

November 28: Blood appeared in the patient's stool and afterward proved constantly present by the guaiac test, and could occasionally be detected by the naked eye.

From the beginning it had been difficult to keep the man's tongue and gums clean, there being a great tendency to formation of sordes. December 26 his gums began to bleed, and, in spite of careful local treatment, in which a most competent dentist was associated, a terrific hemorrhage occurred on December 31, from which the patient did not rally, but steadily sank until his death, at 4 a. m. January 8.

There are one or two things in the course of this case that I think are worthy of emphasis. The first is that the character of the temperature curve, with the occurrence of sweats, the enlargement of spleen, the anemia and leucopenia, very strongly suggested malaria of an estivo-autumnal type. The study of the blood did not reveal the plasmodium, and the therapeutic test with quinin was a failure. From November 28, when blood first appeared in the patient's stool, calcium chlorid was administered regularly in full doses until the man died with profuse hemorrhage from the mucous membrane of the mouth. The local use of adrenalin had only a very temporary effect in the attempt to control the hemorrhage.

In the second week in December, on account of the pronounced leucopenia, yeast and, later, Vaughan's nuclein, and finally cinnamate of sodium were administered and the leucocytes continued to drop in number.

The patient complained of no pain at any time during his illness, nor was there any tenderness anywhere, except very slight tenderness over the liver. The only anatomic abnormality which could be demonstrated during life was the slight enlargement of liver and spleen. The man's mind remained clear, and he was cheerful up to ten days before his death, when the bleeding from his mouth began to be severe.

REPORT OF AUTOPSY.

The autopsy was performed seven hours after death by N. G. Russell and C. A. Bentz. Dr. Conrad Diehl was present.

External Appearance.—Usual postmortem lividity; rigor mortis, moderate; body considerably emaciated; skin, yellowish tint.

Lungs: Left lung crepitated, except at the base. The posterior part was intensely congested, somewhat friable, on section exuded considerable bloody froth. Right lung was somewhat heavier than normal, the upper and middle lobes were emphysematous. The lower lobe was large, dark in color, quite friable, did not crepitate; on section exuded bloody serum.

Heart: Small, pale; muscle was about normal thickness, $\frac{3}{4}$ inch, of a brownish-gray color, soft, friable and flabby. The muscle of the right side was thin; in some places it was almost entirely replaced by fat. The valves appeared normal.

Aorta and Blood Vessels: Moderately sclerosed.

Suprarenal Glands: Normal.

Kidneys: Left kidney was large (swollen), pale, capsule stripped easily. Cortex was somewhat narrow, the blood vessels and medullary rays were distinct. The right kidney was the same as the left. The ureters appeared normal.

Spleen: Large, soft, friable. Malpighian bodies and trabeculae were not distinct.

Gall Bladder: Small, thickened and filled with thick, dark green bile. No calculi in either ducts or bladder.

Liver: Size about normal, pale, yellowish color and firm.

Lymph Nodes: Mesenteric, peribronchial and thoracic, lymph nodes were large, reddish in color and soft.

Intestines: The lower part of the ileum and the cecum showed congestion of the mucous membrane. The solitary follicles and Peyer's patches were somewhat swollen, but not ulcerated.

Smears and cultures were made from the spleen, lymph nodes, liver and heart blood.

Pieces of tissue from the spleen, liver, kidneys, lungs, pancreas, lymph nodes, intestines and heart were preserved in hardening fluid.

Histologic Examination.—The tissues were hardened in Orth's fluid, embedded in collodion, sectioned and stained with hematoxylin and eosin. The following is a brief summary of the findings:

Lungs: Hypostatic pneumonia of the lower right lobe and posterior border of the left lung. Emphysema of the anterior and middle lobes of right lung.

Kidneys: Parenchymatous nephritis.

Heart: Fatty infiltration.

Spleen: Congested.

Liver: Fatty and cirrhotic.

Lymph Nodes: Medullary infiltration and some hyaline degeneration.

Gall Bladder: Thickened.

Bacteriologic Examination.—Cultures from the organs and tissues gave short motile bacilli, having the structural and morphologic characteristics of *Bacillus coli communis*. The organisms isolated from the blood during life (November 30, 1905) had the same cultural and morphologic characteristics as those isolated from the organs and tissues at autopsy. The agglutination (blood) test was positive with both sets of cultures.

Summary.—The man had a septicemia due to an organism belonging to the *Bacillus coli communis* group. This organism in the beginning did not have the characteristics of *Bacillus coli communis*, but under subsequent cultivation developed them. The organs and tissues presented the gross lesions of septicemia.

BERIBERI, ITS ETIOLOGY AND PREVENTION.*

LOUIS H. FALES, M.D.

