

THE SUCCESSFUL APPLICATION OF PREVENTIVE MEASURES AGAINST BERI-BERI.

By HAMILTON WRIGHT, M.D., C.M. (*McGill*),
*Late Director of the Institute for Medical Research, Federated
Malay States.*

As the result of several years of clinical and pathological experience of beri-beri in the Malay peninsula and other parts of the Far East I have been forced to the conclusion that beri-beri is an acute infectious disease with a definite primary lesion.

The reasons which led me to this conclusion were submitted to the Government of the Federated Malay States in May, 1902, the view expressed being (1) that the disease begins after an incubation period of 10—20 days with more or less marked symptoms of gastro-duodenal irritation, often diagnosed as simple indigestion: (2) that the gastro-duodenal syndrome is accompanied or soon followed by various degrees of sensori-motor and autonomic paralysis: (3) that this syndrome lasts from 20—40 days, during which time death may ensue (acute pernicious and acute beri-beri), and that otherwise the cases either recover altogether or pass into a state of residual paralysis. I have made a large number of post-mortem examinations upon beri-beri cases, and have found that only in the acute stage of the disease autopsies showed a more or less marked gastro-duodenitis, with haemorrhagic injection of the crests of the valvulae conniventes. Microscopically a varying degree of necrosis of the mucosa was discovered, accompanied by more or less marked signs of acute inflammation and in the necrosed mucosa a special bacillus of constant morphological character was found. These changes were not observed in the residual stage of the disease; and I believe that beri-beri is thus analogous to diphtheria, in that it is an acute disease with a definite local lesion, from which a toxin is disseminated throughout the body.

Although I have not actually demonstrated that the bacillus seen in the gastric and duodenal mucous membrane escapes in the intestinal discharges, I have assumed both that this is the case and that it is by this means that the spread of beri-beri takes place.

Acting on these conclusions, I made in 1902 certain recommendations with reference to the mode of life and hygienic surroundings of the prisoners in the Kuala Lumpur gaol. More than two years have elapsed since the application of the preventive measures which I advocated was begun—time enough in which to test their efficiency. I have been unable myself to watch the effect of the new *régime*, I am therefore greatly indebted to my successor, Dr C. W. Daniels, for a report of the results, from which I have quoted freely in the following account.

The various hygienic reforms which came into force between May and September, 1902, were as follows :

(1) All prisoners were employed during the greater part of the day at extramural work. From September 23rd, 1902, they were accommodated in open-sided sheds during the day, and were thus kept out of the gaol cells from 6.15 a.m. to 4.30 p.m. The cells are thus closed for only 14 hours per diem instead of 24 hours as under the old *régime*.

(2) The whole gaol was thoroughly ventilated, all the air inlets and outlets being enlarged. The cells and corridors were in consequence not only more airy but all dampness disappeared.

(3) The gaol has been thoroughly disinfected several times. In Feb. 1902 the entire building was washed with 2 p.c. formalin. Since that time each cell in order is disinfected with 1 : 1000 perchloride solution : in this way every cell is treated once in every two or three months.

(4) Under the old *régime* the prisoners defæcated and urinated in their cells. The provision for the act was most primitive. A small box of sand was provided with a few thin sticks of wood for cleansing the anus. Observation soon showed me that the sticks were of small use and that the fingers were more often employed, and afterwards wiped on the floor or on the bedding. It appeared to me that this particular fault in the personal hygiene of the prisoners was the chief factor in the spread of the disease. In consequence on my recommendation defæcation by the prisoners in their cells has been stopped.

(5) The prisoners formerly ate their meals with their fingers, in their cells. This practice furnished a ready way in which the infecting organism might reach the intestinal tract from the dust of the cell which had been contaminated by the defective personal hygiene as

related above. This source of infection has been ended in the gaol. Since May, 1902, all meals except the evening meal at 5 p.m. are partaken of outside the cells. Since April 9th, 1904, all meals have been eaten outside of the cells.

The result of these reforms is shown in the following tables. Table I. shows the admissions for beri-beri (acute and residual) to the gaol hospital

TABLE I.

1902	Cases	Deaths	1903	Cases	Deaths	1904	Cases	Deaths
			Jan.	22	1	Jan.	—	—
			Feb.	—	—	Feb.	2	—
			March	—	—	March	8	—
			April	—	—	April*	1	—
May	90	6	May	—	—	May 1st	—	—
June	83	5	June	—	—			
July	67	7	July	—	—			
Aug.	76	2	Aug.	—	—			
Sept.	131	7	Sept.	1	—			
Oct.	99	5	Oct.	1	1			
Nov.	35	2	Nov.	5	—			
Dec.	55	3	Dec.	—	—			
Totals	636	37		29	2		11	—

* There was one doubtful case of Beriberi in April.

month by month. This table includes those prisoners who, having in previous months and years contracted acute beri-beri, still remained in a state of residual paralysis, and I do not therefore attach any great significance to these figures. That the admissions of cases in this class declined from an average of 73 per month for the period from May 1902, to Jan. 1903, to practically none (average 1.2 per month) from Feb. 1903 onwards, means that they had been cured or that they had been discharged from the gaol in due course.

