hypothesis that beriberi might be due to some ingested poison which could be destroyed by the oxidising action of the chlorine upon it. One ounce of the mixture was given three times a day to each of a party of 25 prisoners; a similar party of 25 men engaged at the same work (rotan shed) was examined to serve as control. The experiment was continued for 5 fortnights, during which time 14 admissions and 3 re-admissions occurred amongst the chlorine gang against 12 admissions and 4 readmissions to hospital amongst the control gang. Whilst the result so far as protection was concerned proved negative, at the end of the period, judging by physical examination, the men of the chlorine gang appeared to be rather sounder than the controls, but the numbers are too small to prove such a point. I may note here, that it was intended to try the effect of adding phosphate of soda to the rice consumed by the prisoners; this however was not carried out as the disease ceased spontaneously in the gaol before the necessary salt arrived. Grijns records some negative therapeutical experiments on *Polyneuritis gallinarum*. On my advice the same substance was to be tried in treatment of cases as a laxative instead of the usual sulphate of magnesia on Christmas Island, but reports thereon have not reached me. One other therapeutical experiment was tried, viz. to see whether the administration of carbonate of ammonia would increase the urea excretion or otherwise act beneficially; the patient was a fractious old man and the experiment was abortive.

begun with small doses and then pushed up to doses of 10 minims three times a day unless contraindications arose. Whilst it is true that the results were so far from favourable that the designed completion of 50 cases was not carried out, yet a few of the patients who recovered had taken heroic quantities of the acid. This being so, we have some therapeutical evidence against there being any direct connexion between arsenic and beriberi, as well as the clinical and the toxicological indications.

(3) MOSQUITOS.

That mosquitos have nothing to do with the spread of beriberi is probable. Three species of mosquitos were obtained on Christmas Island; these have been identified by Mr Theobald as *Stegomyia scutellaris, Culex fatigans* and *Culex alis* (nov. sp.). The former are solely day biters, they are abundant about the European and Malay quarters and some parts of the jungle but very scanty or absent about the Chinese coolie lines. The second were very scanty and only two specimens were taken, it appeared likely that they had been imported with a quantity of coconuts from the Cocos Islands. A mosquito net was unnecessary during the night, but most people used them, as the *Stegomyia* began biting at dawn.

In the outbreak amongst the Tamils to which reference already has been made there were some other Tamils and also Sikhs in houses interposed between the houses in which the cases occurred. If the disease were mosquito-borne it is difficult to account for the want of spread of the disease amongst these people.

Again, many of the outbreaks on board ship seem to be quite incompatible with a spread by means of the mosquito.

(4) COCKROACHES.

It has been suggested that cockroaches have some power of spreading beriberi. My own observations do not show that they were particularly common about the infected centres, which were visited. I only found a single one when rummaging after dust etc. at the Pudu gaol. Whilst it is possible that they may be able to spread the disease, before saying more it is necessary that the real virus of the disease should be demonstrated in them.

(5) Faecal-borne theory of beriberi.

It has been suggested that beriberi is spread through the faeces. If this were so we might expect that other "faecal-borne" diseases, when introduced, would be found to spread in coincidence with beriberi. Since both on Christmas Island and in Pudu gaol[1] cases of dysentery are introduced from time to time, and since this malady has shown no signs of spreading about amongst the inhabitants, it may be argued that this disease cannot have the same mode of spreading as beriberi.

The only disease, other than beriberi, which tended to spread on Christmas Island, apart from the stomatitis which has been mentioned, was a form of conjunctivitis or ophthalmia. This disease gave a good deal of trouble for a time and afterwards recrudesced again, if my memory serves me correctly.

Owing to the practice of manuring kitchen gardens by the Chinese with crude human sewage it might be thought that the vegetables might be the means of disseminating beriberi. But on Christmas Island though the coolies practically had no fresh vegetables at all, beriberi spread widely amongst them.

(6) Theory of Emanations from specifically infected surroundings.

This theory has latterly been advocated by Sir Patrick Manson, and I regret that the observations which I have made are rather directly opposed to the acceptance of this theory of the mode of spread of beriberi.

