On an Epidemic Outbreak of Beri-beri.

In the New Gaol, Kwala Lumpur, Selangor,
Federated Malay States,
During the years 1895, 1896, 1897 & 1898.

Being a Thesis for the Degree of M.D.,
Edinburgh University.

by

A. J. McCLOSKY.

M.B.,C.M.

April 1906.

I N T R O D U C T I O N .

Kwala Lumpur is the chief town in the State of Selangor, on the West coast of the Malay Peninsula and is 30 miles inland from the sea coast.

The New Gaol is situated one mile from the town and the site on which it is built was an old disused graveyard and was low lying and swampy. In the process of its construction the contents of no less than 500 graves were removed. As a burial ground it was chiefly used for chinese who had been employed in the extensive tin mines in the neighbourhood.

It is, therefore, quite possible that the majority of the bodies that were buried here may have died of Beri-beri. I am of opinion this ill-chosen site, on which the gaol is built, has an important bearing on the cause of this epidemic. In fairness to the medical authorities of the State of Selangor, I may state that they were not consulted on the choice of this site and were in no way responsible for it. The construction of the gaol was commenced in January, 1893, and was completed and occupied, with the exception of the Infirmary, in January 1895. The Infirmary was

completed and occupied on 23rd April, 1895, so that between January and April, 1895, the sick prisoners were treated in the Old Gaol Infirmary. At the time of the removal of the prisoners to the New Gaol, on January 1st, 1895, the various gaol yards had not been levelled and these were subsequently done by the prisoners.

Construction of the Gaol:

The general plan of the main building consists of a centre block from which radiate four wing blocks like the letter X. Each block consists of three tiers of cells. Each cell measures 12' x 7' x 9'8", satisfactory as regards size and ventilation, and is built in brickwork.

The roofs are drained by means of pipes brought down the inner sides of the blocks, pierce the ground floor and pass out beneath it. This plan of bringing the roof drains inside the building is bad, and during the heavy rains there was always some leakage which tended to keep the gaol damp. The Gaol Infirmary, the female block, the kitchen and the Administration block are detached from the main building. The whole is surrounded by a brick wall 13'6" high. The Warders' (European and Native) quarters are situated outside the wall, but in the immediate vicinity of the Gaol. (A plan

of the gaol and its approaches is attached).

I was Medical Officer in charge of the Gaol during this epidemic and so had the opportunity of becoming intimately acquainted with the nature and symptoms of the disease from its origin and of making many observations and experiments with regard to it. In this thesis I intend adhering as strictly as possible to my observations of Beri-beri as far as it concerns this epidemic and confining myself to its Aetiology, clinical features and treatment. I regret the absence of microscopical examinations of the tissues and electrical tests; the reason for their absence being want of proper apparatus and the time to work out abstruse patholofical points.

I do not, therefore, attempt to give a treatise on the pathology of the disease, but base my thesis merely on the clinical facts here recorded.

A E T I O L O G Y .

What is the cause of this disease? Innumerable speculations have been advanced; but so far none of these have been proved to be correct. My observations of this epidemic seem to favour the miasmatic theory. Comparing the years 1892, 1893

and 1894, when all the prisoners were accommodated in the old gaol, with the years 1895, 1896, 1897 and 1898, when they were accommodated in the new gaol, it will be observed from the table below that during the latter four years there was a considerable increase in the number of cases of Beri-beri treated in the gaol infirmary with a corresponding increase in the number of deaths:-

Table I.

Years.	1892	1893	1894	1895	1896	1897	1898
Prison Strength		283	285.6	390.3	372.3	428.4	414.5
No. of cases.	10	3	5	140	499	297	68
No. of deaths	Nil	1	nil	27	47	55	2
Percentage of deaths	nil	33.33	nil	19.28	9.41	18.51	2.94
	Old Gaol.			New Gaol.			

None of the cases during the years 1892, 1893 and 1894 contracted the disease in the Old Gaol, but were admitted with the disease already manifest; whereas most of the cases treated during the years 1895, 1896, 1897 and 1898 contracted the disease in the New Gaol, v. Table II.

Table II.

Year	Fresh cases contracted in New Gaol	Relapses or fresh attacks.	Admitted into gaol with disease.	Total admitted into Gaol Infirmary.	Prison strength.
1895	109	7	24	140	390.3
1896	189	222	73	484	372.3
1897	97	not recorded	not recorded	276	428.4
1898	20	20	20	56	414.5

The vast improvement in 1898 as compared with the three previous years is explained, as will be shown later on, by the absence of the causative factor, soil disturbance,which I propose to propound in this paper and to the adoption of prophylactic measures.

It is evident, therefore, that in this case the outbreak of Beri-beri occurred immediately after a change of location of the prisoners, other conditions remaining the same.

It will thus be observed that the prisoners enjoyed perfect immunity from this disease at the Old Gaol. The case mortality from Beri-beri in the New Gaol Infirmary being extremely high in September 1895, and in view of the apparent influ-

ence of locality on the disease, all patients suffering from Beri-beri were transferred to the Old Gaol Infirmary on October 1st, 1895, in the hope that the change of residence might prove beneficial. The result of this arrangement is shown in Tables III. and IV.

Table III.

Year	Month	No. of cases admitted to Infirmary.	No. of deaths	Percentage of deaths to total treated	Remarks.
1895	Sept.	32	13	31.7	Treated in New Gaol Infirmary.
1895	Oct.	35	8	15.57	Treated in Old Gaol Infirmary.
1895	Nov.	23	4	6.15	
1895	Dec.	25	2	4.25	

Table IV.

	No. of cases treated	No. of deaths.	Percentage of deaths	Remarks.
3rd Quarter 1895	44	13	29.54	Treated in New Gaol Infirmary
4th Quarter 1895	112	14	12.5	Treated in Old Gaol Infirmary
1st Quarter 1896	139	6	4.3	
2nd Quarter 1896	141	17	12.05	Treated in New Gaol Infirmary.

In the majority of those who died in the Old Gaol Infirmary, the disease was far advanced before they were transferred. I can confidently say that amongst those who were transferred in the early stage of the disease very few deaths, if any, occurred. This is well brought out in Table IV. for the 1st quarter of 1896. During this quarter all the prisoners were subjected to frequent and careful examinations with a view to discovering the disease in its earliest stages and bringing about an early removal of the patient from the infected area. During the 4th quarter of 1895 the patients were given a fair trial in the New Gaol Infirmary before they were transferred. Unfortunately this system of removing the patients from the New to the Old Gaol for treatment could not be continued beyond the 1st quarter of 1896, owing to an insufficient staff of warders and refractory conduct of the prisoners. The result was an increased death rate during the 2nd quarter of 1896. So long as the affected prisoners were treated in the new Gaol Infirmary, little or no improvement was observed in their condition; they rapidly got worse and died; whereas the patients treated in the Old Gaol Infirmary rapidly improved with little or no medication, especially those removed in an early stage of the disease. I visited both Gaol Infirmaries daily, and the contrast

between the two sets of patients was most marked. As long as they were in the New Gaol Infirmary, they bore an anxious and distressful look and showed no interest in life; and when asked how they were, the invariable answer was "No better" or "worse", followed by a long description of their symptoms and a request for innumerable articles of diet, such as gin, pineapple, sweet potatoes, etc. on which they seem to pin their faith.

In the Old Gaol Infirmary, on the other hand, the patients bore a cheerful, happy and contented countenance, and when asked "How are you this morning?" received the answer "I am better", "I am all right".

The food for the Beri-beri patients, treated in the Old Gaol, was not only exactly similar in every respect to that used in the New Gaol, but it was actually cooked in the New Gaol with the food for the other prisoners and was conveyed in a handcart to the Old Gaol.

Encouraged by the evidently beneficial results of change of locality on the sick prisoners, arrangements were made to experiment still further in this direction. Consequently on October 21st, 1895, sixty prisoners, showing no signs of Beri-beri and in apparently good health were transferred from the New Gaol to the Old Gaol, and from this date to April 1896 a large gang of prisoners were confined in the

Old Gaol, amongst whom no fresh cases of Beri-beri occurred. The monthly average number of prisoners in each Gaol during this period, with the number of fresh cases of Beri-beri occurring among them, is shown by the following figures:-

Table V.

Month.	Av. No. prisoners in New Gaol	No. of Beri-beri cases New Gaol.	Av. No. prisoners in Old Gaol.	No. of Beri-beri in Old Gaol.
Oct. 1895	297	35	72	Nil.
Nov. 1895	337	23	119	Nil.
Dec. 1895	271	25	128	Nil.
Jan. 1896	280	29	133	Nil.
Feb. 1896	286	47	130	Nil.
March 1896	271	43	119	Nil.

From October 1st to December 14th 1895, all foods supplied to the healthy prisoners in the Old Gaol as well as to the Beri-beri patients transferred from the New Gaol, was, as has been stated before, cooked in the New Gaol with the food for the other prisoners and carried to the Old Gaol, the diet, therefore, being exactly the same at both Gaols.

After December 14th, 1895, the rations for the prisoners in the Old Gaol were cooked in that Institution, raw rations being sent daily from the New Gaol. The results of these observations and experiments, originally made with the intention of proving that Beri-beri is a "place disease", seem to indicate very clearly in, at any rate, this instance that there was no connection of any kind between the outbreak of Beri-beri and the food supply.

