

BONDURANT (E.D.)

ENDEMIC MULTIPLE NEURITIS (BERIBERI).

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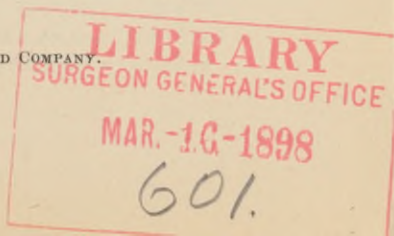
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ENDEMIC multiple neuritis, the Cingalese "beriberi," the "kakke" of Japan, while known in Asia for many centuries, and in modern times prevailing extensively among the native populations of China, India, Ceylon, Japan, the islands of the Pacific, the west coast of Africa (the "sleeping sickness"), certain portions of the coast of Brazil, and the West Indies, has been rarely seen in Europe or in North America.

The infrequency of the disease in the United States, its intrinsically interesting clinical features, and the unsolved problems of its nature and causation, give especial value to a series of seventy-one cases which during 1895-'96 occurred among the patients in the State Insane Hospital at Tuscaloosa, Alabama.

Cases of multiple neuritis had previously occurred in the institution, some six or seven cases in the decade

* Read before the Medical Association of Alabama, at Selma, April 20, 1897.



ending in 1895, but none of these were of the infectious type, two of them being due to alcohol, one to syphilis, one to pressure of a large aneurysm, one to mechanical injury during maniacal excitement, and one following a mild heat stroke.

The first case of the endemic or infectious form developed in February, 1895, in a white female patient, who had been admitted some months previous from Opelika, a small inland town in the eastern portion of the State. The case was of moderate severity, and the patient made a good recovery. The disease was not again seen until November of the same year, when almost simultaneously seven cases developed among the white female patients. During the succeeding six weeks four cases made their appearance in the white male wards, and one negro female was attacked. These thirteen cases were of similar type, although varying in severity. The nerves of the legs were chiefly involved; the subsequent atrophy in muscles was quite considerable; there was little œdema; fever and gastro-intestinal symptoms were present in many instances; only one case exhibited heart complications; several of them illustrated very well the symptomatology of the "dry" or atrophic form of beriberi.*

After a period of immunity the disease reappeared in the late summer of 1896, when, following a season of unusual dryness and distressing heat, fifty-eight patients were attacked, fifty-three of these cases occurring during September and five during October, the last of these on the 21st.

The subjoined tabular statement gives the average

* These cases were reported at the meeting of the Medical Association of Alabama, at Montgomery, in April, 1896, and published in the *Medical News* for October 3, 1896.

population of the hospital during the period covered by the outbreak, the number of cases of beriberi which occurred in each class, and the mortality.

TABLE I.

	WHITE.		NEGRO.		Total.
	Men.	Women.	Men.	Women.	
Approximate number of patients in hospital.....	460	435	140	165	1,200
Number of cases of beriberi.....	43	21	6	1	71
Fatal cases of beriberi.....	11	4	5	1	21
Number of epileptics in hospital..	45	15	15	5	80
Number of cases of beriberi in epileptics	25	5	2	0	32

It will be noted that while the negroes suffered in smaller numerical proportion than did the white patients, the disease in them assumed a severe form, six out of the seven cases terminating fatally. The negro women were almost exempt. The six cases in negro men developed among the fifty patients cared for in a detached ward near the main building of the hospital; no case occurred at the "Graystone Farm," two miles from main hospital group, where ninety negro men are colonized. In both races the men suffered more than the women. Direct contagion could not be traced.

A most remarkable feature of the disease was exhibited in its peculiar distribution among the several classes of insane patients. Every one of the seventy-one patients attacked was the subject of a psychic degenerative form of mental disorder. While some of these had previously suffered from chronic renal troubles or other impairment of health, it was, speaking generally, not the physically but the mentally enfeebled who fell victims

to the disease. There were eighty epileptics in the hospital; of these, thirty-two had beriberi. The remaining thirty-nine cases occurred in imbeciles, paranoiacs, and those terminal demented showing marked degenerative stigmata. No patient having an acute or curable form of insanity took the disease; no one of the six or seven hundred patients actively employed in work on the farm, in shops, laundry, or elsewhere, was attacked; and no case occurred among the two hundred employees of the institution.