A comparison may be made of the gaol at Taiping in Perak and that at Pudu in Selangor; though at one time seriously crippled with beriberi for some years there has been no trouble with the disease in recent times at the former, whilst, as has been pointed out above, the latter has been much ravaged. In general the Taiping climate appears to be rather more prevalently rainy and damp than Pudu. In general, there is not much to say about the buildings themselves, for although the cells are somewhat better at Taiping there is hardly enough difference to account for the circumstance satisfactorily. The differences which I

[1] The general cleanliness and tidy management of this gaol would be against the possibility of spread of a fæcal-borne disease. In some gaols in India dysentery appears to be endemic.

was able to determine in the routines of these two gaols were (1) that clothes at the laundry were boiled or steamed, (2) all dried fish was steamed before being fried, (3) a tahil [1⅓ oz.] of dhal [kind of lentil] was given each day to each man in the rice, (4) blankets which were occasionally washed were used in place of sleeping mats. These methods were in vogue at Taiping but not at Pudu.

I did not witness the process of sweeping out the cells at Taiping, but at Pudu this was done dry, and much unpleasant dust was caused. Nor did I ascertain to what extent the prisoners were transferred from one to another cell comparatively in the two gaols.

The ventilation of the gaol cells at Pudu was certainly defective, but that this in itself was not the sole contributing cause of the beriberi is shown by the fact that the epidemic ceased before the widening of the ventilating slits was begun. During the next four months there were no further cases of beriberi, so it is presumable that the virus either ceased to exist or became latent independently of the ventilating arrangements. When looked at from the point of view of the possibility of some growth occurring in and about the cells, which could give rise to the supposed emanation, the difficulty was to locate where the possible nidus could be. Certainly there did not appear to be any opportunity for active growth of bacteria to take place; the white-washed walls, the simple wooden plank bed, the cemented floor and the ventilating slits were all far too dry for any ordinary bacterial growth to occur with vigour, that is if the conditions under which bacteria behave in the laboratory are of value as a criterion. The dust and "flue" which I collected from the beds, the floors and the ventilating slits gave a large and varied assortment of growths when diluted and plated. Curiously enough amongst the resulting colonies there were singularly few moulds. Many of the plates showed no moulds whatever. It may be considered certain that if such a source is the cause of beriberi, the organism must be different in character to the bacteria with which we are acquainted for a vigorous elaboration of toxic products to have occurred under these conditions. If now we contrast the ill-ventilated gaol cell with the tenements occupied by the sadly stricken coolies on Christmas Island, the site (see Plate X, Figs. 1 and 2) occupied by these houses is a good and airy one, the jungle has been cleared and is quite open to the cliff edge. The ground consists of fissured coral rock incompletely covered by soil, the natural drainage is good and no standing water was ever present near by. An idea of the houses themselves can best be obtained from the photograph Fig. 3. They are 38 in number, arranged

in a street (Fig. 2). The piles on which they are supported are arranged so as to put all floors on a level; thus since there is a slight inclination of the ground, whilst there is just about head room under those at the near end, the floors of the further ones are 9 or more feet above the ground. The floor boards are supposed to be separated by crevices of a quarter of an inch, but as a matter of fact most of them are more widely separated; the floor area measures roughly 35 × 16 feet. There is ample opportunity for ventilation through the floor.

The lowest strake of planks of the side walls is missing all round, so that there is a free air inlet about 9 inches wide between floor and wall.

The shutters are closed at night by the men, but still this does not impede the passage of air about the dwelling to an appreciable extent since the atap thatch of the simple roof is itself pervious to air. Yet more important, the whole of the gable ends are merely curtained by a screen of loose hanging ataps (this is well shown in the photograph Fig. 3). At a nocturnal visit to one of these houses it is difficult to speak of the freshness of the air owing to the usual presence of the heavy reek of opium; still the conditions are such that there cannot be much stagnation and concentration of deadly miasmata. On the whole there was a tendency to dampness within; this was due to some extent to defects in the atap thatch of the roof, and partly, for some days a week, during the wet monsoon, to non-evaporation of the sublimate lotion with which they were drenched week by week at my first visit, and fortnight by fortnight at the second visit.

Outside, the woodwork is tarred or pitched; inside, the walls are whitewashed. To my mind unnecessarily, but at Government desire, the Company have concreted the areas under the houses; but before this I did not detect any marked untidiness. Fig. 4 shows one end of the three hospital buildings which are situated close to the open ocean on a dry rocky site. The horizon on the ocean should show a little above the verandah rail, but it does not appear in the prints. The openness and airiness of the building may be best judged from the figure without entering into a lengthy description.