MADISON, WIS.

Beriberi being a disease more particularly of the tropics and rarely seen in America, it may be well at the beginning of this paper to give a short description of the disease, so that those who are unfamiliar with its manifestations may bear in mind a picture of its principal characteristics.

PATHOLOGY.

Beriberi may be described as an acute or subacute infectious disease occurring endemically or epidemically usually in tropical countries. Its chief characteristic is a sensory-motor paralysis due to an involvement of the peripheral terminations of the sensory and motor nerves. The peripheral terminations are usually affected in the following order: The terminations of the vagus in the heart and lungs, the cardiac accelerator, those which have to do with the knee jerk, the branches of the peronei and anterior tibial, the muscles of leg and thigh, the extensors of the wrists and fingers, etc. After an incubation period of from 7 to 21 days, the patient begins to complain of malaise, loss of appetite, weakness, and pain in the lower part of the chest or the epigastrium. Vomiting may occur and sometimes diarrhea.

To a physician who is a novice as to beriberi, the foregoing symptoms would only indicate an attack of acute indigestion; to the physician who is acquainted with beriberi, and especially if the disease is prevalent, they would be very suggestive. The symptoms resembling acute indigestion are soon followed by sensations of feverishness, or chilliness, although there is no pyrexia. The uneasiness in the epigastrium soon becomes a dull heavy boring pain and is accompanied by distention of the epigastrium. The pulse becomes greatly accelerated

* Read before the Central Wisconsin Medical Association, July 31, 1906.

and if the patient exercises only slightly, dyspnea is usually observed. In from a few hours to two or three days more pronounced nervous symptoms begin to appear. The knee-jerk is found to be absent, although it is occasionally exaggerated. Patient complains that his legs feel heavy. On walking the foot flops to the ground at each step, showing that the anterior tibial and peronei have become involved. Arcs of anesthesia appear. The paralysis spreads rapidly upward to the thigh, hips, fingers, arms, etc. Edema may soon appear, beginning in the legs, depending on the implication of the vasomotor fibers of the vessels of these areas. There may be tenderness on pressure over the affected areas. The nerves of the circulatory and respiratory systems become more markedly involved. The heart becomes more irritable and palpitation occurs on the slightest exertion, with a weak and rapid pulse. The respiration becomes hurried even when at rest. Pulsation over the precordia and carotids becomes pronounced. The area of cardiac dullness increases toward the right. The cardiac impulse becomes diffuse. Signs of engorgement soon appear. Lips and finger nails become blue. There is marked dyspnea. Patient gasps for breath. The face shows great anxiety. Temperature becomes subnormal. Patient succumbs to cardiac or respiratory failure. The mind is clear to the end. This description corresponds to the usual subacute variety.

There is a pernicious form, however, the symptoms of which vary considerably from the foregoing. Loss of appetite, malaise, pain in the epigastrium, etc., may usher in the attack as in the subacute cases, but the peripheral terminations to the vagi in the stomach and heart become so rapidly involved that the patient dies often within a few hours, before other symptoms as peripheral paresis and edema appear. There are other forms of this disease, as the atrophic or paralytic, and rudimentary forms, but time will not allow a description of these in the limits of this paper. This description, however, will be sufficient to make what follows more comprehensive.

SERIOUS CHARACTER OF BERIBERI.

The importance of the prevention of beriberi, especially in the tropics, is forcibly demonstrated by the following facts:

Prior to 1884, an average of 33 per cent. of the available strength of the Japanese navy was continuously disabled on account of beriberi. During the war with China in 1894, 45 per cent. of the Japanese army were made non-efficient for the firing line on account of beriberi. Finally, in 1904 and 1905, in the Japanese army, nearly one-half of the sickness, or 24 per cent. of the entire sick and wounded, consisted of those ill with beriberi, amounting in round numbers to nearly 85,000 men.

This disease must not only be reckoned with in military movements in the tropics, but is also a factor determining to a great measure the success of many public and private undertakings. In the mines of the Federated Malay States a large percentage of the contract laborers are almost continuously disabled on account of beriberi. In Java many of the laborers are likewise disabled and in the Philippines, during the construction of the Benguet road to Baguio, the ranks of the laborers were considerably decimated on account of the same insidious disease.

THEORIES AS TO ETIOLOGY.

On looking over the voluminous literature of beriberi, I am struck with the diversity of opinions and theories advanced as to the cause of this disease and its prevention.

The most important theories promulgated as to the etiology of beriberi are:

1. That beriberi depends on the nutritive value of food.

(a) Kakaki claims to have banished beriberi from the Japanese navy by increasing the proportion of nitrogen in the ration. During a period before Kakaki's changes, about one-third of the entire strength of the navy, became affected during the year, while after the change 1/63 were affected by the disease.