The cases were of many varieties and every grade of severity. The common symptoms were those of neuritis—*i. e.*, muscular weakness, tenderness, pain, paræsthesiæ, loss of deep reflexes, followed by atrophy of muscles, and the electrical reaction of degeneration—accompanied by rise of temperature, gastro-intestinal disturbance, general anasarca, and tachycardia. The cases which occurred in September and October of 1896 were, as a class, more severe than were the cases seen the year previous; the neuritis was more extensive, oftentimes implicating the entire peripheral nervous system, and cardiac complications were frequent. A large proportion of them were typical examples of "beriberi hydrops," a form not seen during the first and milder outbreak.

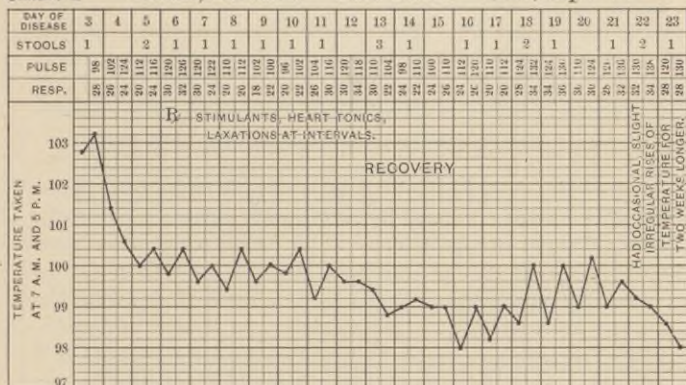
A clinical picture of the disease can best be given by a description of cases illustrating its several varieties.

CASE I.—White female, thirty-six years old, imbecile, has had epileptic convulsions since infancy, the number of seizures during the past five years averaging about ten in a month.

Early in September of 1896 she had a series of epileptic convulsions during three successive days, and after this lay in bed, stuporous, dull, at times comatose. There was some fever, bowels were constipated, tongue coated

with thick white fur, edges red, breath offensive, no desire for food, urine scanty, high colored, and albuminous. After several days it was noted that her heart action was becoming rapid, and her lower limbs, chest, and face œdematous. At this time patient was so stuporous that no satisfactory test of nerve reactions could be had, and it is impossible to date the first appearance of neuritic symptoms. The patient at the expiration of a week came out of the state of coma, and ten days after the

Chart I *Disease, Beriberi. Case No. I. Date, Sept. '96*



initial convulsions above mentioned her condition was as follows: She lies on her back, nearly helpless; moves her arms and head, but can not move her limbs or turn in bed. All tissues are œdematous, from scalp to feet inclusive; all muscles are tender to the touch and relaxed; she groans with pain when the calves of her legs are grasped in the hand. Foot-drop and wrist-drop marked. Tendon reflexes absent. Superficial reflexes retained. Tests of tactile sensation and temperature sense unsatisfactory, owing to deficiency in intelligence of patient. The œdema is great and of a peculiar brawny feel, pitting only upon firm pressure; is most pronounced

over the upper portion of the chest, shoulders, and neck, and in the lower limbs. Pulse soft and weak, 130 in a minute; heart impulse accompanied by a distinct thrill; violent pulsation of vessels in the neck. From the heart apex along the sternum, at the suprasternal notch, and over the vessels at the root of the neck, a distinct, loud, blowing systolic murmur is audible, the point of chief intensity being at the left edge of the sternum at the second and third intercostal spaces. Respiration rapid (34), but at times deep and sighing.

