special and unusual groups of nerves, that difficulty arises. In these one should watch for the slightest sign of involvement of the more commonly affected nerves. The clinical course will clear up the difficulties.

PROGNOSIS

This depends on how much damage has been done, to what nerves, in what length of time, and the ability to procure the elements needed in the diet of the patient. The danger is great while the diet is defective, while the nerves to the heart and lungs show most involvement, and when the onset has been rapid. Recovery will be complete if proper diet is instituted and maintained, no vital nerve has been affected sufficiently to cause death, and no other disease carries off the patient. If former habits of diet are resumed, recurrence in aggravated form is probable.

TREATMENT

Whenever an unbalanced diet is used, an attempt should be made to regulate it better. When people depend mainly on bread, whole-wheat flour should be used. Fresh meat, vegetables and other articles should be added when possible. Such foods as beans should be used when fresh meat and vegetables can not be obtained; and attention should be given to the method of cooking so that the preventive substances are not destroyed. Canned goods probably have these destroyed by heat.

A patient with beriberi, whose symptoms have not gone so far as to be immediately dangerous, if put on a diet including fresh meat, whole-wheat bread, beans, peas, macaroni and potatoes, will, within ten days, recover from all functional symptoms. His perfect recovery will depend only on the length of time necessary for the regeneration of the nerves already affected, together with the correction of such pathologic conditions of deformity, wasting, anemia, etc., as have supervened.

Absolute rest in bed is important at first in all cases. During acute attacks, such supportive treatment as is indicated should be instituted, along with the attempt to introduce such food as will supply the lacking elements. When the stomach rebels, milk from mash-fed cows should be used. Digitalis must be used with care. Strychnin and quinin seem indicated by clinical and experimental observation (Cooper). All electric treatment and massage should be delayed until such time as progressive degeneration has ceased.

While the antineuritic substance is curative in minute quantities and has been separated, its preparation is too complicated at present for general use, and the use of proper foods is still the best way. In the severer cases, such a preparation would be life saving. It is to be hoped that in the future, it may be prepared in some stable form capable of distribution.

There is, possibly, a broader significance to the subject of deficiency neuritis than may be apparent to the average practitioner. Beriberi is thought of as a sort of extra disease that he doesn't have to bother with, a sort of tropical disease he will never see. It is easy enough to recognize in its "overforms," such as we see when a diet is obviously one sided. The vaguer symptoms and results may be present when the diet is not so considered. Meat is getting dear, however, and foods are coming to be more and more denatured by modern methods. I do not know whether affections of the nerves are correspondingly on the increase, but the text-books are significantly silent or unenlightening on the etiology of functional and degenerative diseases of the nerves. It may be that the recognition of neuritis as a deficiency disease, due to a one-sided diet in which ordinary white flour is the principle factor, may clear up some previously unexplained cases of functional nerve trouble, neuritis, and trophic disturbance.

The cause of beriberi among rice eaters is an accepted fact. It is not generally recognized among wheat eaters. Because of this fact Mr. Ohler has attempted to repeat with wheat, some of the experiments which have been accepted by the profession as proof in the case of rice. A report of these experiments will be found in the *Journal of Medical Research*.[3] Their full significance remains to be worked out.

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THE PATHOLOGIC AFFINITIES OF BERIBERI AND SCURVY

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INTRODUCTION

During a recent visit to South Africa and Rhodesia with General Gorgas, advantage was taken of the favorable opportunities there for observing scurvy. A number of post mortems were made and some pathologic material collected.

I was struck at the first necropsy by the remarkable appearance of the heart, for it suggested at once the picture seen in a well-marked case of right-sided hypertrophy and dilatation in beriberi.

Since my return, I have examined miscroscopically the vagus and its branches, as well as the heart muscle, in some of the cases, and found degenerative changes of the same type seen in beriberi.

Each necropsy presented the same cardiac lesion, and following up the observations, a number of clinical, pathologic and epidemiologic observations have been made which show family relationships between scurvy, a classical example of a food deficiency cachexia, and beriberi, and also as Watkins-Pitchford[1] pointed out in 1912 with certain other members of the group of dietetic cachexias.