(b) Several French authorities believe that beriberi is due to a scarcity of fat. It is claimed that an extensive epidemic of the disease at Chaudabun, Siam, was arrested by the increase of fat to the ration.

2. That beriberi is caused by a toxic agent taken in with the food has many advocates. Gelpke believes the toxic agent to be dried fish; Muira, raw fish; Grimm, infected fish; others that the toxin develops in moldy rice.

3. Carbon dioxid. Ashmead¹ has written several articles trying to prove that the excessive inhalation of CO₂ is the cause of beriberi.

4. That beriberi is due to an infection by protozoa. Glogner and Heanley advocate this theory. Heanley² draws an analogy between malaria and beriberi which is suggestive.

5. The arsenical poison theory is strongly advocated by Ross.³ He believes that beriberi is a neuritis caused by the ingestion of arsenic.

6. That beriberi is caused by a specific germ, probably bacterial, has many advocates. There is great diversity of opinion, however, as to how the micro-organism enters the body or whether it enters the body at all.

(a) Manson⁴ believes that the organism exists in some culture media outside the body, that the toxin which produces the disease enters the body not in the food or drink, but through the skin or by inhalation.

(b) Pekelharing and Winkler⁵ believe that a germ is introduced in the body but that the bacterium to produce the disease must be introduced by repeated infections.

(c) According to Braddon beriberi is due to the ingestion of a specific germ which develops in growing rice.

(d) Hamilton Wright⁶ maintains that the specific micro-organism is not a special habitat of any particular food, but that it may be ingested with contaminated food or drink. Having gained access by way of the alimentary canal, it becomes localized in the mucosa of the stomach and intestines. There it elaborates an extracellular toxin which, being absorbed, produces the symptoms of beriberi.

BACTERIOLOGY.

Several authorities have described a specific micro-organism as the cause of beriberi. Pekelharing and Winkler⁵ describe a white staphylococcus, which they

1. "Contribution to the Etiology of Beriberi," Univ. Med. Mag., Phila., 1892-3, v. p. 153.

2. "Some Analogies which Favor the Protozoal Hypothesis of Beriberi," Indian Med. Gaz., Calcutta, 1905, xl, pp. 212-214.

3. "Arsenic in the Hair of Beriberi Patients from Penang," Brit. Med. Jour., 1902, i, 329.

4. "Prophylaxis and Treatment of Beriberi," Brit. Med. Jour., Lond., 1902, ii, p. 830.

5. Mittheil. über d. Beriberi, Deutsch. Med. Wochschr., Berlin, 1887, xlii.

6. "Enquiry into the Etiology and Pathology of Beriberi," Jour. Trop. Med., Lond., 1905, viii, pp. 161, 180, 197, 209.

claimed to have isolated from the blood of beriberi patients. Hunter⁷ describes an organism which resembles very closely that described by Pekelharing and Winkler. Ross⁸ describes an angular diplobacillus which he claims to have isolated from the blood and cerebrospinal fluid of a large number of beriberi cases.

Okato and Kokubo have recently reported the finding of a coccus in the blood and urine of beriberi patients. Out of 129 cases examined, 65 were positive, both as to cover-glass examinations and cultures. In 34 both were negative. In 11 the microscopic examination alone was positive. In 19 the cultures were positive and the microscopic examination negative.

None of the above work has been confirmed, but the kakkeococcus of Okato and Kokubo has been reported too recently to make it certain whether or not the true micro-organism of beriberi has been discovered. Herzog,⁹ in experimenting with this coccus in Manila has, according to the last reports, come to no positive conclusions.

According to this résumé the older authorities placed great stress on food as a factor in causing beriberi while more recent writers are inclined to disregard food entirely and to consider beriberi only in the light of a disease caused by a specific micro-organism. After an extensive study of the disease in the Philippines and a careful consideration of the literature I believe that the pendulum has swung too far and that food must still be considered in determining the etiology of the disease.

How can Wright⁶ and Manson⁴ sweep aside the experience of so many able men who have suppressed beriberi by certain changes in the diet? Was Takaki's triumph in eradicating beriberi from the Japanese navy, a feat considered one of the greatest successes in preventive medicine of modern times, only a coincidence? Can Manson or Wright cite such an overwhelming success in the suppression of beriberi by carrying out their theories? Wright⁶ claims to have eradicated beriberi from the Kuala Lumpur gaol by instituting certain hygienic reforms, as the frequent disinfection of the prison, the better care of fecal discharges, better ventilation, etc., but Travers,¹⁰ writing some months after Wright's report, denies that Wright's reforms were carried out and that the suppression of beriberi was due to other causes.