There had been some rise of temperature from the beginning, but the fever diminished after the first few days (see Chart I). Most of the symptoms increased in intensity, the height of the disease being attained about three weeks after its commencement, when the patient was scarcely able to execute any movement, and the entire body and limbs were exquisitely tender to touch. The plantar reflex had also disappeared, and there was some diminution in the acuteness of tactile feeling. Bowels and bladder moved involuntarily during two weeks at the height of the attack. The patient had also three epileptic convulsions during the time her heart action was rapid and œdema marked, and each time came obviously near dying, the pulse becoming almost imperceptible, cyanosis well marked, and respiration irregular and shallow. Œdema and cardiac symptoms gradually subsided together, the former more rapidly, and not perceptible after the fourth week. The heart action remained above normal rapidity for six weeks. The gastrointestinal symptoms persisted for about four weeks, and were most pronounced during the second and third, when there were obstinate constipation, tympanites, and complete anorexia. The muscular tenderness and weakness subsided by degrees, most rapidly in the arms and body. There was some—not excessive—atrophy of the muscles of the upper extremities and chest, and a higher degree of atrophy in the muscles of the legs. After two months the patient had no pain, had partially regained the use of all muscles, and was able to sit up. At the expiration

of three months she was able to stand with assistance, and in another month to walk. At the present time, six months after the onset of the disease, her lower limbs are smaller than normal and weaker, but the patient walks on a level floor without much difficulty. Superficial reflexes have returned, tendon reflexes are still absent. There is partial reaction of degeneration in the muscles of the calf of her leg and in those of the anterior tibial group.

The treatment of the case consisted in cathartics in the early stage, with opium to relieve pain; digitalis, strophanthus, and camphor when heart symptoms were most prominent. After the acute stage was passed, massage and passive exercise of limbs.

Examination of blood one week after the first appearance of the disease, and at intervals thereafter, did not discover the malarial plasmodium or other parasite. The hæmoglobin percentage was 70. There was a slight increase in the number of leucocytes, and double the usual number of eosinophile cells (noted at first examination only). The urine contained albumin and some hyaline and granular casts during the acute stage of the disease, which have persisted in diminishing quantity until now. The patient had no renal disease prior to this attack.

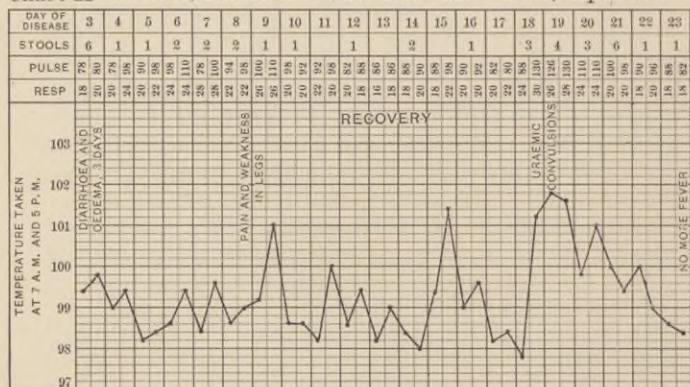
The above-cited case is a good example of the "wet" variety of beriberi, and fairly represents a large group of cases—nearly or quite half of the fifty-eight cases which occurred in 1896 being of this kind. The next case is of similar type, but terminated fatally before the nervous symptoms reached their full development. It is typical of the so-called "pernicious" form of beriberi.

CASE II.—White man, aged thirty-three years, epileptic since childhood, very weak-minded and eccentric.