At one extreme is rickets, a type of pure "osteocachexia," to use Watkins-Pitchford's term; at the other extreme a pure "neurocachexia," such as polyneuritis gallinarum. Between these two types are scurvy, infantile scurvy, the experimental scurvy of guinea-pigs, ship beriberi, beriberi (two or more types), infantile beriberi, and epidemic dropsy and neuritis.

The accompanying diagram shows qualitatively the affinities of the differnt types of food deficiency cachexias; and following is a brief diagnosis of the types:

Rickets.—A disease of infants characterized by defective nutrition of the body and diminution of lime salts in growing bones, particularly the ends of the long bones and ribs at the junction of the ribs with cartilage ("rickety rosary" and "craniotabes"). Rapidly repeated pregnancies and suckling a child during preg-

3. Jour. Med. Research, xxxi, No. 2.
1. Watkins-Pitchford: Transvaal Med. Jour., May, 1912.

nancy (according to Osler) seem to be important factors in the production of the disease. It is most common in children fed on condensed milk and food poor in fat and animal proteins (Cheadle).

Infantile Scurvy.—A cachexia of infants or children in which there is profound anemia, subperiosteal, subplural, and other hemorrhages; spontaneous separation of epiphyses from shafts of femur and tibia, and separation of ribs from cartilages, as in extreme cases of scorbutus. The gums, if the teeth have erupted, may be spongy. It is caused by proprietary foods, and there are instances, as in one of my necropsies, of its developing in breast-fed babies.[2]

Scurvy.—A disease occurring among laborers, or soldiers, in barracks and camps, or inmates of asylums, etc., and in times of famine; following the continued consumption of a one-sided or overcooked diet and one free from fresh vegetables. It is characterized by muscular joint subserous and subperiosteal hemorrhages; anemia, gingivitis, necrosis of bones, separation of costal cartilages from ribs in extreme cases, and [right-sided cardiac hypertrophy, dilatation and fatty degeneration, with degeneration of the vagus and its branches (personal observation)]. Certain epi-

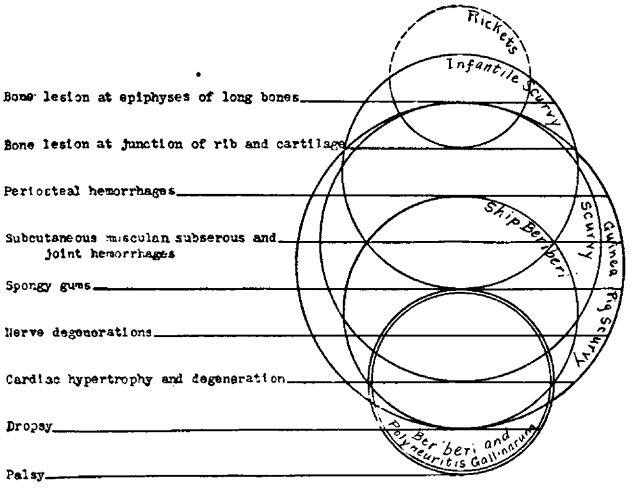

Chart showing affinities of the food deficiency syndromes.

demics display symptoms which disclose affinities with beriberi.

Experimental Scurvy of Guinea-Pigs.[3]—A disease, resembling Barlow's disease, produced in guinea-pigs by feeding a one-sided, or overcooked and unaccustomed diet of various sorts of grain, groats, and bread. There are hemorrhages, muscular, subperiosteal, and subcutaneous, often of the hind legs and ends of the ribs and between rib and cartilage, loose teeth and fragility of bone, and rarely polyneuritis and fatty degeneration of the heart. It is favorably influenced by "antiscorbutics."

Ship Beriberi.—A disease appearing usually on board sailing ships during long voyages, which may be due to eating preserved, dried, or tinned food in the absence of fresh vegetables. Symptoms are: weakness, shortness of breath and "other symptoms of weak heart causing often sudden death from acute paralysis of heart," edema of legs, and other parts of body. In a few cases sore gums, cutaneous and intramuscular hemorrhages, and peripheral neuritis are seen. It often appears in spite of the use of lime juice. Recovery is rapid when fresh food is supplied.