BERIBERI IN BILIBID PRISON, MANILA, P. I.

This report, based on the epidemic of beriberi which occurred in Bilibid prison, Manila, during which there were 5,448 cases of the disease, is written for the purpose of showing that food as a factor can not be eliminated in determining its etiology.

Bilibid prison is the penitentiary for the entire Philippine archipelago, and also served as the city jail. At the time of which I write, the number of prisoners ranged from 1,700 to 2,000. The prisoners consisted mostly of the several Christian tribes of the islands, but the Moros, Igorrotes and Negritos were also represented. Among the prisoners were also found Spaniards, Chinese, Japanese, Indians, American negroes, Americans and Euro-

peans. The officers of the prison were a warden, two assistant wardens, and an attending physician. The warden was supreme in all matters. The physician, although having charge of the sick, had only advisory power in regard to sanitary matters, rations, etc.¹¹

Previous to the epidemic of beriberi, which started in December, 1901, there had been but few cases in the prison. For some months there had been no deaths from this cause, but the disease might be said to be endemic, for it was seldom there were not one or two cases. In December it was noticed that a great many prisoners began to complain of pain in the epigastrium, loss of appetite, and malaise. It was first thought that the new ration, which had recently been instituted, was causing indigestion. When, however, symptoms of heart and respiratory failure with paresis began to appear, the affection was soon recognized. The disease did not seem to make its appearance in any special portion of the prison, but all parts seemed to be affected simultaneously.

In regard to race, the Filipinos suffered most severely. The Chinese were almost exempt, only one or two contracting the disease, while the Americans were entirely immune.

The epidemic spread rapidly. While in December there were in the prison 52 cases of beriberi, with 2 deaths, in January there were 167 cases with 12 deaths.

To determine the cause of the epidemic and institute preventive measures was now the problem. Was the sudden appearance of this epidemic due to insanitary conditions, as overcrowding; had some new source of infection suddenly gained entrance into the prison; was it due to meteorologic conditions; had the recent change of ration been the determining cause? These questions and others presented themselves for solution.

The first measures tried for the suppression of the epidemic were along the lines of sanitation and disinfection. To begin with, all beriberi cases were, on the first manifestation of the disease, transferred to certain quarters set aside for beriberi. Overcrowding was remedied as much as the available room would permit. The entire prison was disinfected several times and certain portions many times. Platforms, floors and walls in all the buildings were scrubbed or sprayed with carbolic acid or 1/1,000 solution of bichlorid. The prisoners' blankets, mats, and everything belonging to them were soaked in a solution of 5 per cent. carbolic acid and then dried in the sun. Before returning to their quarters the clothing of the prisoners was removed, each prisoner given a full bath with 1/2,000 bichlorid solution, after which fresh clothing was provided. The buildings and quarters, clothing, bedding and prisoners were not only disinfected, but a large portion of the prison grounds was drenched with a saturated solution of chlorid of lime. To avoid possible infection of contaminated hands and dishes, immediately before eating each prisoner washed his hands and dish in water, bichlorid solution 1/1,000, and then in water. This routine of disinfection was continued several months, but in spite of it

7. Hunter, W. K.: "Bacteriology of Beriberi," Glasgow Med. Jour., 1897, xlviii, p. 116.

8. "Beriberi in the Japanese Army During the Late War. The Kakkeococcus of Okata-Kokubo," Philippine Jour. of Sci., February, 1906.

9. Wright, H.: "Successful Application of Preventive Measures Against Beriberi," Jour. Hyg., Cambridge, 1905, v, pp. 129, 133.

10. Relating to the paper entitled "The Successful Application of Preventive Measures Against Beriberi," by H. Wright, Jour. Hyg., Cambridge, 1905, v, pp. 556-559.

11. The same defects exist in regard to the powers of physicians in the Philippine Civil Service as exist in the Army. In all departments physicians have over them those higher in authority who are not physicians. The physicians may suggest, but their suggestions and recommendations are often not complied with. It was unfortunate that during the epidemic of beriberi in Bilibid Prison the physician was greatly handicapped by the fact that recommendations in regard to food and sanitation were often turned down by the warden.

and other sanitary measures employed, beriberi did not abate, and at times seemed even to increase.¹²

The result of the above experimentation shows that at least the epidemic in Bilibid was not due solely to a place infection as advocated by Manson, for: 1. The most thorough disinfection had no effect in reducing the number of cases. 2. A change in location of the prisoners was not necessary to bring the epidemic to an end. For some months previous to December, when the epidemic began to wane, there had been no disinfection.