There are in the same town three other large Institutions under the care of the Medical Department, viz. the District Hospital, containing 450 beds; the Leper Hospital with 130 beds; and a Hospital for Incurables, containing 60 beds. These Institutions and the New Gaol are within three miles of each other and the conditions of temperature, moisture of air, etc. are practically the same. The food supplied for the inmates of these Institutions is of the same kind as that used in the New Gaol and is obtained from the same contractor. Throughout the years 1895, 1896, 1897 and 1898, during which Beri-beri has been endemic at the New Gaol, there has been no outbreak of the disease at any of the Institutions mentioned.

If, therefore, infected food was the cause of Beri-beri among the prisoners in the New Gaol, why did it not cause a similar outbreak in the District

Hospital, Leper Asylum and Hospital for Incurables?

If, however, it is assumed that the food became infected during the period it was kept in the New Gaol, then the complete immunity enjoyed by the prisoners at the Old Gaol effectually disposes of this supposition.

The facts which I have so far brought forward tend to show that the cause of this epidemic is local and that the poison is most likely to be found in the exhalations from the soil (miasm). I am of opinion it is most probably a specific organism generated in the soil and fostered by dampness.

I say fostered by dampness because a recrudescence of the disease during this epidemic always occurred in the months of September, October, November and December, when the rainfall was abundant and the air laden with moisture.

Effect of soil disturbance:

I shall now attempt to show the effect of soil disturbance, within and around the Gaol on this epidemic. Every instance of fresh soil disturbance was followed by a recrudescence of the disease, and, on the other hand, prohibition of the prisoners from doing any earthwork and cessation of all soil disturbance were followed by marked improvement. Disturbance of the soil, no doubt, sets free the germs

which are then carried away in the exhalations from the soil; stagnation of air within the gaol walls favouring its condensation.

The absence of such conditions as stagnation of the air and condensation of the poison outside the gaol would account in a great measure for those resident outside, but in the immediate vicinity of the gaol, such as the warders, not being attacked.

From January 1895 to November 20th, 1897, the soil within and immediately around the Gaol was never at rest; it was constantly being disturbed in the process of levelling the yards, making the approaches to the Gaol, and constructing the Recreation Ground for the Warders. During the year 1898, there was no disturbance of soil within and around the Gaol s and a reference to Table I. will show what a marked improvement followed in the number of cases and number of deaths as compared with the three previous years. I must, however, mention here (this will be again referred to) that during 1898 the whole Gaol was frequently subjected to disinfection and all Beri-beri patients were treated with mercury internally from the very earliest manifestation of the disease. The disinfection of the Gaol, I certainly think, helped to keep the disease in check, and to the internal administration of Mercury, I partly attribute the low death-rate.

I shall now attempt to show that particular instances of soil disturbance produced a recrudescence of the disease.

During June, July and the early part of August, 1896, there was no disturbance of the soil; but, on 11th August, cutting of the soil and levelling of the road in front of the Gaol (the Gaol Road on the plan) commenced. This work was undertaken by the prisoners themselves with the result that a marked increase in the number of Beri-beri cases occurred in September, v. Table VI.

Table VI.

	No. of cases admitted.	No. of deaths.	Percentage of deaths.	Remarks.
June 1896	39	3	5.17	No soil disturbance.
July 1896	22	2	5.26	
Aug. 1896	15	nil	nil	1-11 no soil disturbance. 12-31 Soil disturbance.
Sept.1896	51	7	11.27	Soil disturbance.

During the 2nd quarter of 1897 there was no disturbance of soil, but on June 30th 1897, the construction of the Recreation Ground was commenced and

was not completed until November 20th 1897. This cutting and turning up of the soil was immediately followed by an exacerbation of the disease in the 3rd and 4th quarters of 1897, and ceased almost as abruptly during the 1st quarter of 1898 when all soil disturbance had ceased, v. Table VII.

Table VII.

	No. of cases treated.	No. of deaths.	Percentage of deaths.	Remarks.
2nd Quarter 1897	40	5	12.5	No soil disturbance.
3rd Quarter 1897	93	9	9.6	Soil disturbance.
4th Quarter 1897	122	24	19.6	
1st Quarter 1898	21	nil	nil	No soil disturbance.

The following table further shows the marked decrease in the number of cases admitted into the Infirmary during December 1897 after cessation of all soil disturbance on November 20th.

Table VIII.

	No. of cases admitted.	No. of deaths.	Percentage of deaths.	Remarks.
Oct. 1897.	35	8	12.5	Soil disturbance.
Nov. 1897	51	16	19.2	1-20 Soil disturbance. 21-30. No soil disturbance.
Dec. 1897	7	nil	nil	No soil disturbance.

I have so far disposed of the Miasmar theory of causation which postulates that there exists some agent, more or less fixed to particular places, producing a poison, the absorption of which (by inhalation or otherwise) by those exposed to it results in Beri-beri. In doing so, I have also touched on the food supply, and,I submit, have effectually eliminated food as being per se a factor in the causation of Beri-beri.

Rice Theory:

I shall now supplement my previous remarks on food-stuffs with special reference to rice, as it has been suggested that Beri-beri is a grain intoxication, the result of absorbing toxic material found

under certain conditions in cereal grains. The diet of the prisoners, as well as the patients in the various large Hospitals in the district consists mainly of so-called Rangoon rice with fresh meat, (beef, pork or goat), salt fish and fresh vegetables.

The following is the scale of diet supplied to the prisoners:-

Articles of diet.	K; Ordinary diet.	L.* Penal Diet.	M;** Punishment diet.	Remarks.
	Ounces daily.	Ounces daily.	Ounces daily.	
Rice	19	20	18	Including 6 oz. for congee at 6 a.m.
Fresh Meat (including bone)	6	-	-	Goat for Indians beef for Malays; beef and pork alternately for Chinese.
Salt Fish do.	4	-	-	
Vegetables	7	4	-	
Beans	-	5	6	
Towgay	2	-	-	
Coconut Oil	1	-	-	For Chinese, Indians & Malays
Curry Stuff	1	1	-	
Salt	-	0 3/8	1	
Pepper	-	0 3/8	-	
Wheat Flour	4 1/2	4 1/2	4 1/2	

*This scale of diet is to be adopted for all Native Prisoners sentenced to imprisonment with hard labour for periods of three months and under; it is also to be adopted for three days in each week (Monday, Wednesday and Friday) during the first six months of their sentence:- (a) For all short-sentenced prisoners with sentences of more than three months; and (b) for all revenue grade prisoners with sentences of more than three months; and (c) for lower grade prisoners.

** This scale is the punishment diet referred to in No.115 of the Prison Rules.

The rice for the prisoners is cooked by steam for 2½ hours, forced through at a high pressure by means of a small vertical engine - a pressure sufficiently high to exterminate all known organisms and destroy all known toxins.

As mentioned before, the new Gaol was occupied in January 1895, previous to which all prisoners were confined in the Old Gaol, about 1½ miles distant. While in the Old Gaol, as already mentioned, none of the prisoners contracted Beri-beri, although they were fed on the same kind of rice and which was procured from the same source as that given them when in the New Gaol.

It is a fact, certainly, that the majority of the victims of Beri-beri are those whose staple food is rice. But if rice is the cause of Beri-beri, how is the perfect immunity from the disease, enjoyed by

the prisoners during the years they were accommodated in the Old Gaol, although eating rice daily, to be explained?

Further why do the Indians, who are also rice eaters, not contract the disease to the same extent as the Chinese and Malays, if rice is the cause? With reference to this latter proposition, it has been suggested that the Indians eat a different rice from that of the Chinese and Malays.

This is so. The Indians eat Bengal rice which is subjected to a process of steaming with the husks on before it is put on the market, whereas the Rangoon rice which the Chinese eat is not so treated. It is suggested that the poison is in the husk and is, therefore, destroyed by steaming, whereas in the Rangoon rice, in the process of separating the husk by pounding the poison is able to infect the grain and produce in it a chemical poison analogous to the diseased maize which causes pellagra. Here again the argument is brought forward, how is the freedom from the disease amongst the prisoners in the Old Gaol explained? Further, I have had under treatment for Beri-beri Indians who have always partaken of Bengal rice.

Water Supply:

On August 10th, 1895, cholera broke out in the Gaol. One hundred and twenty six cases occurred, causing 68 deaths. This outbreak of cholera was distinctly traceable to the water-supply, no new cases having occurred after the introduction of a fresh supply of water from the Reservoir. Now, as this outbreak of Beri-beri followed shortly the outbreak of Cholera and at the time when Reservoir water was first laid on, it was thought at one time that the water-supply might be the causative factor. Previous to reservoir-water, well-water was used. It was, therefore, thought advisable to test the effect of the water supply on the further progress of the disease and this was done by giving the prisoners at the New Gaol well-water and those at the Old Gaol reservoir water for a period of three months. The results of this trial are shown in the table below (Table IX.) and prove that the water-supply could not be credited with having been a factor in the causation of the disease. On April 1st, 1896, all water for drinking purposes in the New Gaol was taken from a well; the reservoir supply being entirely stopped until July 1st when it was again utilized. Reference to the table will show that there was a slight improvement in the number of prisoners attacked during the months in which well-water was used;

but this improvement was maintained to an even more marked extent in July when reservoir-water was again resorted to. The slight improvement may be attributed to the fact that all disturbance of the soil within the gaol had ceased and the yards had been covered with several feet of earth brought from a distance and had been turf-clad. The death rate was higher during the months in which well-water was used and this is explained by the patients not being removed to the Old Gaol for treatment as in the three previous months. Sick prisoners in the Old Gaol still continued to improve in spite of the fact that reservoir-water was used in that building. Boiling the water was also tried and was found to have no effect in checking the disease.