Beriberi.—A disease due to eating exclusively polished rice or other food from which some unidentified principle or "vitamin" is absent. It occurs endemically in certain tropical or subtropical regions and is characterized chiefly by degenerative changes in the peripheral nerves with motor and sensory disturbances, dropsy, right-sided cardiac hypertrophy and dilatation with fatty degeneration. There are undoubtedly several types of this disease such as infantile and asylum beriberi, wet and dry[4] beriberi, and that seen in Brazil (Lovelace)[5] "endemic dropsy," and endemic peripheral neuritis, etc. Dechambre described an epidemic of scurvy as occuring during the Siege of Paris, in which a few cases displayed beriberi symptoms.

Polyneuritis Gallinarum of Eijkman.—A disease produced in birds by feeding overcooked food and wheat flour, etc. Paralytic symptoms rapidly develop with subcutaneous edema, especially of the legs, and fatty degeneration of muscles of the heart; a disease resembling tropical beriberi rather than ship beriberi, neuritis being present more frequently than in ship beriberi.

Table 1 is meant to display the chief pathological features of the different diseases in this group in such manner that the affinities of the group may be seen at a glance.

Funk[6] mentions five groups of symptoms observed in diseases caused by food theoretically deficient in "vitamins":

1. Nerve degeneration with paralysis and contractures.
2. Cardiac: dilatation of right heart, dyspnea, cyanosis, oliguria.
3. Anasarca, hydropericardium, hydrothorax, ascites.
4. Scorbutus with mouth lesions, skin, subperiosteal hemorrhages and bone lesions.
5. Pellagra syndrome, stomatitis, gastro-intestinal lesions, skin erythema and multiple nerve symptoms.

While Funk may be right about the etiology of Groups 1, 2, 3, and 4, it would seem to be unwise to include pellagra in this group, for among other reasons, while pellagra is a disease "occurring in maize-eating countries *par excellence*" the eating of overcooked, undercooked and overmilled, kiln-dried maize in large quantities takes place among negro laborers and natives in South Africa to an enormous extent, for it is frequently their sole article of diet. Yet pellagra is practically unknown among that class.[7] *They do, however, suffer from scurvy.*

THE CARDIAC AND NERVE DEGENERATIONS IN BERIBERI

It it can be shown that scurvy, a typical example of a food deficiency cachexia, possesses certain pathologic features identical with those of beriberi, about which the etiology in the minds of some is as yet undetermined, it will help to establish a place for beriberi among the dietetic cachexias.

It is well known that in beriberi the heart is usually enlarged; the enlargement is especially common on the right side, the right ventricle being usually hyper-

2. It may be noted in passing that both infantile beriberi and infantile scurvy may develop in breast-fed babies.
3. Holst and Frölich: Ztschr. f. Hyg. u. Infektionskrankh., May, 1912.
4. Vedder and Clark: Philippine Jour. Sc., Sec. B, 1912, vii, 423.
5. Lovelace: Peripheral Neuritis in the Amazon Valley, Am. Jour. Trop. Dis., 1913, i, 140; The Etiology of Beriberi, THE JOURNAL A. M. A., Dec. 14, 1912, p. 2134.
6. Funk: München. med. Wchnschr., 1913, No. 47.
7. Swift and Brown: Jour. Trop. Med. and Hyg., June 1, 1914, report six cases of pellagra in eight years at Bloemfontein Asylum.

trophied and dilated, this abnormal size of the right heart being often the only striking feature of the necropsy. There is some fatty myocardial degeneration. These changes are thought to be due to degenerative changes in the vagus and its cardiac branches.

Hypertrophy and dilatation of the right heart is recognized as being due to such mechanical processes as stenosis of the pulmonary artery, reduction of the vascular area of the pulmonary artery, as in emphysema and interstitial pneumonia, valvular lesions on right side, tumors and aneurysms pressing on the pulmonary artery, and adherent pericardium.

In addition to these, this lesion is constantly found in beriberi and the Rand type of scurvy.[8] It is the type known as excentric hypertrophy or hypertrophy and dilatation characterized by thickening of the muscle and an increase in the size of the cavity. It seems to be due to a gradual strain on the right ventricle from loss of vagus innervation.

According to Scheube, Ellis found the average weight of the heart in beriberi in 125 post mortems to be about 13.37 English ounces, or 378 gm., while in 204 other cases it was but 9 ounces, or 254 gm. This increase is due chiefly to the hypertrophy of the right ventricle.