He had a mild attack of beriberi in 1895, the nerves of the legs being chiefly affected, and the general systemic disorder of mild character. He recovered entirely

in a few weeks and remained well until September, 1896, when he one day complained of pain, stiffness, and weakness in his legs. The second day he was in bed suffering pain in all his limbs and in his body, with numb-

Chart II *Disease, Beriberi. Case No. VII. Date, Sept, '96*



ness and tingling in his legs, and some muscular weakness. Tendon reflexes absent; heart action rapid—100 and over; impulse distinct; thrill present. Shortness of breath, and much distress and anxiety. Slight cyanosis. Exaggerated pulsation of vessels of the neck. All heart sounds rough and loud, and at the left edge of the sternum over the pulmonary valve a long-drawn blowing diastolic murmur was heard. There was little or no œdema, but great tenderness of the calves. The third day the heart action was more rapid (140) and weaker, respiration hurried and panting, and there were dyspnoea, great distress, and restlessness, with "pain all over." A loud, blowing, systolic murmur added to the diastolic murmur audible the day before. During the day he grew steadily worse, hiccough set in, and he died seventy hours after the onset of the first symptoms, of heart failure and asphyxia.

He had no fever and no gastric disorder. Treatment consisted of laxatives in the beginning, with heart stimulants and small doses of opium.

The case next recorded shows the peripheral neuritic symptoms to better advantage, these being neither overshadowed nor masked by the anasarca, tachycardia, and systemic disorder, as is so frequent in cases of the "wet" variety.

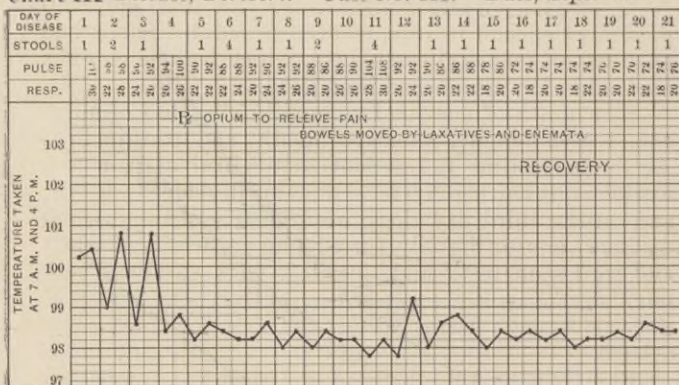
CASE III.—White woman, thirty-five years old, congenitally deficient in intelligence. In adult life she had an attack of maniacal excitement, which became chronic. General bodily health had been good.

The attack of neuritis began with fever, loss of appetite, furred tongue, offensive breath, and constipation. At the same time the patient complained of pain and weakness in her legs. The symptoms developed rapidly, and on the third day there were great gastro-intestinal disorder, nausea, heavily furred tongue, with red edges and sordes on teeth and lips; legs were paralyzed and felt "dead," as she expressed it, but pressure and passive movements were painful. There was also some œdema.

Shortly after this the pain and tenderness appeared in her arms and body, together with slight œdema in the same area, and at the same time an increase in rapidity of heart action was noted. At the height of the disease, a fortnight after its onset, the nerve symptoms were: Almost complete paralysis of the muscles of the legs, with marked foot-drop; partial paralysis of the muscles of the thighs, body, and upper extremities; hand grasp almost nil; wrist-drop marked; all muscles relaxed and flabby. All tendon reflexes abolished; plantar reflex abolished; pain "almost all over" of an aching, distressing character, worse at night; great tenderness of muscles; partial anæsthesia of the skin of the legs, loss of acuteness of tactile feeling; delay in transmission of painful im-

pressions. The patient's constant complaint was that her "legs felt dead." The pain subsided after three weeks, but tenderness on pressure persisted for several weeks longer. The œdema was general, but not excessive, and subsided after two weeks. The heart action remained rapid for six weeks, and during a portion of this time there was a distinct blowing systolic murmur at the apex and along the sternum. The vessels of the neck throbbed violently, and the pulse at wrist was soft and

Chart III Disease, Beriberi. Case No. III. Date, Sept. '96



weak. The gastro-intestinal symptoms were about what are seen in a constipated typhoid case, and persisted for five weeks. Temperature fell to normal in less than a week, however (see Chart III). There was at no time much distress in breathing, although respiration was abnormally rapid. Urine contained albumin and tube casts throughout the attack, and during the acute stage was scanty and high-colored. As œdema subsided, atrophy in the muscular tissue became noticeable and reached an extreme degree in the legs. The muscles of the body and upper extremities were also atrophied, although less than those of the legs.