Table IX.

Month.	No.of cases admitted to Infirmary.	No. of deaths	Percentage of deaths.	Remarks.
Jan. 1896	29	3	?	
Feb. 1896	47	3	4.10	Reservoir water.
March 1896	43	nil	nil	
April 1896	42	8	12.12	
May 1896	36	6	11.32	Well-water.
June 1896	39	3	5.17	
July 1896	22	2	5.26	Reservoir water.

Relation of Beri-beri to Meteorological conditions:-

The disease is to a certain extent influenced by the damp condition of the atmosphere because exacerbations of the disease, apart from any relation to soil disturbance, always occurred during the last four months of the year when the rainfall, as a rule, was excessive and the atmosphere laden with moisture. Even if the rainfall is not excessive, the atmospheric moisture is always high during these months, much more so than the other months of the year.

The above facts are well brought out in the following statistics:-

Table X.

	1895.			1896			1897			1898.		
	Admissions	Deaths	Rainfall	Admissions	Deaths	Rainfall.	Admissions	Deaths	Rainfall	Admissions	Deaths	Rainfall
1st Quarter	4	nil	18.86	119	6	18.5	71	17	40.9	9	nil	23.65
2nd Quarter	10	nil	27.02	117	17	19.5	30	5	24.2	2	nil	29.09
3rd Quarter	43	13	19.7	88	9	19.8	82	9	18.1	12	1	26.72
4th Quarter	83	14	34.6	160	10	51.03	93	24	32.2	33	1	26.82

I would lay special importance on the figures for 1898, as during that year there was no soil disturbance to influence the disease in any way and because the rainfall for the 4th quarter was not excessive compared with that for the three previous quarters, yet the number of cases admitted was considerably in excess. This bears out what I said before that, apart from any soil disturbance and apart from the rainfall, there was always an aggravation of the disease during the last four months of the year. The only condition that might influence the disease during the last four months of the year is increased atmospheric moisture. Table XI. shows the aggregate number of cases admitted into the Gaol Infirmary and the aggregate rainfall for the three periods of four months, for the years 1895, 1896, 1897 and 1898.

Table XI.

	1895.		1896		1897		1898.	
	Admissions	Rainfall.	Admissions,	Rainfall.	Admissions	Rainfall	Admissions	Rainfall.
1st 4 months.	7	33.16	161	26.66	74	48.75	9	40.14
2nd 4 "	18	23.78	112	20.5	74	29.35	13	27.06
3rd 4 "	115	43.32	211	61.85	128	37.49	34	39.08

This epidemic of Beri-beri in the New Gaol, therefore, had a remarkable relationship to the rhythmical meteorological changes experienced here, and it is strikingly brought out in the table below that cases increase in number during the North East Monsoon.

Table XII.

	1895	1896	1897	1898
	Admissions	Admissions	Admissions	Admissions
N. E. Monsoon.	87	279	164	42
S. W. Monsoon.	53	205	112	14

It is doubtful whether mere increase in the rainfall has any direct influence in the causation of Beri-beri. It is probable rather that it affords moisture necessary for the development of the specific organism.

Table XIII. shows the monthly admissions, the number of deaths and the meteorological conditions for the four years 1895 - 1898.

TABLE XIII. Relation of Beri-beri to Meteorological Conditions.

Month.	1895. Temperature.					1895. Rainfall.		1895. Cases of Beri-beri.		1896. Temperature					1896. Rainfall.		1896. Cases of Beri-beri.	
	Solar Maximum.	Minimum on grass.	Shade Maximum.	Shade Minimum.	Range.	Amount in inches	Degree of humidity.	Admissions.	Deaths.	Solar Maximum.	Minimum on grass.	Shade Maximum.	Shade Minimum.	Range.	Amount in inches.	Degree of humidity	Admissions	Deaths.
Jan.	150.0	63.9	90.9	70.1	20.8	2.56	82	1	Nil	149.9	69.6	89.5	70.3	19.2	7.75	81	29	3
Feb.	151.6	64.6	91.3	70.4	20.9	10.53	79	1	Nil	147.9	67.9	89.6	70.0	19.6	.91	82	47	3
March	153.8	64.6	90.6	70.6	20.0	5.75	79	2	Nil	149.7	67.7	89.3	71.1	19.2	9.87	78	48	0
April	153.5	64.9	91.2	73.4	17.8	14.32	77	3	Nil	149.2	67.6	89.5	69.6	19.9	8.13	80	42	8
May	154.9	64.7	91.5	72.2	19.3	6.78	78	7	Nil	150.6	67.4	89.5	70.0	19.5	6.17	81	36	6
June	152.0	65.4	91.0	72.7	18.3	5.92	77	0	Nil	149.6	67.7	89.8	70.3	19.4	5.28	83	36	3
July	150.6	66.9	90.5	70.2	20.3	3.93	79	1	Nil	150.7	67.4	89.6	70.5	19.1	3.04	82	23	2
Aug.	148.2	66.5	89.4	69.6	19.8	7.15	80	10	Nil	148.6	67.5	89.8	70.4	19.4	6.01	82	16	0
Sept.	151.9	66.1	89.8	70.9	18.7	8.65	83	32	13	147.7	67.4	89.9	70.4	19.5	10.82	81	50	7
Oct.	143.6	67.5	88.8	70.8	18.0	23.44	81	35	8	146.6	67.5	90.8	70.8	20.0	17.11	82	46	2
Nov.	149.8	67.1	89.2	71.2	18.0	4.6	80	23	4	147.1	67.3	90.4	70.8	19.6	18.85	80	67	2
Dec.	147.0	66.8	88.9	70.7	18.0	6.63	82	25	2	146.7	67.3	88.5	70.6	17.9	15.07	81	44	6
Mean	150.5	65.7	90.2	71.0	19.1	100.26	79	140	27	148.7	67.7	89.6	70.3	19.3	109.01	80	484	42

1897.

Month.	Temperature.					Rainfall.		Cases of Beri-beri.	
	Solar Maximum.	Minimum on grass.	Shade Maximum.	Shade Minimum.	Range.	Amount in inches.	Degree of humidity.	Admissions.	Deaths
Jan.	149.8	67.4	90.6	71.0	19.6	16.12	76	31	9
Feb.	145.3	67.5	89.9	71.9	17.9	7.06	82	16	5
March	150.6	67.6	91.3	73.0	18.3	17.77	79	24	3
April	151.5	67.2	91.7	73.1	18.5	7.8	76	3	1
May	146.8	67.0	89.7	71.2	18.4	8.54	77	10	3
June			90.4	72.3	18.1	7.91	78	17	1
July			90.5	71.3	19.1	5.19	82	12	1
Aug.			90.7	71.8	18.8	7.71	79	35	2
Sept.			90.7	71.8	18.9	5.27	74	35	6
Oct.			91.3	72.3	19.0	9.28	78	35	8
Nov.			90.3	72.1	18.2	8.7	76	51	16
Dec.			89.4	72.1	17.2	14.93	82	7	0
Mean			90.5	71.9	18.5	Total 116.28	78	276	55

Instrument out of order.

1898.

Temperature					Rainfall.		Cases of Beri-beri.	
Solar Maximum.	Minimum on grass.	Shade Maximum.	Shade Minimum.	Range.	Amount in inches	Degree of humidity.	Admissions.	Deaths.
		89.9	71.5	18.3	6.62	81	4	Nil.
		90.6	70.6	20.0	6.72	71	3	"
		91.5	71.5	19.8	10.31	73	2	"
		92.1	71.1	21.0	16.49	73	0	"
		92.9	71.5	21.3	6.30	24	1	"
		96.1	74.3	21.8	6.3	78	1	"
		92.6	70.8	21.7	6.35	77	5	"
		92.6	70.8	21.8	8.11	80	6	1
		92.5	71.2	21.3	12.26	80	1	Nil
		89.4	72.3	17.0	9.53	78	1	"
		87.7	72.0	15.6	12.35	87	10	"
		89.6	73.3	16.2	4.94	80	22	1
		91.4	71.7	19.6	Total 106.28	77	56	2

Instrument out of order.

Conclusions as to Aetiology:

As this epidemic of Beri-beri had no direct, if any, connection with food, most certainly not with rice, nor any connection with the water-supply, it is safe to conclude that neither food nor drinking water play a part in the production of the disease. I am of opinion it is due to a poison in the soil. The nature of the poison is probably an organism which is carried away in the miasm and enters the system through the alimentary tract and produces its initial lesion in the stomach. My reason for saying that the initial lesion is in the stomach is because the initial symptom complained of, as will be shown later, is referred to the stomach and because of certain post-mortem changes seen in the stomach in cases of acute Beri-beri.

That it is produced by a living germ is proved by the fact that the cause can be transported from place to place and therefore cannot be of a climatic or meteorological nature, and that when so transported it can multiply and spread and therefore cannot be of an inorganic nature. Spontaneous multiplication is a property peculiar to living things, therefore the originating cause of Beri-beri is a living thing - a germ. Disturbance of the soil facilitates the dissipation of this germ from the soil. Moist soils

are known to harbour pathogenic organisms and why not the germ of Beri-beri? Infection of the building accounts to some extent for the continued prevalence of the disease after all soil disturbance had ceased. Various theories as to the causation of Beri-beri have been propounded. Some of them which corroborate the explanation of this epidemic may be briefly referred to:-

1. Dr Anderson of the Imperial Naval Hospital, Tokio, Japan, says:- "It is an atmospheric poison of local origin."

2. Dr Duane Simmons, President of the Foreign Health Board, Yokohama, says:- "It is a specific miasm or ground exhalation."