Now to my mind it is quite inconceivable that enough emanation could be concentrated in the air within these buildings to cause or protract disease. When it is remembered that the cooly does not spend more than 9 hours of the 24 within these buildings (except perhaps on Sundays, when many at any rate go out into the jungle after birds &c.), it becomes still more inconceivable that an emanation can be so potently

evolved that enough can be absorbed in about a third of the daily cycle to produce such grave illness; still more that its absorption should continue within the hospital buildings and lead to a fatal issue.

Thus by comparing the beriberi stricken inmates at the gaol[1] with the free air life of the phosphate cooly, we can but be driven to the conclusion that the matter of ventilation has no great or real determining influence.

VII. Conclusion and Summary.

Finally, I must express my hearty thanks to the many medical men and others who have given me aid and hospitality in my wanderings after the truth about beriberi. I must mention particularly Dr Giddy and Captain Vincent on Christmas Island, Drs Ellis, Leask and Murray Robertson in Singapore, Drs Travers, Gimlette, Watson, McClosky and Mr Galloway in Selangor, and Drs M. J. Wright and Connolly in Perak, and Messrs Mason, Ponsford, Sanderson, and Hembrey in Pahang, without whose aid and kindness I could have seen but little. I must also thank my friend Dr G. S. Buchanan, Secretary of the Royal Commission on Arsenical Poisoning, for forwarding me much information.

It would seem from my observations that certain of the current theories of the causation of beriberi will not account for the conditions which have been encountered by me. The *dietetic or physiological*, the *unsound food*, the *arsenical*, and the *emanation theories* all appear to be insufficiently in accordance with the attendant circumstances to have accounted for the spread of the disease.

It is suggested that certain articles of diet, by virtue especially of containing phosphorised and fatty matters, may tend to ward off the disease when given in sufficient amounts. So far as there was any semblance of a positive result in the observations it is suggestive that beriberi is communicated from person to person more or less directly or through fomites as an actual infection. This infection is not of the nature of a septicaemia (since the internal organs at death prove sterile), but to a surface condition about the upper air passages. From the observation of the throats of a number of patients it is surmised that the redness which is therein seen, especially in early cases, may be intimately connected with the disease. The appearances and disappearances of beriberi and the more or less seasonal waves of prevalence of the disease

[1] The day-time is mostly spent at work under open sheds.

are not unlike those which are seen in the case of the infective diseases which we meet with in this country. The proneness of the newcomer to suffer fits in also with such a view. The difficulty in tracing out the source of infection in a disease like diphtheria is often great, especially since we know that the causative organism may be carried by unsuspected refractory or immune persons or animals.

With considerations such as these it is held by the writer that there is no inherent impossibility in explaining many of the circumstances connected with the spread of beriberi.

Observed facts seem to show that beriberi should rather be considered in the light of a "gang" or "institutional" disease than as a "place" or "house" disease.

EXPLANATION OF PLATE X.

Fig. 1. Shows general view of the cooly lines on Christmas Island from "Loading Point."

The building on the extreme right is the store; next to it is the house occupied by the Chinese contractor's agents; a little below and to the left are two white roofs, these are open, covered areas of which the lower is the kitchen (next to which on the left is the house occupied by the cooks) and the one above is where the coolies mess. Still further to the left near the cliff edge is the bathing house, which owing to improved water supply can now I believe be used; annexed to it is now a room for drying clothes. The small erections used by way of w.-c.s. can be detected; the excreta are cast over the cliff into the ocean. On the point at the extreme left some of the hospital buildings can be perceived. The cove on the right has a sandy beach and is used by the coolies for sea-bathing.

Fig. 2. Shows the arrangement of the coolie houses as a street.

Fig. 3. Shows the construction of one of the houses in more detail; in front is a table used for gambling in the evenings till 9 p.m.

Fig. 4. Gives an idea of the site and construction of one of the hospital buildings. The shed below is the mortuary, where corpses are placed for not more than a few hours previous to their burial.

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The following references do not include all the works which have been consulted for the purposes of this paper. The bibliography of beriberi is large, and so accessible are the titles of the papers, especially through Dr Scheube's "Die Beriberikrankheit" (Jena, 1894) and "Die Krankheiten der warmen Länder" (Jena, 1903), that it seems superfluous to give an extensive list.