Electrical examination after the acute stage showed typical reaction of degeneration in the muscles of the arms, forearms, hand, shoulder, and chest. The completely paralyzed muscles of her legs refused to respond to the faradaic current, or to the galvanic, until power of motion was partially regained, when moderately strong currents gave the usual reaction of degeneration complete.

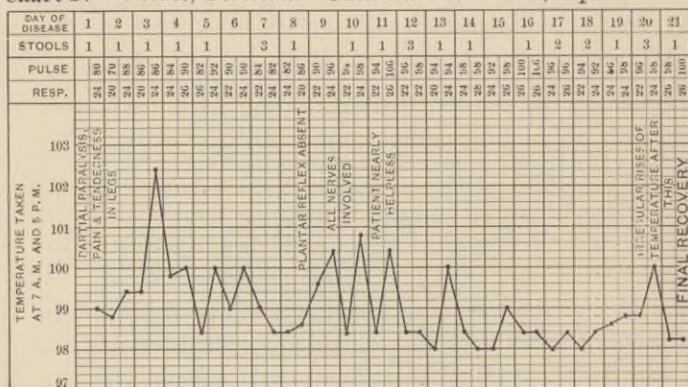
The patient remained practically helpless in bed for three months, slowly improving during the latter portion of this time. Power returned first in the muscles of her arms and body, then very gradually in those of her legs. Seven months after the attack, however, her legs are still small, weak, and the patient is unable to walk, although she stands alone. Tendon reflexes are still absent.

A striking feature of the disease was its variability in mode of onset. Some cases began suddenly with fever and gastro-intestinal irritation, as is commonly seen in the acute infections, the local neuritic symptoms appearing either simultaneously or after a few days. In other instances the onset was insidious, the initial vague aches, pains, and discomfort gradually crystallizing into the clinical picture of neuritis without fever or general systemic disorder, it being in many of these cases impossible to date the commencement of the attack. In still other cases the initial symptom was suddenly occurring dyspnoea, with tachycardia and violent pulsation of the vessels of the neck; oedema of feet and ankles was in others the first indication.

The temperature was in about half of the cases elevated in the beginning, but usually subsided to normal within a week or less. In a small proportion of the cases there was no fever for some days after the local symptoms of neuritis were well established. In a still

smaller group there were throughout the disease irregular rises of temperature, evidently due in most cases to intercurrent complications (see Charts I to V).

Chart IV *Disease, Beriberi. Case No. XI. Date, Sept. '96*



In most of the febrile cases, as well as in nearly all of those in which the neuritic symptoms were severe, there was marked disturbance of the digestive apparatus, consisting in anorexia, nausea, offensive breath, a heavily furred, raw, red-edged tongue, with, in some cases, gaseous distention of the intestines and much tenderness on pressure over the abdomen. Constipation was the rule, although in one or two cases there was diarrhœa. In some instances this gastro-intestinal disorder formed the most prominent feature of the case, quite overshadowing the local neuritis. These cases resembled a typhoid fever, minus the typhoid temperature. Where they appeared together, the fever usually subsided much sooner than did the digestive disturbance. Neither the fever nor the gastro-intestinal symptoms were seemingly

essential features of the disease, since cases exhibiting all of the milder nerve symptoms remained free from systemic disorder throughout the course of the disease.

Chart V *Disease, Beriberi. Case, No. XV. Date, Sept. '96*
H.H.—White man, at 36.