3. Dr Roy of Bohwanipore says:- "It is due to a poison generated in a suitable soil, under a certain amount of heat favourable to its production and multiplication."

4. Dr Bentley says:- "It is probably a specific particulate organism generated in the soil."

5. Native Japanese Doctors ascribe it to unhealthy exhalations from the soil.

Other causes such as overcrowding and various food stuffs such as rice, pork, fish, and the absence of fat in the dietary have all been suggested as possible causes. My own experience does, in no way, support these views.

That overcrowding may assist the spread of the disease, when once started, seems possible. I have seen Malays living in isolated huts in the jungle

contract Beri-beri. Such, surely, are not over-crowded; but they are exposed to the exhalations from a damp, jungle soil.

With regard to rice as a possible cause, I have already discussed this.

With regard to pork, I may state that the Japanese and Malays are prohibited by their religion from eating this article of diet, yet they are very subject to Beri-beri. In the Gaol the Malay prisoners got no pork, yet contracted Beri-beri.

With regard to the absence of fat in the dietary, I may state that the Chinese prisoners who were the chief sufferers in this epidemic, got six ounces of pork, half of which was fat, four times a week. A favourite dish of the Chinese coolies is a soup made of pork in which lumps of fat can be seen. Yet the coolies working in the tin mines are particularly subject to the disease.

In the Beri-beri epidemic of 1898 in the Paulo-Condore Penal Settlement, French Cochin China, this absence of fat in the dietary is assigned as the cause of the epidemic and Dr Laurent considered that the decline in the incidence of the disease and its subsequent disappearance is chiefly due to the improved dietary. The diet was improved by the inclusion of 250 grammes of fresh pork per diem, five times a week and 50 grammes of pork fat per diem for

two days a week. The report further says:- "Besides this(the inclusion of fat pork) certain hygienic measures were also decided on and carried out." I am inclined to attribute the decline and subsequent disappearance of the disease to these hygienic measures rather than to the inclusion of the fat element in the dietary.

The fish theory is eliminated by the following experiment, the figures for which, I regret, were lost. Fish was cut off the diet of a number of healthy prisoners during a period of six months, Several of these prisoners contracted Beri-beri, although not an ounce of fish of any description entered into their diet.

The nitrogen-starvation theory - Takaki suggests that Beri-beri is due to an improper proportion of nitrogen and carbon in the dietary which, instead of being 1 N to 15C , he found in all epidemics to be 1 N to 22 C. The proportion of nitrogen to carbon in the prison scale of diet is high, about 1 N to 12 C. There must be something more than nitrogen starvation at the root of Beri-beri.

In the Singapore Gaol in 1898 there were two kinds of diet in use - ordinary and penal. The ordinary diet was a sufficiently liberal one, including fresh meat and wheaten flour; the penal diet contained no meat.

Nevertheless, the greater number, absolute and proportionate, of cases of Beri-beri occurred among those on ordinary diet, that is, on the diet richest in nitrogen.

There was no nitrogen starvation among the inmates of the Richmond Asylum before or during the epidemic of Beri-beri.

There are other additional instances on record in which Europeans, enjoying a liberal diet, have fallen victims to Beri-beri.

Relation of Beri-beri to race and general physical condition.

In the Gaol epidemic the Chinese prisoners were the chief sufferers; their number attacked was quite out of proportion to that of the Indian and Malay prisoners. The disease further had a marked tendency to attack the robust prisoners rather than the weak and delicate. Some of the hardest workers in the Gaol, men of exceptionally strong physique, succombed to the disease. It is a well known paradox that Beri-beri attacks those who are healthy and well fed rather than the sickly and indigent, and this is upheld as a strong point in favour of the rice theory. Thus the rice that fed, conveyed also the poison that ultimately slew.

Relation of Beri-beri to period of Incubation.

During the years 1895 and 1896 I carefully watched 198 prisoners, healthy at the time of admission into Gaol, for the first sign or symptom of Beri-beri and the result was as follows:-

39	Developed	the	disease	during	1st	month	of	incarceration.
30	"	"	"	"	2nd	"	"	"
29	"	"	"	"	3rd	"	"	"
18	"	"	"	"	4th	"	"	"
22	"	"	"	"	5th	"	"	"
23	"	"	"	"	6th	"	"	"
17	"	"	"	"	7th	"	"	"
11	"	"	"	"	8th	"	"	"
7	"	"	"	"	9th	"	"	"
4	"	"	"	"	10th	"	"	"
2	"	"	"	"	11th	"	"	"
198								

This experiment shows that the period of incubation is variable and is probably dependent on the intensity and concentration of the poison. More cases, however, developed the disease during the 1st, 2nd and 3rd months of incarceration respectively than during any one other month. During the years 1895

and 1896 disturbance of the soil within the gaol was constantly going on, with short intervals of cessation, so that the intensity of the poison varied. During the year 1898, however, when all soil disturbance had ceased and consequently the strength of the poison was more constant, I observed that most of the cases showed the disease at the end of the third month of incarceration. In connection with this point, I shall now refer to two other epidemics of Beri-beri, one on a ship and the other in a prison in Western Africa, which corroborate my observation that most cases in 1898 developed the disease after three months of incarceration.

"In 1896, a ship, the "Lodestar" of London, arrived at Falmouth with a cargo of rice from Rangoon after a long voyage, during which the entire crew was attacked with Beri-beri and three had died from the disease. The ship was sold to German owners and re-named the"Steinbok".At Amsterdam she received a new crew and took in fresh provisions; she then sailed for Java where she loaded with sugar. She left Java on January 27th, 1898, all being well on board. On April 29th, when off St. Helena and after being <u>three months</u> at sea, Beri-beri broke out, and when the ship arrived at Bermuda the entire crew, with the exception of one man and one boy, were down

with the disease."

In the Lancet, April 3rd, 1897, the following account of an outbreak of Beri-beri in Dakar Prison, Senegal, Western Africa, appeared:-

"A hundred and forty nine military prisoners caused twenty four admissions during the twelve months; but out of 446 civilians, only 26 were attacked. Length of residence and want of occupation were the chief determining cause. The Civilian prisoners used to be sent out to work in the vicinity but the enlisted men claimed exemption and lay idle all day long, wrapped up in their blankets. The affection generally commenced towards the third month of incarceration. At no stage of the disease were febrile phenomena to be detected, in all cases the most complete apyrexia. The only treatment of any permanent efficacy was an entire change of scene."

This epidemic in the Dakar Prison bears a very close analogy to that in the Selangor Gaol in other respects besides in the period of incubation. In the Selangor Gaol the long-sentenced prisoners, corresponding to the enlisted men in the Dakar Prison, were not sent out to work and they formed the greater number of victims. The short-sentenced prisoners worked a good deal outside the gaol and were protected from the disease to a marked extent. In the

year 1896, out of 411 prisoners who contracted the disease in the Gaol, only 77 were short-sentenced prisoners, and in 1897, out of 276 cases admitted to the Gaol Infirmary, only 52 were short-sentenced prisoners.

As in the Dakar Prison, so here, there was complete apyrexia and the only treatment of any efficacy was an entire change of scene, viz. the removal of the patients to the Old Gaol. The points of analogy then are:-

(1) The relation of the disease to the period of incubation, viz. three months.

(2) The greater susceptibility to the disease of these prisoners who were confined within the Gaol and not sent out to work.

(3) The total absence of fever.

(4) The efficacy of change of scene in the treatment of the disease.

Varieties of Beri-beri:

As far as this epidemic is concerned, we may consider four varieties:-

(1) The Acute Form.

(2) The wet or dropsical form.

(3) Mixed forms.

(4) The dry or paralytic form.

The dropsical form was met with in most of the cases and next to this came the mixed form. By mixed form I mean all those cases in which dropsical and paretic symptoms were present at the same time.

The paralytic form was rarely met with as a primary disease, only as a sequelae to the dropsical or mixed forms.

Signs and Symptoms.

The disease first made its appearance in the New Gaol in alarming proportions in September 1895, during which month thirty-two cases occurred with thirteen deaths, whereas for the previous eight months there were only twenty-five cases with not a single death, v. Table XIV.

Table XIV.

	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
No. of cases admitted into Infirmary.	1	1	2	3	7	nil	1	10	32	35	23	25

The epidemic continued until November, 1897, after which date it may be said to have lost its epidemic character, although a few new cases still

originated. These, however, were mild cases and very amenable to treatment in the infected locality; no change of scene was found necessary. A large porportion of the cases in the year 1898 were relapses, v. Table II.

Beri-beri in this epidemic was a very fatal disease, characterised especially by general dropsy, muscular tenderness, numbness, absent knee-jerks and a tendency to dyspnoea.

Paralysis was not a marked feature.

A feature of the disease was the utter unexpectedness of the issue. There was never any possibility of predicting how a case might eventuate. Those most severely attacked may recover, while cases, to all appearance trifling, may die without warning.

Such acute cases as the following were not uncommon during this epidemic -

A prisoner would go out to work at 6 a.m. apparently well and making no complaint; he would suddenly, while working and without any warning, experience great oppression in the chest and epigastric region and dyspnoea; his face livid; he would be brought into the Infirmary, say about 8 a.m., and death would supervene within a few minutes.