TABLE 1.—SHOWING AFFINITIES AND PATHOLOGIC FEATURES OF SCURVY, BERIBERI, ETC.

Bone lesions at epiphyses of long bones	Rickets	Infantile scurvy	*	Guinea-pig scurvy			
Bone lesion at junction of ribs and cartilage	Rickets	Infantile scurvy	Scurvy	Guinea-pig scurvy			
Subperiosteal hemorrhages		Infantile scurvy	Scurvy	Guinea-pig scurvy			
Joint, subserous, subcutaneous and muscle hemorrhages		Infantile scurvy	Scurvy	Guinea-pig scurvy	Ship beriberi		
Spongy gums		Infantile scurvy	Scurvy	Guinea-pig scurvy	Ship beriberi		
Nerve degeneration			Scurvy	Guinea-pig scurvy	Ship beriberi	Beriberi	Polyneuritis gallinarum
Cardiac hypertrophy and degeneration			Scurvy	Guinea-pig scurvy	Ship beriberi	Beriberi	Polyneuritis gallinarum
Dropsy				Guinea-pig scurvy	Ship beriberi	Beriberi	Polyneuritis gallinarum
Palsy						Beriberi	Polyneuritis gallinarum

* May occur during adolescence but probably is not encountered in adults.

Jojot[9] records ten necropsies in the cardiorespiratory form of disease (beriberi) at Camp Saint Jacques. Symptoms: dyspnea, intense cardiac erethism, extreme anxiety, and sudden death by syncope. Jojot notes the occurrence of hydropericardium, 180 c.c., and cardiac hypertrophy, 310 to 490 gm.

Pekelharing and Winkler,[10] state that "hypertrophy of the right heart is always present and associated with dilatation, sometimes small, sometimes great, and not infrequently enormous." The muscular coat of the heart is often pale. Of 64 necropsies,

5 hearts weighed 250 to 300 gm.
42 hearts weighed 300 to 350 gm.
10 hearts weighed 350 to 400 gm.
7 hearts weighed 400 to 450 gm.

according to Hamilton Wright.[11]

Observed atrophy in the terminations of the vagal inhibitory fibers in the heart muscle, axonal reaction in the cells of the ambiguous and vagal portion of the combined nuclei in the bulb, together with axonal reaction in the cells of the intrinsic cardiac neuroses, and in cells of the postganglionic accelerator fibers are conclusive evidence that almost the entire cardiac nervous system is affected by the beriberi virus in acute pernicious beriberi.

So extensive a poisoning of both inhibitory and accelerator systems accounts for a large measure of the cardiac symptoms in the latter class of cases. The loss to the heart of the control normally effected through the accelerator and inhibitory systems leaves the organ in practically the same state as the heart of a lower animal experimentally isolated from its guiding nerves.

The rapidity of beat seems to indicate that the inhibitory mechanism is first attacked. Partial exhaustion and dilatation of right heart soon follows. Arhythmia occurs. The right heart fails to empty itself and the pulmonary and venous circulation becomes surcharged. Dilatation of right side increases.

CLINICAL OBSERVATIONS ON SCURVY OF THE RAND TYPE

My first impression of the cases of scurvy in South Africa was that the disease was of an infectious nature, but that rapidly gave way to a confirmed opinion that it was due to dietary errors.

The diet of the negro laborers was certainly one-sided, considering the amount and character of the labor required of them. It consisted, as General Gorgas[12] has pointed out, too largely of carbohydrates. Five to eight feet of rock is no mean task to drill through in a day on a diet composed chiefly of corn-meal. Looking over the dietary, it was seen that the disease apparently had some relation to the following factors: Overmilled corn as the chief article of diet; overcooked corn as well as overboiled food of whatever kind; roasted meat, the natives customary way of cooking, was never supplied; and insufficient and overboiled vegetables.

Cases, too, very definitely came on during a drought and in locations where it was impossible to supply fresh vegetables and fresh meat. Several types would appear. Dr. Peall, of Arcturus, Southern Rhodesia, reported the appearance of neuritis and ataxy with enlarged right heart and edema of tibiae. Thus confirming the observations of Balfour of Khartoum and other African observers in this particular.