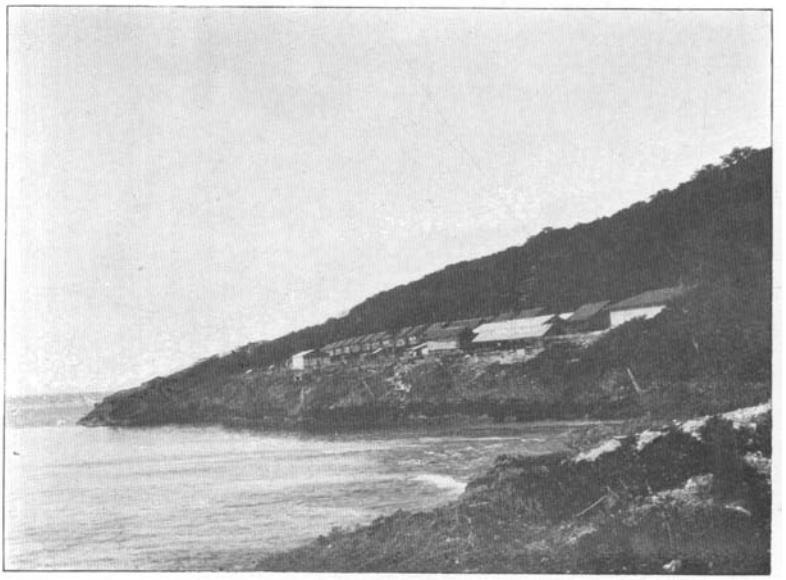

Fig. 1.

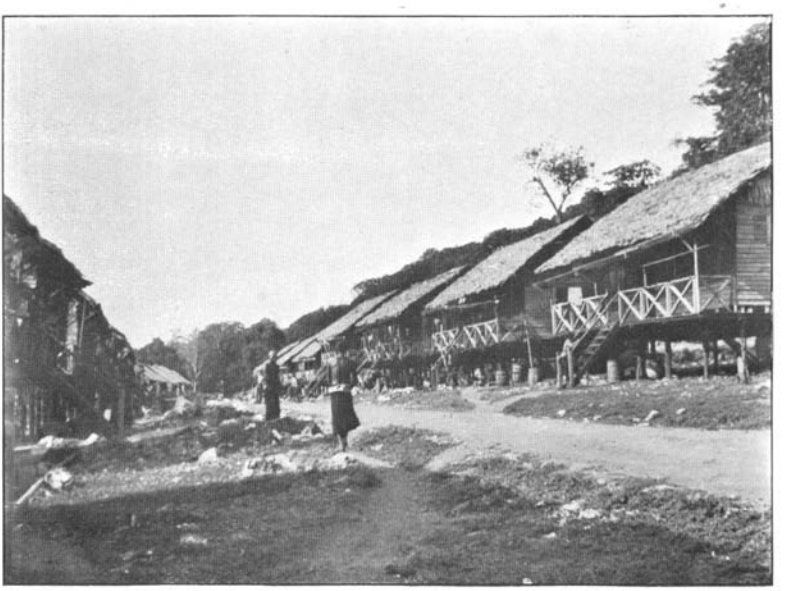

Fig. 2.

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Fig. 3.

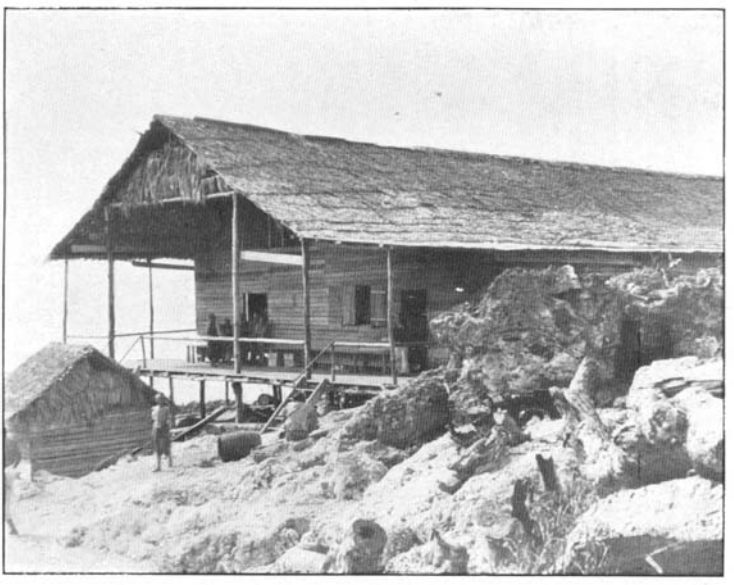

Fig. 4.

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