One prisoner was actually found dead in his cell in the morning; he had been to work the previous day,

apparently well and had made no complaint whatever. In such acute cases death happens by syncope from paralysis of the heart. The above are examples of the acute variety of Beri-beri.

The clinical history in most of the cases, however, was asfollows:- The first symptom complained of was a sense of oppression and fulness in the stomach, increased after the smallest quantity of food. The prisoners came generally complaining in the Malay language thus:- "Sahya t'âbulîk mâkan nasi, prŭt Kennyang, Kasi Susu," which, interrupted into English, means "I can't eat rice, my stomach feels full, give me milk." This complaint was made by the prisoners when they looked apparently well and in good health and were able to do hard labour. This symptom was unaccompanied by any of the other signs or symptoms of the disease. This seems to indicate that the initial lesion is in the stomach. At first I looked upon this symptom very triflingly, merely as a form of dyspepsia or as a feint for a change of diet, but soon discovered that it was a premonitory sign of true Beri-beri. This premonitory symptom frequently subsides for a time, for a few days or even a week. At the end of this time the patient again seeks advice. He now comes complaining of numbness of the legs, oedemia of legs, pain

in the calves, heaviness and weakness of legs and difficulty in walking. He is, however, able to walk tolerably well and with, certainly, no characteristic gait except a slight stiffness. He has loss of patellar reflex , slight oedema along the inner surface of the tibia and tenderness of calves. Cutaneous sensibility is distinctly impaired in the feet and legs. The organs of special sense were not affected; the functions of the brain were intact in all cases and there was no tremor. The digestive symptom, save for the complaint of fulness, was normal. Vomiting was occasionally present in the later stages of the disease and was a most serious prognostic sign.

As the disease advanced the oedema would spread to the feet, knees, thighs and gradually become general, involving the penis, scrotum, trunk, upper limbs, neck and face. It is most often symmetrical. The patient in this condition resembles a case of advanced Bright's disease or is like the bloated body of the drowned.

I enclose photograph of a patient in this condition. This particular patient made an excellent recovery. In the face the oedema is sometimes enormous, the eyelids being so greatly tumefied that the patient cannot keep his eyes open, but is obliged to separate the lids with his fingers. The oedema

was very often and always in fatal cases accompanied by effusion into the serous cavities (ascites, hydrothorax, hydropericardium). Later on distressing dyspnoea, palpitation and violent heart's action and painful sense of oppression over the heart would supervene; the pulse becoming smaller and weaker and the patient soon dies nearly suffocated. Aphonia was sometimes present in the severe forms, due, no doubt, to paralysis of the laryngeal muscles from involvement of the recurrent laryngeal nerve. There was never any tendency to nutritive changes in the skin. The superficial reflexes were invariably present. Fever was absent in most of the cases and when present, was intercurrent and slight and bore no constant relation to the disease.

Anaemia, which has been given as a prominent feature in Beri-beri, was entirely absent in this epidemic. In fact, the disease, as I have already mentioned, showed a marked tendency to attack the strong, healthy and robust prisoners rather than the sickly.

If recovery was taking place, the oedema would gradually disappear, the general health improve and the patient would leave Hospital and resume labour, but only for a time, as he invariably soon got a relapse. If he gets over this relapse, he goes through

the same proceedings as before, but ultimately dies in one of his relapses.

The above description was the type of the disease usually met with in this epidemic.

In a few instances the disappearance of the oedema was followed by paresis of the muscles of the lower limbs, chiefly the extensors, leading to well-marked ankle-drop and giving rise to the characteristic Beri-beri gait. Occasionally there was complete paralysis of the muscles and wasting of the special groups affected. The patient now becomes quite helpless, he is unable to stand or even raise himself up to the sitting posture; he cannot dorsiflex his foot and can only feebly flex the thigh on the trunk. The extensor muscles of the upper limbs were less frequently affected, leading to wrist-drop. In bad cases a marked clawing of the hands was observed, due to paralysis of the Interossei muscles. The patient in this condition is unable to feed himself, for he cannot hold anything in his paralysed hands.

The Beri-beri gait resembles very closely that seen in Locomotor Ataxia. The legs, wide apart so as to have a broad basis, are suddenly raised high up so as to free the toes from the ground and then are brought down violently flat on the ground with

a movement at the same time backwards. His body is bent well forwards in order to keep the centre of gravity over the line of support and to keep himself from falling backwards - the common extensors of the thigh being too weak to do their work.

Peculiar attitudes assumed by the paralytic Beri-beric.

(1) When standing, he does so with the aid of a stick; his feet wide apart and the body bent forwards, leaning on the stick. If he attempts to bring his feet together, he falls down.

(2) When getting up from the sitting posture to the erect, he keeps the feet wide apart and rests both hands on the cot. With the feet close together and without the aid of his hands, he is unable to assume the erect posture.

The paralytic form of the disease, which is so frequently met with in the various hospitals throughout the Straits Settlements and Malay States, was never seen in this outbreak as a primary form. It was always, when met with, secondary to the dropsical or mixed forms. I am, therefore, of opinion that oedema always precedes the paresis and palysis.

Natives, as a rule, seek hospital treatment after giving Nature and native drugs a prolonged trial and when they find they are helpless, i.e. when the oedema has disappeared and paralysis has set in. This explains why the paralytic form of the disease is more frequently met with in the hospitals than the

dropsical. If the paralytic form does occur as a primary disease, then why in this epidemic did no case occur out of the hundreds of prisoners attacked?

Observations of this epidemic, therefore, seem to prove that the vaso-motor and sensory nerves are always first attacked and the motor nerves subsequently.

Systolic murmurs were frequently heard over the cardiac areas, most marked over the Pulmonary and Tricuspid. These murmurs were incompetent murmurs due to dilatation of the ventricles. No organic lesion of the valves were ever found. Accentuation and reduplication of the second sound at the pulmonary area was sometimes heard.

Temporary albumenuria was present in a few cases and the quantity of urine was sometimes diminished.

Nothing remarkable was observed with regard to the temperature; it was essentially a non-febrile disease.

Relapses were common.

In cases of so-called recovery, it is worthy of note that the patellar-reflex still remains absent and when it does return is worthy of further investigation.

I have examined the knee-jerks of several prisoners as long as two years after their last attack and

found the reflex still absent. The following table (Table XV.) shows thirteen prisoners whose patellar reflexes were tested on three different occasions subsequent to their last attack and in most of them they were still absent. At the dates of examination the prisoners were apparently well and doing hard out-door labour.

TABLE XV.

Prisoners.	Date of admission to Gaol.	Date of last attack.	Dates of Examination.		
			5.5.98	30.11.98	5.4.99
Upper Grade 17	Jan. 1895	19.11.97	K.J. Absent	Present	
" " 23	" "	28.9.97	" "	Absent	Discharged
Middle " 1	" "	9.11.96	" "	"	Absent
Upper " 28	May "	2.10.97	" "	discharged	
Middle " 16	July "	7.11.96	" "	Absent	Absent
" " 52	" "	8.1.97	" "	"	"
Upper " 29	" "	7.8.97	" "	"	"
" " 31	" "	28.9.97	" "	Discharged	
" " 37	Aug. "	9.6.96	" "	"	
" " 72	" "	4.8.97	" "	Present	
Middle " 56	" "	13.8.97	" "	Absent	Absent
" " 46	" "	9.11.97	" "	Present	
Lower " 16	Nov. "	15.11.97	" "	Absent	Absent

Examination on May 5th, 1898, showed the patellar reflex absent in all the prisoners. On November 30th, 1898, it had returned in three of the prisoners; while examination on April 5th, 1898 showed the reflex still lost amongst those still remaining in gaol.

The distinguishing features of Beri-beri, then, are:-

(1) Its occurrence as an endemic and epidemic disease.

(2) Its proneness to produce dropsy and cardiac disability.

(3) The non-implication of the cranial nerves, with the exception of the Vagus.

(4) The non-implication of the intellectual and emotional centres and of the rectum and bladder.

(5) The complete absence of trophic skin lesions.

(6) The high rate of mortality under certain conditions.

(7) The exceptional liability to suffer from it which is shown by individuals of good physique.

Treatment.

A. Of the epidemic:

In my Annual Report on the Gaol for 1895, I suggested, as a remedial measure, the disinfection of the soil and buildings with a strong solution of perchloride of mercury; but it was not considered

feasible at that time to adopt the suggestion.

Nothing, therefore, was done to check this epidemic until November 23rd,1897, when a Commission of Enquiry, consisting of the State Surgeon, Dr Travers, District Surgeon, Dr Lucy and myself, was appointed by the British Resident of Selangor to report on the prevalence of Beri-beri at the New Gaol, with a view to taking some decisive action to ameliorate the condition. The Commission sat on December 2nd, 1897,and subsequently sent in a full report of the origin of the disease with statistics and all circumstances possibly affecting the disease and with recommendations as to checking the epidemic.

The recommendations of the Commission were as follows:-

(1) That no earthwork of any kind be carried out in the proximity of the gaol. The Commission, being fully convinced that the poison was local and that it was to be found in the soil, laid great stress on this point.

(2) The gaol premises being infected, that the buildings be disinfected and whitewashed throughout. This was done by a solution of perchloride of mercury, 1 in 500.

(3) That the whole Gaol compound be systematically watered with the same solution of mercury.

(4) That the number of prisoners in the Gaol be reduced as far as possible by the detention of all long sentenced prisoners in the out-station lock-ups. Short-sentenced prisoners were not exposed to the prison sufficiently long enough to suffer to any great extent. This is very strikingly

shown by the following figures:- Out of 411 prisoners who contracted Beri-beri in the Gaol during the year 1896, 334 were long-sentenced prisoners, while only 77 were short-sentenced men.