An interesting feature of the disease was brought out by ascertaining the incidence and death-rate among different types of natives, for during the year 1913, at one of the mines, the incidence per 1,000 per year was 12.37 among Cape Colony "boys," while it was

8. Buzzard, in Reynolds System of Medicine, 1870, Vol. I, notes cases of scurvy with fatal syncope and attributes it to the muscular structure of heart being weakened by malnutrition. Thus, cardiac syncope is also a feature common to beriberi and scurvy.
9. Jojot: Ann. d'hyg. et de méd. coloniales, 1911, No. 1, p. 88.
10. Pekelharing and Winkler: Beriberi, 1887.
11. Wright: Studies Inst. Med. Research, Federated Malay States, 1903. ii. No. 2.
12. Gorgas, Sanitation on the Rand, THE JOURNAL A. M. A., June 13, 1914, p. 1855.

7.45 among "tropicals," yet the death-rate was only 0.19 per 1,000 per annum among the former, while it was 2.66 among the tropicals, or 14 times as great. The tropical negro therefore gets scurvy in far more serious form. This may be explained from the observation that the scorbutic "tropical boy" had not yet begun to vary the monotomy of his diet by purchase of food at Kaffir stores outside the mine compound.

The clinical cases of scurvy seen varied in severity from those that had only spongy gums and very slight infiltration of calves, to others with severe and extensive hemorrhages of the muscles, joints, and serous membranes, or bone and cartilage lesions. The appearance of the patient was apathetic; many of them had been in bed six to nine weeks. Motion and locomotion were impaired, the recovering cases walked with a slight straddle, but there was no foot drop. The sick ones were either unable to stand or perhaps stood well with one leg, while the other was partly flexed, i. e., the heel elevated, the toes and ball of foot resting on the ground, apparently due to infiltration and atrophy of leg muscles. Their gums were swollen and pouting at the margins, which were bright red and fungating. On pressure, the blood spurted on the adjoining teeth. The muscles of both legs were uniformly, or unequally infiltrated and the overlying skin was smooth and shiny. After the infiltration had subsided, there was atrophy of the calves with wrinkled shiny skin. The knee-jerks in those examined were generally slightly exaggerated in all save one case. In this, the right was normal, the left practically absent. Several cases, on auscultation, disclosed tachycardia as in beriberi.

At Bulawayo, through the kindness of Dr. Eaton, a case was seen in which the gums at the time of examination were negative, the legs and tibiae were also negative, yet the sternum had collapsed, due to separation of the costal cartilages from the ends of the ribs. One could feel the cup-shaped depression in the ends of the ribs. There was also a big hematoma over the left scapula and shoulder.

Doctors at Bulawayo describe a condition known as "big leg," a condition of edema following active exertion, such as marching to the mine, occurring in some cases after three weeks in the detention camp. This is of interest, because undue exercise seems to have been an exciting or accelerating factor in precipitating attacks of beriberi and scurvy, as in soldiers after a march.

Not only has the Rand type of scurvy affiliations with beriberi, in that cardiac degeneration and degeneration of the vagus occur in typical scurvy as well as in beriberi, and by reason of the appearance of beriberi or neuritic features in certain epidemics of scurvy, but Dr. G. P. Turner of Johannesburg has observed that many of the negro miners dying of various diseases, at necropsy disclose slightly edematous calves without any other sign of scurvy or beriberi. Scurvy has definite affiliations with rickets, and infantile scurvy too, for in the case seen at Bulawayo there had been extensive destruction of the chondrocostal junction, with depression of the entire sternum.

Couvy[13] gives an interesting account of an outbreak of beriberi and of scurvy in a garrison of soldiers at Akjoucht (Mauritanie, May-September, 1908). There were 150 natives and eleven Europeans. Beriberi appeared first among the natives; later cases of scurvy appeared among them. The officers, however, during the same period, had only scurvy, with no symptoms of beriberi.

13. Couvy: Ann. d'hyg. et de méd. coloniales, 1911, No. 1, p. 97.

TABLE 2.—DIET AT OUTBREAK OF DISEASE ....