TABLE II. *Early Acute Cases only.*

1902	No.	1903	No.	1904	No.
		January	6	January	0
		Feb. to Aug.	0	February	0
September	28	September	1	March	3
October	32	October	1	April*	1
November	4	November	2		
December	11	December	0		
Totals	75		10		4

* Doubtful case.

Table II. illustrates a fact of transcendent importance. It is a record of those cases of acute beri-beri which actually developed in the gaol subsequent to the initiation of the hygienic measures which I have described.

These reforms were instituted between May and September, 1902, and it is fair to assume that they became effective about September. As a result the number of acute cases fell from 28 in September, 1902, to none in February, 1903, and for seven consecutive months no fresh case occurred. Towards the end of 1903 four cases occurred; of these two had been in gaol for two months, and one for 37 days. If we admit the possibility of an incubation period of two months, it is possible that these patients contracted the disease before admission to the gaol. In the first four months of 1904 only three certain cases of acute beri-beri occurred; two of them had been in gaol for seven months and one for over a year.

It is of great importance to note that the disease disappeared from the gaol not only in the months of February to September when the dry south-west monsoon blows, but also in the months between September and March when the wet or north-east monsoon prevails. It is during the latter monsoon that beri-beri becomes almost epidemic in the public institutions and mining camps of British Malaya. It was during the latter monsoon that the disease got out of control in the Kuala Lumpur gaol before the nature of the disease was recognised and before adequate preventive measures could be devised against it. So far as this gaol is concerned therefore the preventive measures which I recommended against beri-beri were effective during that part of the year most favourable to the development of the disease.

As it might be urged that the practical abolition of beri-beri from the gaol, following the application of the hygienic measures, was but part of a general diminution of the disease throughout the Malay peninsula, Dr Daniels has obtained, through the kindness of Dr McClosky, (Table III.) a record of the admissions and deaths from beri-beri at the District Hospital of Kuala Lumpur during the period under review. The patients in this hospital are drawn from the same district as were the prisoners committed to the gaol during the same period, so that the two series of figures are fairly comparable. The table shows that, while extramural beri-beri remained epidemic, the intramural disease diminished and finally disappeared.

TABLE III. *Dr McClosky's Table of Admissions and Deaths at the District Hospital, Kuala Lumpur.*

1902	Admissions	Deaths	1903	Admissions	Deaths	1904	Admissions	Deaths
			Jan.	67	16	Jan.	48	17
			Feb.	61	7	Feb.	50	11
			March	61	11	March	73	10
			April	69	12	April	70	9
May	84	23	May	67	17			
June	67	16	June	80	16			
July	67	14	July	52	12			
Aug.	56	10	Aug.	57	17			
Sept.	40	5	Sept.	51	18			
Oct.	54	12	Oct.	80	12			
Nov.	55	6	Nov.	85	30			
Dec.	67	14	Dec.	66	24			
Totals	490	102		796	192		241	47

The abolition of beri-beri in this gaol has, I consider, been attained by hygienic reforms founded on the view which I have advanced that the infective agent is contained in the excreta of the patient during the acute stage of the disease and that infection results from faecal contamination. I would suggest that, as an additional preventive measure, the stools of those suffering from acute beri-beri be disinfected.

It should not be forgotten that, from May 1st, 1902, to the present time, the prisoners have been on a diet of lower grade in the nitrogen content than obtained during the previous year or so, during which beri-beri was almost epidemic. This fact should set at rest the question of diet as a factor in the causation of beri-beri.

REFERENCES TO AUTHOR'S PREVIOUS PAPERS.

- (1) An Inquiry into the Etiology and Pathology of Beri-beri. *Studies from the Institute Medical Research, Federated Malay States*, Vol. II. No. 1, May, 1902. Singapore, Kelly and Walsh, Ltd.
- (2) Beri-beri in Monkeys. *Brain*, Winter Number 1903-4.
- (3) On the Classification and Pathology of Beri-beri. *Studies from the Institute Medical Research, Federated Malay States*, Vol. II. No. 2, December, 1903. London, Bale Sons, and Danielsson, Ltd.