(5) That the prisoners be sent as early as possible in the morning to work at a distance from the Gaol, not returning until the evening, in order to keep them as much as possible away from the infected area.

All the recommendations of the Commission were carried out in detail with marked beneficial results as the subjoined figures will show:-

Table XVI.

Month.	Prison strength.	No. of admissions into Infirmary.	No. of deaths.	Percentage of deaths.
Oct. 1897	388.21	35	8	12.5
Nov. 1897	349.08	51	16	19.2
Dec. 1897	350.01	7	nil	nil.

The recommendations of the Commission were put into force on December 10th, 1897.

On referring to Table I. it will be observed that the good effects were maintained throughout the year 1898.

In connection with the marked improvement which followed the carrying out of the recommendations of

the Commission, it must not be forgotten that all soil disturbance in and around the gaol had already ceased on November 20th 1897. I am also inclined to attribute this marked improvement to the internal administration of mercury, not only to those actually suffering from the disease but also to all prisoners who had had an attack with a view to preventing relapses or subsequent attacks. This administration of mercury internally was commenced on November 8th, 1897, and to this I also attribute the few admissions in December, 1897 and during the year 1898.

B. Of the disease:

In the early part of the epidemic symptoms only were treated, no specific being known.

Oedema and general dropsy were treated by diuretics, saline purgatives and diaphoretics, the latter including pilocarpine and vapour baths.

The chinese have a strong belief in gin and pine-apple in the treatment of this disease because of their diuretic action and frequently asked for these articles of diet which were freely given with no marked beneficial results. Nitroglycerine was found to give temporary relief in cases of urgent dyspnoea. Blistering and capping over the praecordium and venesection were all tried for urgent dyspnoea and cardiac distress with no avail.

Paracentesis of the pericardial cavity was also resorted to in a few cases, but no great amount of fluid could be removed and no relief was obtained, although at the necropsy the pericardial sac was invariably found to contain a large amount of fluid.

Various other drugs such as quinine, arsenic, iodine and special dietaries were tried with no beneficial results. Change of scene was the only efficacious means of checking the mortality until the mercurial treatment was adopted on November 8th, 1897.

Mercurial treatment:

The preparation of mercury prescribed was the Liq. Hydrarg. Perchlor., commencing with one drachm thrice daily and increasing the dose by one drachm every third day until salivation was produced. It was recommenced after all signs of salivation had ceased. The drug was given early in the disease, as soon as a prisoner complained of fulness in the stomach and inability to eat rice - the first symptom in Beri-beri. There seems to be a great tolerance of this drug in cases of Beri-beri, as much as one ounce thrice daily was administered before the physiological effects of the drug were produced.

Previous to the administration of mercury, the patients showed no signs of improvement, so long as

they remained in the infected area. The patients at this time were being transferred to the District Hospital for treatment - a distance of three miles. When mercury was given they showed rapid signs of improvement and the necessity for their removal to the District Hospital no longer existed. From November 8th, 1897, twenty four cases were treated with mercury in the New Gaol Infirmary with only one death.

None of these cases developed distressing dyspnoea which was a common symptom amongst the non-mercurial patients and which was generally a precursor of a fatal termination.

The contrast between the cheerful and contented countenance of the mercurial patient and the distressing countenance of the non-mercurial patient was most marked.

In my morning visit, on asking the patients how they were, the non-mercurial patients would say they were no better and would request a change of medicines and ask for various articles of diet; whereas the mercurial patients looked quite happy and contented, and invariably said they felt better. In fact, many of those who were not getting mercury asked for it, seeing how their fellow sufferers improved on it.

During 1898 fifty-six cases were admitted into the Gaol Infirmary, suffering from Beri-beri and all

were treated with mercury, with only two deaths. Out of the 56 cases, 20 developed the disease in the Gaol, 20 were relapses or fresh attacks and 16 were admitted with the disease already developed. Both the fatal cases occurred in prisoners who imported the disease, so that no deaths occurred amongst those who contracted the disease in gaol.

Only one out of the 56 patients developed dyspnoea and he ultimately recovered - the only case of recovery during this epidemic after this symptom had supervened.

As the administration of mercury commenced on November 8th, 1897, as all soil disturbance within and around the gaol ceased on November 20th 1897, and as the disinfection of the gaol and the other recommendations of the Commission were carried out on December 10th, 1897, it is difficult to attribute the ameliorated condition during the year 1898 to any one particular factor of the three. On the other hand, the poison was evidently strong enough to originate the disease, then how was the low case mortality in 1898 to be explained? No other drugs were used and there was no change of locality.

It has been argued against the treatment by mercury that it has been tried in the various State Hospitals with no marked beneficial results. This is true and I think may be explained by the fact that

the patients in these Hospitals only come under treatment in an advanced stage of the disease when the nerves have become so degenerated that they are beyond recovery.

In the gaol I had the opportunity of commencing treatment early and to attack the germ while still in the stomach. In the various State Hospitals the form of Beri-beri usually met with is the paralytic; the acute and dropsical forms are rarely seen. The two forms (I mean the Gaol Beri-beri and the Hospital Beri-beri) may be looked upon as two distinct diseases, and if so, must, therefore, require different treatment. They may be likened to acute alcoholism and alcoholic neuritis or to diphtheria and post-diphtheritic paralysis.

The following comparative table for the four years, 1895 to 1898 is interesting:-

Table XVII.

Years.	No. treated.	No. of deaths.	Percentage of deaths.	Remarks.
1895	152	27	17.76	Soil disturbance, all the time, on and off. No mercury given.
1896	499	47	9.41	
1897	297	55	18.51	
1898	68	2	2.94	No soil disturbance. Mercury given

Post mortem appearances presented were very uniform and consisted chiefly in infiltration of the subcutaneous connective tissue throughout the body, excess of serous fluid in the pericardial, pleural and peritoneal cavities, congestion and oedema of lungs and brain, hypertrophy and dilation of the heart, especially the right side. These were the changes mostly observed. In a few cases haemorrhagic spots in the stomach were observed.

Pathology of the disease:

I may commence this subject by saying that I do not venture any dogmatic opinion on the pathology of Beri-beri. The stress of work to which I was subjected as a Government Surgeon and general practitioner and want of adequate material prevented me from making minute microscopical examinations and electrical tests. I, therefore, base any opinion that I offer as to the pathology of the disease entirely on naked eye observations and clinical studies of the complaint during my residence in the Tropics. I am of opinion the disease is essentially a nervous one, the activity of the poison being seen in the nervous system, involving the vasomotor, sensory and motor functions. The pathological change is probably of the nature of a true Wallerian degeneration of the nerves.

A D D E N D A .

I left Selangor on furlough in May 1899 when this thesis for the most part had been written and it was my intention to have presented it in April 1900 for the M.D. degree, but I was not able to get the extension of leave I anticipated and so was not able to do so. On my return to Selangor in May 1900 I was not again placed in charge of the Gaol, so that I have no personal knowledge of the course of the disease since early in 1899. It has, however, been reported that during the year 1899 the improved health state of the Gaol was maintained, 81 cases being recorded with a death-rate of 8.64%, and that the year 1900 saw a decided rise in the Beri-beri curve, 122 cases being recorded with seven deaths. From the year 1900 to the year 1903 Dr Hamilton Wright, late Director of the Institute for Medical Research, Kwala Lumpar, Selangor, worked on Beri-beri at this same Gaol and has written valuable studies on the disease. Many of his observations confirm mine. Wright's work has added considerably to our knowledge of Beri-beri, especially with reference to the pathology of the disease. In acute Beri-beri Wright has described changes in the Nuclei of Auerbach's and Meissner's plexuses, the nerve fibres in the walls of the stomach and duodenum, the cardiac terminations of the vagi, the cells of the first and

second pairs of thoracic sympathetic ganglia and the nervi vasorum of vessels in the oedematous areas. Later, changes are described in the motor fibres to the muscles of the lower and upper limbs, in the ventral motor cells of cord, pons and bulb and sensory cells of the posterior spinal ganglia.

With regard to the causation of Beri-beri, Wright has propounded the theory that it is due to a specific organism which gains entrance to the body via the mouth; that it developes and produces a toxin chiefly in the pyloric end of the stomach and duodenum and that the toxin, being absorbed, acts atrophically on the peripheral terminations of the afferent and efferent neurones. Further that the specific organism escapes in the fæces and lodges in confined places through accident or the careless personal habits of those affected by the disorder and that, in the presence of congenial meteorological, climatic and artificial conditions of close association from overcrowding, the organism becomes virulent and gaining entrance to the healthy body in food, etc. contaminated by it, gives rise to an attack of the disease. This theory, I submit, fits in with the facts observed by me. During the years that Wright made his observations and experiments, there was no soil disturbance and Wright explains the prevalence of the disease as being due to infection -

the infection being imported into the Gaol through the admission of infected prisoners. If this is the correct explanation of the presence of the disease in the New Gaol, then how is the immunity of the healthy prisoners, transferred to the Old Gaol, although associating with infected prisoners, to be explained?

Manson's theory as to the causation of Beri-beri:

Manson has recently suggested that this disease is an intoxication produced by a toxin elaborated by a germ whose nidus is situated outside the human body.