Diet of Native Soldiers	Kilos	Diet of Europeans	Kilos
Rice	0.500	Flour	0.450
Fresh meat	0.500	Fresh meat	0.500
Salt	0.015	Fat	0.020
Sugar		Coffee	0.025
		Sugar	0.030
		Salt	0.010
		Wine, dried beans and preserves	

Among those suffering from beriberi, as well as scurvy, the suppression of rice from their diet and its replacement by other articles was followed by the disappearance of cardiac troubles, asphyxia, pulmonary edema, and subcutaneous edema, but had no effect on the classical lesions of scurvy—spongy gums, muscular pain, and induration of limbs.

This instructive observation of Couvy illustrates the personal racial or idiosyncratic factor of this group of diseases, for, as far as can be ascertained, following the use of meat with one other article of food (polished rice), and associated with excessive consumption of salt[14] (saline spring water), two distinct syndromes appeared among the men who were getting the same ration (native soldiers).

This constitutional, or idiosyncratic, factor in the production of these diseases does not seem to have been sufficiently emphasized and certainly has not yet been explained. For example, a certain exclusive diet will cause scurvy in guinea-pigs, yet the same diet causes polyneuritis in pigeons. On the other hand, a deficient dietary in a tropical African negro mine laborer causes severe scurvy, in a Cape Colony African negro mine laborer, mild scurvy, and in some African negroes a diet that causes scurvy in one set of men causes neuritis in others. Furthermore, an exclusive diet of rice, which usually causes beriberi in man, has caused scurvy in certain instances, for Hölst and Frölich[15] call attention to the fact that "rice which is so often supposed to be the cause of tropical beriberi occasionally has provoked scurvy." Delpech[16] mentions in his article on scurvy in Paris, 1870-71, "a patient who got the disease who for four months had eaten meat only four times; besides this he had nothing but rice." Other scurvy patients, described by Bucquoy[17] had eaten rice or bread almost exclusively.

NOTES ON THE PATHOLOGY OF SCURVY

Three severe cases of scurvy, practically free from complication (one had a little tuberculosis), came to necropsy at one of the hospitals. The anatomic findings were closely similar and may be summarized in a composite anatomic diagnosis:

Hemorrhagic extravasation into muscles of both legs, left forearm and left psoas muscles, involving the muscle fascia between muscles, old and recent.
Subperiosteal hemorrhage, shaft of left femur.
Hemorrhage into left knee joint.
Old subcapsular hemorrhage (knee joint).
Ulcerative gingivitis with hemorrhages.
Separation of mandibular periosteum.
Anemia of all viscera.
Hypertrophy and dilatation of right heart.
Fatty degeneration of musculature of right heart.
Hyperplasia of femur marrow.
Edema of lungs.
Scaly desquamation of both legs.

14. It would be interesting to ascertain experimentally what modification in the syndromes might be induced by adding *excessive amounts of salt* to the vitamin-free diet of animals.
15. Hölst and Frölich: Jour. Hyg., 1907, vii, 667.
16. Delpech: Ann. d'hyg., 1871, xxxv, 295.
17. Bucquoy: Union méd., September-October, 1871.

This is purely a picture of scurvy, without a single beriberic feature save, of course, the cardiac change. The picture presented by the heart in these cases is very striking. It is seen to be enlarged and very pale, but as it is lifted from its bed, the pallor of a yellowish tint is sharply limited to the broad right ventricular wall. The left ventricular wall externally is of good color. This is striking, but becomes accentuated when the heart is sectioned, for the left ventricle is seen to be of normal thickness and capacity, contracted, not dilated, and the muscle of good deep color; while the right ventricular wall is definitely thickened 8 to 10 mm. more or less in thickness, and of a yellowish pallor. The chamber is dilated and the muscle stands stiffly out from the septal wall and does not collapse on it as in a normal heart. This lesion appears to be pathognomic of the Rand type of scurvy and it brings this type of the disease into relation with beriberi and other dietetic cachexias.

An examination of the cardiac muscle and of the vagus nerve and its branches (Marchi preparations) shows that there had been extensive diffuse fatty degeneration of the musculature of the right ventricle, the muscle cells being stippled over with small fat droplets. There are also small areas in which the droplets are larger. The intermuscular stroma contain many extracellular small droplets of fat. The cardiac nerves do not appear to be degenerated. The sections of the vagus at different levels all show considerable degeneration of the fibers. This is different from the finding of Pekelharing and Winkler in beriberi, who state that the nearer one approaches the periphery of the nerves, the greater the nervous degeneration; the nearer one gets to the center, the less distinct it is, and in the great nervous trunks and in the roots no changes are found in beriberi.