Overcrowding had little or no effect on beriberi in Bilibid prison, for: 1. During the time of the epidemic certain buildings which were the most crowded showed no greater proportion of beriberi cases. 2. Beriberi cases did not diminish when crowding was lessened. 3. About two years after the epidemic had ceased, when the prison contained over 4,000 prisoners instead of 2,000, and was crowded far beyond its capacity, there was no increase of beriberi, although the disease was endemic at the time.

The epidemic could have been due to no new source of infection gaining entrance to the prison, for all portions seemed to simultaneously be affected, although many of the quarters were widely separated. From the chart it is seen that meteorologic conditions had little importance. Hamilton Wright's theory that the infective agent is contained in the excreta of the patients, and that the infection results from fecal contamination, would have been practically impossible during a portion of this epidemic because for several months during its height, the prisoners' hands and dishes were scrubbed and disinfected in a 1/1,000 solution of bichlorid before eating. This was done in July, August and September, months in which beriberi showed no diminution, but rather an increase.

The possibility that the epidemic might in some way be related to the food was early considered and the attending physician had several times recommended to the warden that certain changes in the ration be made. The warden did not think it possible that such a "good ration" could have anything to do with beriberi, so the request was not complied with. Careful observation, however, brought to light a few facts which increased the probability that the ration was to some degree responsible for the epidemic: 1. The entire prison ration had been changed in November. In December the epidemic began. 2. Hospital attendants did not contract the disease. 3. Prisoners who worked in the kitchen very seldom contracted beriberi. 4. During the cholera epidemic there was an increase of beriberi.

In explanation of these facts it was found that in distributing food to patients, hospital attendants often retained more than their share of the vegetables, milk, etc. The same was true of those who worked in the kitchen. They were able to select their diet to a certain extent. As the Filipino is a lover of vegetables, more of these were taken than were given out to the prisoners generally. In regard to the increase of beriberi during the cholera epidemic, it was suggestive that the vegetables had been greatly cut down at that time for fear of bringing more cholera infection into the prison.

In view of the foregoing observations it was again recommended that the ration be changed with the hope that beriberi might be eliminated. The recommendation was finally adopted the following October, and by

the last of the month the new ration was instituted. The effect of the change was almost instantaneous. When the change in ration was made the latter part of October, there were in the prison more than 100 cases of beriberi; and during that month there had been 34 deaths from the disease. By the end of December the number of cases had dropped to 14. The number of deaths had decreased to 9 in November and 3 in December. Table 1 demonstrates graphically the relations of the changes of ration to the epidemic. It is clear, therefore, that the appearance and disappearance of the epidemic were coincident with changes in the ration.

TABLE 1.—SHOWING THE MONTHLY NUMBER OF CASES OF BERIBERI WITH DEATHS IN BILIBID PRISON, MANILA, P. I.

Year.	Month.	Cases.	Deaths.
1901	November	2	0
Ration changed:			
1901	December	52	2
1902	January	169	12
1902	February	1087	16
1902	March	576	15
1902	April	327	15
1902	May	310	19
1902	June	451	17
1902	July	233	33
1902	August	571	24
1902	September	522	31
Ration again changed, October 20:			
1902	October	579	34
1902	November	476	8
1902	December	89	3
1903	January 1-15	4	0
		5448	229

After the change of ration, about Dec. 1, 1901, beriberi, which had hitherto been a negligible disease in the prison, began suddenly to increase. Again, after the ration was again changed in October, 1902, beriberi began to decline rapidly, and in a few months the mortality, which during certain months exceeded a yearly mortality of 250 per thousand, declined to almost nothing.

To study better the reason for these results the ration instituted about Dec. 1, 1901, will be compared with the ration substituted the latter part of October, 1902.

TABLE 2.—RATION INSTITUTED ABOUT DEC. 1, 1901, AND CONTINUED TO OCTOBER, 1902, WITH ITS NUTRITIVE VALUE.

Ration.	Albumi- nates.	Fats.	Starch.	Salts.
Sugar	28.35	27.35	.14
Bread	151.20	12.09	74.39	1.96
Rice	453.60	35.43	377.40	2.26
Beef	226.80	48.52	11.77	3.62
Potatoes	85.05*	1.13	.09	.50
Onions	28.35*
Pepper	.5
Vinegar	10.00
Salt	18.00	18.00
Ginger root	28.35
Total gm. . .	97.17	17.24	491.04	26.52

* Potatoes and onions have an average of 56.70.

TABLE 3.—RATION SUBSTITUTED IN OCTOBER, 1902, WITH ITS NUTRITIVE VALUE.