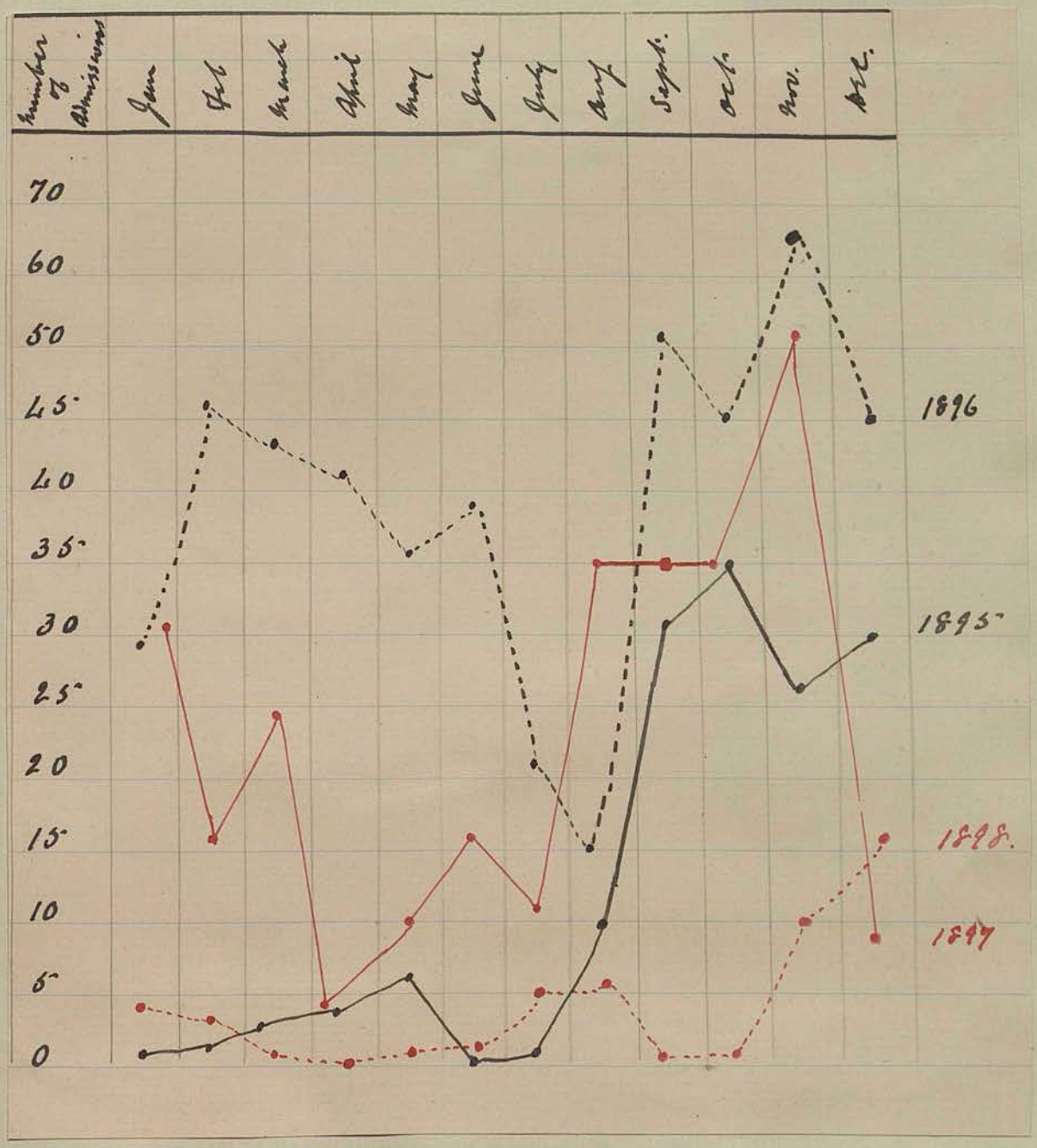
He says:- "The evidence that the multiplication of this hypothetical germ occurs outside the human body is stronger than any evidence which has been advanced in favour of its multiplication inside the human body. Did the germs multiply inside the human body we might expect that the disease it produces would run a more or less definite course as in other infections. But this is not so, for, if we remove the subject of Beri-beri from the infected area or the conditions in which he has sickened, after an interval, the progress of the disease, if it had not proved fatal in the interval, will be arrested and slow convalescence supervene. Now, if Beri-beri is caused by a germ living in the human body, it is not

likely that the germ will be killed off thus early. Beri-beri is like alcoholism, keep the drunkard off liquor and in a few days he begins to recover; give him liquor and he will not recover. Take the sufferer from Beri-beri away from the circumstances under which he is being poisoned and in a few days he begins to mend; keep him under these circumstances and he will continue sick and very likely die."

The evidence which I have brought forward with regard to the efficacy of change of locality in this outbreak corroborates what Manson says in the above quotation. Although my observations and experiments, as set forth in this thesis, may not have revealed the true cause of Beri-beri, they have, I submit, effectually set at rest the question of diet as a factor in the causation of the disease.

I attach an observation which I made in the District Hospital, Kwala Lumpur, in 1902, on the treatment of Beri-beri with arsenic.

Chart showing the number of admissions into the Infirmary from January 1895 to December 1898, month by month for each year.



Treatment of Beri-beri with Arsenic, at the District Hospital, Kwala Lumpur, Selangor, F.M.S., by A. J. McClosky, M.B.,C.M.

The suggestion that Beri-beri might possibly be a form of Arsenical poisoning induced me to try this drug on my Beri-beri patients, with a view to observing whether there would be any marked aggravation of the nerve symptoms and whether these patients would be readily influenced by this drug and exhibit some of the characteristic symptoms of poisoning other than Neuritis.

Thirty eight patients in all were treated with Arsenic and the results were not satisfactory. The cases were not specially selected. The majority of them was of the chronic paralysed type of the disease.

The duration of illness before commencement of treatment in 29 of the cases was from one month to fifteen months; in nine only was it of short duration, from four to 20 days.

The method of administration was as follows:- A start was made with a dose of three minims of Liq. Arsenicalis P.B. given thrice daily after a meal; this was increased by the addition of one minim to each dose, after every four or five days, until a

maximum dose of 10 minims was reached (i.e. 30 minims per diem). This dose was maintained unless any untoward symptoms such as Diarrhoea, supervened, when the drug was stopped and resumed later on.

Diarrhoea supervened in eight of the cases, four of whom died. Only three of these eight cases, one of whom died, had consumed more than 6 grains of arsenious acid. It is probable that the diarrhoea in these cases was intercurrent and had no relation to the administration of the drug, in as much as some developed diarrhoea after very small quantities of the drug, whereas others consumed large quantities without any bad symptoms. It must, however, be remembered that individuals vary much in their susceptibility to Arsenic.

All the cases treated were Chinese and all were "miners" (alluvial tin) with the exception of three. All were primary attacks with the exception of one.

Of the thirty eight cases treated 14 died, 20 were discharged and four absconded.

The mortality was 36.84 per cent; subtracting two deaths as due to intercurrent diarrhoea gives 31.57%.

Of the twenty patients discharged, six were cured, eight relieved and six not relieved.

Of those who absconded two were relieved and two not relieved.

Of the nine most recent cases of illness two died, five were relieved, one not relieved and one cured.

The case that recovered (Case No.27) gave a history of 15 days illness on admission; he was 96 days in Hospital and consumed in all 21 grains of arsenious acid.

The patients were considered recovered in so far as the numbness complained of had disappeared and common sensation restored, all oedema had disappeared and the paretic limbs had regained their normal functions. The knee-jerk in all the cases was still absent.

The average stay in Hospital of those who died was							47	days.
"	"	"	"	"	discharged cured	"	65	"
"	"	"	"	"	relieved	"	32	"
"	"	"	"	"	not relieved	"	12	"

None of the cases exhibited such symptoms of arsenical poisoning as Inflammation of Conjunctiva, Suffusion of eyes, lachrymation, skin lesions, gastric symptoms, falling off of hair and burning pain on the soles of the feet.

No pigmentation was observed, though this might have been overlooked in the dusky skin of the Chinaman.

The hospital diet consumed by these patients consisted of Rangoon rice, pork, salt fish, eggs and vegetables, with fresh and tinned milk occasionally.

There was no relation observed between the quantity of arsenic consumed and its effect on the patient and on the disease. Most of the fatal cases had consumed comparatively small quantities of the drug, whereas some of the non-fatal cases consumed large quantities.

The drug does not appear to be a satisfactory medicine for the treatment of Beri-beri and recovery in the six cases may be attributable to rest, good feeding, good hygienic conditions and removal of the patients from the place in which they sickened rather than to any beneficial effects exerted by arsenic.

A series of other cases treated contemporaneously with chlorine gave the following figures:-

No. treated.	Cured	Relieved	Not relieved	Died.	Mortality
45	20	17	2	6	13.33%

As in the case of those treated with arsenic the patients were not specially selected. They were all Chinese with the exception of two Malays and all were "miners" with the exception of four. Thirty seven

were primary attacks and eight were relapses. The duration of illness in twenty three of the cases was from one to eight months, in 22 from 5 to 25 days. Of the 22 most recent cases of illness three died, 12 were cured, six relieved and one not relieved.

Cases under other treatment, e.g. mercury, are not compared as they were not under special observation at the same time, and results of treatment vary so much under different atmospheric conditions.

The conclusion to be drawn from the observation of the thirty eight cases treated with arsenic is that arsenic has no curative influence on Beri-beri and that Beri-beri is not a form of arsenical poisoning, as no gastric symptoms and no skin lesions were observed in any of the cases. Moreover, if arsenic were the cause of Beri-beri it would be expected that there would be a marked aggravation of the symptoms together with skin lesions, etc.