It may be urged by some that there is latent beriberi on the Rand and that many of the cases of scurvy overshadow the beriberic symptoms. This can definitely be ruled out, for in all the cases I examined with regard to this point the knee-reflexes were exaggerated, not absent, as in beriberi; there was no anasarca, and no paralysis or extreme general atrophy as in beriberi. Besides, the pathologic material examined was taken from cases which anatomically were uncomplicated scurvy.

Searching the available literature on scurvy for descriptions of this very striking cardiac lesion, it was seen that fatty degeneration had been described, but no one seems to have noted the peculiar features of the cardiac hypertrophy and degeneration of the vagus. This may be accounted for in the following way. From a comparative study of the pathologic features of the various dietetic cachexias it is seen that there is a relationship among them. Scurvy shades off into ship beriberi in one direction and towards rickets in an other.

All outbreaks of scurvy are not alike. Some appear in apparently well-nourished laborers of robust physique, while others appear in conditions of severe starvation as in time of famine, in campaigns, in jails, asylums, etc. In some outbreaks there are very definite beriberic features, in others they are quite absent. We may presume then, that in certain outbreaks, or in certain cases of scurvy, the cardiac hypertrophy and degeneration which I have described may be seen, in others, it may be absent. As a matter of fact, I have seen one well-marked case of asylum scurvy associated with tuberculosis in which the peculiar cardiac hypertrophy was absent. It is likely that cardiac hypertrophy may not occur in scorbutic persons debilitated by starvation and associated chronic disease. Again the type of scurvy seen among tropical negroes on the Rand and in Rhodesia appears to be different from that observed among troops, in jails, asylums, and in time of poverty and famine in that the former does not yield so readily to treatment as the latter is said to do.

CONCLUSIONS

The striking excentric hypertrophy and dilatation of the right heart with extensive fatty degeneration of the same musculature, the left heart remaining apparently normal, and the severe degeneration of the vagus nerve described here from several fatal cases of scurvy from the Rand furnish new and additional facts which show the intimate relationship between scurvy and beriberi as to etiology. The affinities between these two diseases and certain other cachexias lends emphasis to the opinion that they are all the result of the continued use of one-sided and deficient diet.

A COMPARISON BETWEEN THE UROCHROMOGEN AND DIAZO TESTS IN THE PROGNOSIS OF TUBERCULOSIS*

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As a result of personal dissatisfaction with the prognosis of tuberculosis, my attention was drawn to an abstract of an article by Moritz Weiss,[1] in which the prognostic value of Ehrlich's diazo reaction in the urine of tuberculous subjects is emphasized. He also describes a test of greater reliability and simplicity which was evolved from study of the diazo reaction. In the original article Weiss refers to his earlier writings on the subject, which date back to 1906. During seven years' observation he has confirmed the ideas of Koch and others by observing that death invariably followed the persistent presence of the diazo reaction in the urine of the tuberculous. In addition to his own work, Weiss refers to that of Dr. Heflebower, who reported similar results at the United States Government Sanatorium at Fort Bayard, N. M. Dr. Heflebower's article[2] reviews the general literature and mentions Gwerder's use of potassium hypermanganate with the diazo reagent. Heflebower, using the technic of Weiss, carefully compares his test with the diazo and demonstrates the superiority of the former.

Early in his observations, Weiss was puzzled by the occasional absence of the diazo reaction in obviously advanced cases and its frequent disappearance just before death. This led him to suspect that the unknown substance on which the diazo reaction depends was derived from an antecedent which does not give a positive reaction. On adding a 1:1,000 solution of potassium permanganate drop by drop to positive specimens and the reagent combined, he noted that the characteristic red of the reaction became brown; also, when the permanganate solution was added to positive specimens alone, a canary yellow

* Read before the Philadelphia Pathological Society, May 28, 1914.

1. Weiss, M.: Aid in Prognosis in Pulmonary Tuberculosis, Wien. klin. Wchnschr., 1913, xxvi, No. 42; abstr., THE JOURNAL A. M. A., Nov. 22, 1913, p. 1943.
2. Heflebower: Am. Jour. Med. Sc., 1912, p. 221.