Ration.	Albumi- nates.	Fats.	Starch.	Salts.
Sugar	28.35	27.35	.14
Bread	302.40	24.18	148.78	3.93
Rice	255.15	17.71	18.87	1.13
Beef	226.80	48.52	11.77	3.62
Dried fish	56.70	7.08	.85	.28
Potatoes	119.07	2.38	.14	25.00
Onions	102.06	1.84	.50	5.9
Pepper	.5
Vinegar	10.00
Salt	18.00	18.00
Ginger root	28.35
Total gm. . .	101.71	19.37	395.73	29.13

12. This disinfection was not done entirely on account of beriberi, but also for the purpose of stamping out an epidemic of Asiatic cholera which had gained a foothold in the prison.

TABLE 4.—NUTRITIVE VALUE OF RATIONS IN TABLES 2 AND 3 COMPARED WITH THAT OF VOIT, WHICH HAS LONG BEEN CONSIDERED STANDARD RATION.

Ration.	Albuminates.	Fats.	Starch.	Salts.
Ration previous to October, 1902	97.17	17.24	491.04	26.62
Ration after October, 1902.	101.71	19.37	375.73	29.13
Voit's* ration reduced to Filipino weight, 125 lbs..	94.	45.	400.00	

* Voit's ration, albuminates 118 gm., fats 56 gm., starch 500 gm., is for a man weighing 151 pounds and working nine or ten hours a day. The Filipinos do not average above 125 pounds in weight, about four-fifths of the above average weight. The reduced standard is, therefore, four-fifths of Voit's original standard.

TABLE 5.—COMPARATIVE STUDY OF THE RATIONS AFTER BEING REDUCED TO NITROGEN CARBON AND HYDROGEN AND COMPARED WITH MOLESHOT'S STANDARD.

Ration.	Nitrogen.	Carbon.	Hydrogen.	Sulphur.	Salts.	Nitrogen as to Carbon.
Ration previous to October, 1902	172.1	4,166.5	61.9	13.2	140.2	1 to 24.2
Ration after October, 1902.	209.8	3,816.2	70.4	17.2	185.8	1 to 13.4
Moleshot's ration reduced to correspond to Filipino weight	256.	3,789.	143.	23.	172.	1 to 15.*

* 1 to 15 considered to be correct proportion of nitrogen to carbon.

TABLE 6.*—RATION PREVIOUS TO OCTOBER, 1902.

Name.	Grains.	Rubner's Figures.
Proteids	97.17	x 4.8 = 466.41 calories.
Fats	17.24	x 9.5 = 163.41 calories.
Carbohydrates	491.04	x 4. = 1,964.16 calories.
Total calories		2,594.35

TABLE 7.*—RATION PREVIOUS TO OCTOBER, 1902.

Name.	Grains.	Rubner's Figures.
Proteids	101.71	x 4.8 = 488.208 calories.
Fats	19.37	x 9.5 = 184.015 calories.
Carbohydrates	395.73	x 4. = 1,582.92 calories.
Total calories		2,255.143

TABLE 8.*—VOIT'S STANDARD REDUCED TO FILIPINO WEIGHT.

Name.	Grains.	Rubner's Figures.
Proteids	94	x 4.8 = 451.2 calories.
Fats	45	x 9.5 = 427.5 calories.
Carbohydrates	400	x 4. = 1,600. calories.
Total calories		2,478.7

* Tables 6, 7 and 8 show the value of the two rations in calories as compared with the ration of Voit.

FOOD AS A FACTOR IN BERIBERI.

A comparative study of the tables suggests two theories as to the cause of beriberi epidemic:

1. Previous to the change of ration in October the individual daily ration consisted of 172.1 gr. of nitrogen, and 4156.5 gr. of carbon, which is a proportion of 1 of nitrogen to 24.2 of carbon. The ration instituted in October contained 209.8 gr. of nitrogen to 3816.2 gr. of carbon, a proportion of 1 to 13.4.

2. In the ration previous to October the average daily amount of vegetable for each individual was 56.7 gm.; after October the amount was increased to 221.13 gm.

The first theory would point to nitrogen starvation as advocated by Kakaki as the cause of this particular epidemic. The second theory would place beriberi in the same category as scurvy, that is, deficiency in vegetables as the determining cause of the disease.

In 1883 the cruise of the *Ryujō*, a Japanese warship, brought the beriberi question to a crisis in Japan and led to Kakaki's investigations. In a voyage of 271 days to New Zealand and South America, stopping at the ports of Wellington, Callao, Valparaíso, and Honolulu, 160 cases of beriberi developed out of a crew of 350 men.

The year following, at the same season, Kakaki changed the ration and sent the warship *Taukuba* on the same cruise. The cruise occupied 287 days, the same ports were visited, with the same number of days' stay at each port. During this cruise only 16 cases of beriberi occurred.