<u>References</u>:

E.S. Reynolds, British Medical Journal, Dec.22nd, 1900.

Ronald Ross and E. S. Reynolds, Ibid. October 5th, 1901.

Table I.

38 Cases arranged according to total dose of Arsenious Acid administered.

	Cured	Relieved	Not Relieved	Died	Total
1 gr. & less	2	2	4 (1)	5	13
1 gr. - 2 grs.	-	5	2 (1)	-	7
Over 2 " - 6 "	-	-	1	6 (3)	7
- 6 " -10 "	-	2 (1)	1 (1)	-	3
- 10 " -20 "	1	1	-	1	3
- 20 " -30 "	3	-	-	1 (1)	4
- 30 " -37½ "	-	-	-	1	1
Total	6	10	8	14	38

Diarrhoea cases in brackets (-).

Table II.

Cases arranged according to number of days under treatment by Liq. Arsenicalis.

	Cured	Relieved	not Relieved	Died	Total
Up to 9 days	-	-	2	1	3
10 " 19 "	2	3	3	4	12
20 " 29 "	-	4(2)	2 (1)	1	7
30 " 39 "	-	-	-	2	2
40 " 59 "	-	1	-	3	4
60 & over	4	2	1 (1)	3	10
Total	6	10	8	14	38

Absconded in (---).

Table III.

Cases arranged according to number of days under treatment by chlorine mixture.

	Cured	Relieved	not Relieved	Died	total
Up to 9 days	-	6	-	2	8
10 - 19 "	3	2	1	-	6
20 - 29 "	3	2	-	1	6
30 - 39 "	3	3	-	3	9
40 - 59 "	4	-	1	-	5
60 & over	7	4	-	-	11
Total	20	17	2	6	45

Illustrative Cases.

Case No.28.

T.H. Admitted on 5th April, 1902. Complained of numbness and weakness of hands and legs.

Knee-jerk absent, has tenderness in the calf muscles of both legs; legs are not oedematous; there is marked paresis of the legs and he is unable to walk.

On 12th April legs became totally paralyses.

On 21st April commencing general oedema was observed.

On 24th April Diarrhoea supervened; he had been taking 7 min. Liq. Arsenicalis thrice daily since the 21st. Arsenic stopped.

On 29th April marked increase of oedema.

On 12th May diarrhoea ceased, but arsenic was not resumed.

This patient died on the 16th May and had consumed in all 2½ grs. of Arsenious Acid.

Case No.35.

T.S. Admitted on 19th February, 1902.

Complained of numbness and weakness of hands and legs. Knee-jerk absent, tenderness in calf muscles, marked paresis of legs, unable to walk, no oedema of legs.

On 3rd March oedema of legs observed.

On 14th March oedema increasing, face puffy, there was total loss of power in the legs. This case got gradually worse and on the 22nd May diarrhoea supervened and continued till his death on 29th May. He was taking 5 min. of Liq. Arsenicalis thrice daily from 26th April to 22nd May, when it was stopped. This patient consumed in all 16 grs. of Arsenious Acid.

Cause of Death - Intercurrent Diarrhoea.

Case No.36.

S.M. Admitted on 19th February, 1902.

Complained of numbness and weakness of hands and legs. Knee-jerk absent. Calves are not tender, gait ataxic, legs oedematous.

On 24th February - Numbness has disappeared from the legs, but present still in the hands.

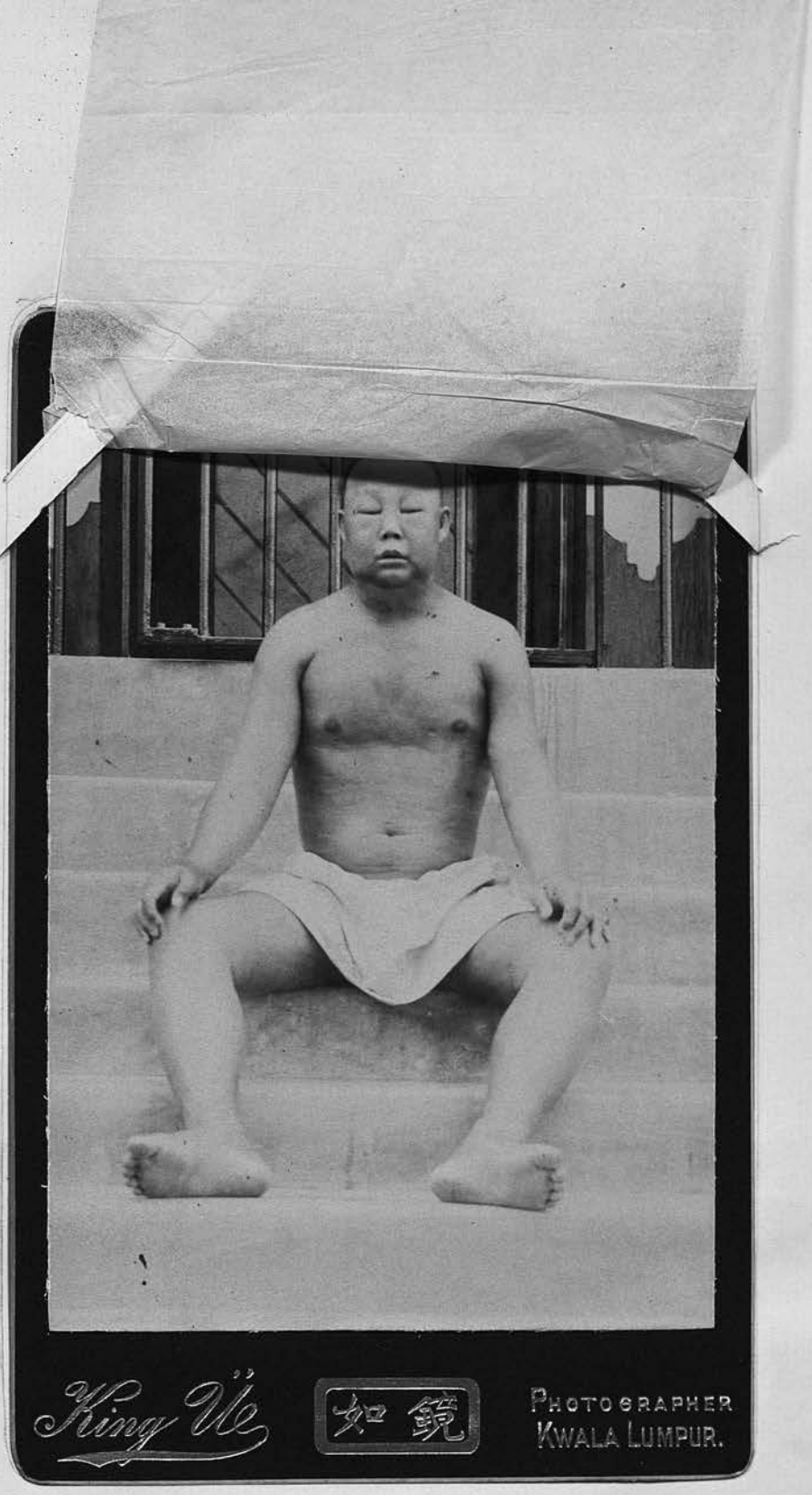
On 29th February - Numbness of hands and oedema of legs were gradually disappearing.

On 21st April Numbness of hands and oedema of legs have entirely disappeared; patient is now able to walk with a firm steady gait. Improvement was maintained and he was discharged on 30th May.

This patient consumed in all 22¼ grs. of Arsenious Acid.

Cases 28 and 35 illustrate two of the fatal cases in which diarrhoea supervened. It might be argued that the diarrhoea was brought on by the use of Arsenic, but when case No.28 is compared with case No.36 this does not seem feasible. The former developed diarrhoea after consuming in all $2\frac{1}{2}$ grs. of Arsenious Acid, whereas the latter had consumed in all $22\frac{1}{4}$ grs. of Arsenious Acid without developing Diarrhoea.

Case 36 illustrates a case of recovery after $22\frac{1}{4}$ grs. of Arsenious Acid had been consumed.


King Ue
如意
PHOTOGRAPHER
KWALA LUMPUR.

Plan of Pudu Gaol. Kuala Lumpur. Selangor.

Branch line from Railway

Reservation Ground

Cells.

A.	84	
B	87	
C	132	
D	135	(3 condemned cells)
Contr	82	(11 Refractory & 5 Crank.)
	520	
Female Ward	6.	
	526.	

Length of outer wall 700 ft.

Breadth - 500 ft.

Sentry box

W.C.

Work shop

Work shop

Work shop

Cells

C Block

B Block

A Block

D Block

Centre Block

Cells

Cells

Cells

Work shop

Cells

Hospital

Kitchen

Laundry

Tailors & shoe makers shops

Administration Block and Civil Prison

Convicts Yard Entrance

Female Ward.

Sentry box

Sentry box

Carriage Shed

To Petty Range & Rifle

Gaol Road

Dr. Alford's Quarters

Dr Bailey's Quarters

Dr Dobson

Dr Simpson

Dr Duncan's 2 other Warders Quarters.

Married Native Self Warders (Malays)

Reading Room

Dressers Quarters

Dr Dixon's Quarters.

Dr Galloway's Quarters

Single Sub Warders Quarters, (Indians)

← to Pudu

Pudu Road.