The clinical manifestations of inflammation of the peripheral nerves varied in intensity, distribution, and character, but always consisted in weakness, perversion, or abolition of function of the affected nerve trunk. In all cases the disease began in nerves of the legs, the peroneal being first and chiefly involved; and in about one fourth the neuritis was confined to the legs. In the remaining three fourths of the cases the disease extended by more or less rapid upward progression to the nerves of the trunk and the upper extremities. Some of the rapidly advancing cases presented all of the symptoms of acute ascending or Landry's paralysis. In a small proportion of the cases the entire peripheral nervous system seemed to be almost simultaneously attacked, œdema and tachycardia accompanying ("pernicious" form of beri-

beri). In a few instances the nerves of the face were implicated; in one case aphonia resulted from participation of the laryngeal branches of the vagus, and in one case there was great difficulty in swallowing liquids, probably from the same cause. In one patient the disease, beginning in the legs, eventually became more severe in the upper extremities, the man being able to walk while the arms were almost useless.

The sensory symptoms were frequently those to first attract attention—pain and tenderness in the area of distribution of the affected nerve, at first aching and not very severe, but becoming progressively more intense, and at its height very distressing. The pain was not darting nor lancinating, but boring, aching, stinging, burning, often worse at night, and usually increased by movement. Accompanying the pain were various paræsthesiæ—numbness, tingling, “creepy feelings,” sensations of “bugs crawling” on the limb, of “ants biting,” as if “legs were asleep,” as if “legs were dead,” etc. In late stages of severe cases there was diminution in acuteness of tactile perception, but in no case complete anæsthesia. In several cases distinct delay in transmission of painful impressions was noted. Tests of sense perception in the extremely demented and imbecile class with which we had to deal were often impossible, and always difficult and unsatisfactory, and no reliable data as to muscular or pressure sense were obtained. The temperature sense was not notably disordered in the few cases in which tests could be made. In mild cases and in the early stages of severe ones the skin reflexes were not abnormal; after the more severe cases were fully developed there was usually complete abolition of these reflexes in affected

portions of the body. The tendon reflexes disappeared early, and were absent throughout the course of the disease in practically every case. They did not reappear for some time after recovery was apparently complete, the patellar reflex often remaining absent for months after the patient was up and able to walk.

The motor symptoms, appearing with or shortly after the sensory disorders, were: Stiffness in muscles supplied by affected nerves, progressing through simple weakness and disinclination to exertion to some degree of paralysis, this in severe cases becoming complete. In cases in which the disease had an extensive distribution the patient was oftentimes unable to move hand or foot, or to turn in bed. The affected muscles became relaxed, soft, and mushlike, and, as above mentioned, very tender on pressure. The severity of the pain and degree of paralysis bore a close relationship, but, whereas the pains and other sensory disorders passed away after a few



FIG. 1.—Beriberi, late stage, showing atrophy of muscles of legs and foot-drop.

weeks at most, the motor paralysis was in all cases followed by a corresponding degree of atrophy in the muscles, with weakness or partial paralysis, which persisted for weeks and months. This atrophy, while imperceptible in some of the milder cases, was in the severe ones a prominent feature, the limbs being sometimes reduced to skin and bones, with a little intervening fibrous

tissue. The atrophy was most pronounced in the legs, but also, in some cases, well shown in the upper extremities, shoulders, pectoral, and other trunk muscles.



FIG. 2.—Beriberi, late stage. Extreme atrophy of leg muscles, with partial contracture.

Wrist and foot drop were typically shown. In a few of the most severe cases there was some contracture, which in two cases threatened to prove permanent

in the legs, producing a distinct talipes equinovarus in both, with, in one, stiffening of knees, due to contraction in the muscles of the posterior aspect of the thigh.

In all cases in which there was noticeable muscular atrophy the electrical reaction of degeneration was present, this consisting in diminution of faradaic excitability on both muscle and nerve, the muscles in severe cases showing no response to an interrupted current so strong as to be unbearably painful. With the galvanic current a reversal or some modification of the law of contraction was commonly obtained, the usual result with currents of moderate strength being a slow vermicular contraction, and $ACC = KCC > AOC > KOC$. In one case the following unusual result was given: $KOC = KCC = AOC = ACC$. There was no increase in galvanic excitability, as a rule, but often decided diminution of the same, with at the same time fairly well-marked DgR. These abnormalities disappeared as the muscles regained their function.