The nutritive value of the ration during the first voyage was:

	Grams.
Proteids	109.29
Fats	15.8
Carbohydrates	622.32

The nutritive value of the ration during the second cruise was:

	Grams.
Proteids	196
Fats	43
Carbohydrates	775

Kakaki attributed his success to the increase in the nitrogenous constituents of the second ration over that of the first. It might be well to note that a comparative study of the supplanted diet and the diet ordered by Kakaki shows not only the increase in nitrogenous food as stated above, but also an increase in fresh vegetables. The daily amount of fresh vegetables for each man was increased from 215 gm. to 450 gm.

The question which now confronts us is: What relation has a deficiency of vegetables in the diet of beriberi?

Literature gives us but little on this question. John G. Haggard¹³ reports that in New Caledonia water-cress has been found an almost certain cure for beriberi. A study of the epidemic in Bilibid prison seems to favor the lack of fresh vegetables rather than the small proportion of nitrogenous food as the determining cause of beriberi for the following reasons:

1. Those who had access to the preparation of the vegetables and had the opportunity to take more than their share of the same had no beriberi.

2. When the vegetables were almost entirely discontinued for a time as a part of the ration, the mortality from beriberi became higher.

3. Some months after the epidemic had been under control, prisoners from the prison, many of whom had had beriberi, were sent to work on the Benguet road. So many of these were attacked by beriberi that it was found useless to try to use them, and they were returned to the prison.¹⁴ I was unable to obtain their exact ration, but was informed that they were provided scarcely any vegetables.

4. At the time of the epidemic of beriberi in the prison, many of the prisoners suffered with a peculiar form of sickness which we finally decided to be scurvy. The number of these cases exceeded 50. There was gradual loss of strength, the gums became red, spongy and swollen, there were extensive hemorrhages into the muscles, usually into the muscles of the legs. The hemorrhages would be followed by marked induration. The muscles became almost board-like in consistency, and the patient could walk only with great difficulty. On post-mortem, hemorrhages were found in various parts of the body. One case of hemopericardium was noted. On the addition of lemons to the diet of these patients, or increasing the vegetables, they recovered. There is no doubt, therefore, that with the epidemic of beriberi we

13. *Lancet*, 1904.

14. The latency of beriberi is here shown. The majority of these prisoners had had beriberi during the epidemic, which was suppressed by dietetic measures. The lack of fresh vegetables again caused the infection, which had remained latent in the body, to become active. There was comparatively little beriberi among the laborers employed on the Benguet road.

had an epidemic of scurvy, although in much smaller proportion. This, I believe, is very significant.

To recapitulate briefly, we have a change in ration, followed by epidemics of beriberi and scurvy. Certain facts, as stated above, point to a scarcity of vegetables in the ration as a possible cause of the epidemic of beriberi. The determining cause of scurvy has long been recognized to be a deficiency in vegetables. Therefore, it would seem that these two diseases are somewhat similar in their etiology.

The most important anti-scorbutic in vegetables is supposed to be potassium carbonate. A comparative estimate of the potassium carbonate in the rations made by the help of Nothnagel's tables, shows an increase of these salts from 10.34 gr. in the ration previous to the change in October to 17.39 gr. in the new ration, the ration which caused the disappearance of beriberi and scurvy. The fact noted by many writers that beriberi is very prone to occur among these people who make rice their main diet is, I think, explained by the following table:

In one ounce¹⁵ of

	Potassium Carb. Grains.
Large potatoes, boiled	1.875
Small potatoes, raw	1.310
Lime juice	.852
Lemon juice	.846
Unripe oranges	.675
Mutton (raw)	.673
Beef (raw)	.599
Corned beef (sl. salted)	.572
Peas	.529
Beef (salted)	.394
Onions	.333
Wheat bread	.258
Cheese (Dutch)	.230
Wheat flour	.100
Oatmeal	.054
Rice	.010

From this table it is very evident that when rice is made the staple food, there is little opportunity for the ingestion of potash salts. It also explains the disappearance of beriberi when a sufficient amount of vegetables, especially of potatoes, is added to the diet. Accepting the above, how can we explain the appearance of two diseases different in character, but both apparently following a deficiency of potash salts in the ration?

As already stated, beriberi is considered by many as due to a micro-organism. Indeed, it can not be doubted that the pernicious form with its rapid onset and sudden death is due to some acute infection which rapidly produces intoxication of certain vital peripheral nerves.

The acceptance of a micro-organism as its cause in no way interferes with the theory that the ration is the determining cause of the disease. The micro-organism being present, the disease is produced in an individual whose blood is deficient in potassium salts. If the normal amount of potassium salts were present in the individual the micro-organism would not find a suitable medium for its propagation, and the disease would not occur.

Much the same theory may be advanced in regard to the cause of scurvy. Babes has described a bacillus as the cause of scurvy. Rosenell subsequently described a similar bacillus. Following the capture of Port Arthur, Okado and Saito,¹⁶ studying scurvy among the Russian prisoners, described a bacillus which they recognized as the origin of scurvy. The phenomena of this disease produced in animals, by this bacillus, are similar to those of human scurvy.

Is it not, therefore, reasonable to suppose that both

in beriberi and scurvy we have a certain condition produced by a deficiency of potash salts, and that this condition causes the organism to be peculiarly susceptible to certain forms of infection? With this susceptibility, and, under certain conditions, as the presence of the proper organism, race, climate, etc., beriberi is produced. Under other conditions the result is scurvy.¹⁷

BERIBERI ON THE ISTHMUS OF PANAMA.*

IRA A. SHIMER, M.D.

Captain, Assistant Surgeon.

U. S. ARMY.

It is not the purpose of this paper to give any new information on the etiology of beriberi. The subject has been extensively investigated by Pekelharing and Winkler, Manson, Wright and the Japanese school, and germs have been isolated, which, it is claimed, are the cause of the disease. These claims, however, have not been substantiated. Beriberi has been attributed to faulty diet, to various forms of intestinal parasites, to exposure to the elements, and, finally, in absence of definite knowledge, to malaria. The consensus of opinion is that beriberi is a place infection and probably of microbic origin; it is immaterial whether, as Manson states, the germ is located in the soil of infected spots and produces a toxin which gains entrance to the body and causes the symptoms characteristic of the disease, or whether the germ itself gains access to the body and there generates a toxin which produces the disease.

The knowledge gained by the Japanese during the late war toward the prevention of beriberi by the changed dietary proves little or nothing, because, as pointed out by Scheube, late professor of the Medical School at Tokio, so many hygienic reforms were put into effect at the same time, which by themselves would tend to improve the general health. Beriberi attacks persons living in damp, ill-ventilated and overcrowded buildings; in this locality it has been especially noted that it is common among the pearl fishers. With the knowledge gained during the past few years relative to the transmission of disease by the mosquito, is it not extremely probable that this insect plays an important part in the transmission of this disease? To sum up, the causative agent of beriberi has not been determined, nor has the mode of the transmission of the infection been demonstrated.

Beriberi is a specific form of multiple peripheral neuritis, with frequent involvement of the cardiopneumogastric system, the latter manifesting itself by both functional and organic (dilatation of the right side of the heart) disturbances.

17. For other literature on the subject the reader is referred to: Banks, Charles E.: "Scurvy." Reference Handbook of Medical Sciences; Buchanan, W. J.: "Beriberi and Rice," *Lancet*, Lond., 1898, II, 577; Chittenden: "Physiologic Economy of Nutrition;" Clark, F.: "Beriberi," *Brit. Med. Jour.*, Lond., I, p. 1152; Dykes, C.: "An Outbreak of Beriberi in an Assam Jail," *Indian Med. Gaz.*, Calcutta, 1904, xxxix, pp. 201-203; Ellis, W. G.: "Contribution to the Pathology of Beriberi"; *Lancet*, Lond., xl, p. 985; MacLeod: "Beriberi and Food," *Brit. Med. Jour.*, 1897, II, p. 1459; Morris, H. C. L.: "Etiology of Beriberi," *Brit. Med. Jour.*, Lond., 1897, II, p. 500; Seaman, L. L.: "The Real Triumph of Japan," Appleton & Co., 1906; Sodre: "Twentieth Century Practice," N. Y., 1898, xiv, p. 469-524; Hunter, W. K.: "A Note on the Etiology of Beriberi," *Lancet*, Lond., 1898, I, p. 1784; Hutchinson, Robert: "Food and Principles of Dietetics"; Wright, H.: "Outline of Acute Beriberi and Its Residual Paralysis," *Rev. Neurol and Psych.*, Edin., 1905, III, pp. 645-662; Wright, H.: "Beriberi in Monkeys," *Brain*, Lond., 1903, xxvi, pp. 488 and 513; Yeo, I. B.: "Food in Health and Disease," Chicago, W. T. Keener & Co.; Reports, Weather Bureau, Manila, 1901-1902.

* Published under the Imprimatur of the American Society of Tropical Medicine.

15. Nothnagel's "Encyclopedia of Practical Medicine."

16. "First Report on the Etiologic Investigation of Scurvy," Sei-I-Kwai. *Med. Jour.*, Tokio, xxiv, No. 9, xxv, No. 1.