Vasomotor and trophic disorders, other than the œdema in affected parts, which is referred to below, were not frequent. In one case there was a herpetic eruption on the legs; in another, large, ulcerlike sores appeared on the legs. In some of the cases there were, after the acute stage had passed, coldness and sluggishness of circulation in the affected extremities.

Almost without exception the portions of the body supplied by affected nerves grew œdematous, this swelling, oftenest of ankles and legs, being occasionally the first symptom to attract attention. The situation in which first noticed, its degree, and the order of its appearance in different portions of the body, paralleled the de-

velopment, intensity, and progression of the neuritis—*i. e.*, it began in the feet and extended upward; it was slight, local, and transient in mild cases, was extreme and persistent in the severe ones, some of which showed great anasarca, extending from the scalp to the soles of the feet. This œdema was in some instances soft and readily pitted, in others dense and brawny. The face, neck, shoulders, and chest were often prominent situations. Effusion into pleural cavities, one or both, was noted in half a dozen cases, but fluid in the pericardial sac was not diagnosticated during life nor found in any one of the seven fatal cases subjected to post-mortem examination. In the severest cases the œdema endured for not longer than four weeks. It occurred as a consequence of disease in the vasomotor nerves rather than as a result of renal disorder, although in some cases this latter may have contributed somewhat. All the patients attacked being old residents of the hospital, the state of the renal function prior to onset of the neuritis was fairly well known, and one to a half dozen or more urinalyses, made at different times, had been recorded. Many of the patients had suffered from mild chronic forms of renal disorder, and in a small proportion of them albumin and casts existed in the urine in some quantity. In say one half of the cases—the milder half—no exacerbation of renal disease nor development of any perceptible degree of renal inadequacy was detected. In the other half—including the severe cases and those in which there were fever and gastrointestinal disturbance—there was some evidence of renal irritation or disease, shown by the appearance of albumin or increase in its amount, together with increase in number of casts. These abnormalities in the

course of a few weeks returned to their former stage, or disappeared entirely. Only one case had uræmic convulsions, and even in this one the albumin and casts disappeared from the urine after two months.

It was, as is stated above, in the cases in which nerves of trunk and upper extremities as well as those of legs were affected that this œdema was most marked, and it was in the same class of cases that implication of the respiratory and cardiac nerves occurred, practically every case in which the nerves of the body and arms were involved showing some cardiac disturbance. About forty of the cases which occurred during 1896 showed the heart symptoms very typically (*vide* Cases I and II above).

It will be noted that the chief and earliest symptom of heart implication is rapidity of action, with weakness. This begins about or shortly after the time the nerves of the body and arms become attacked—sometimes constituting an early or even the first symptom of the disease, in others coming on several days or a week or two after the first appearance of the neuritis in the legs. In the milder cardiac cases only rapidity and weakness of pulse are noted. In the more severe and typical cases the pulse-rate increases, the apex beat becomes more pronounced and is attended by a peculiar thrill. On auscultation, a loud blowing, systolic murmur is heard over the front of the chest, and widely diffused, in certain cases resembling the *bruit de diable* of exophthalmic goitre. The murmur is sometimes loudest at or near the apex; again, it is most pronounced over the pulmonary valve, at the left edge of the sternum; in a small proportion of cases a diastolic murmur coexists. At the same time violent and exaggerated pulsation of the ves-

sels of the neck forms a striking feature of many cases. Accompanying these physical signs there is usually a sense of weight or oppression in the chest, rapid, panting respiration, and dyspnœa, the symptoms being in some cases most distressing and terminating in death in a few days (as in Case II above).

The rapidity of heart action in these cases will average about 130, but may show a higher rate. In one extreme case the pulse for three weeks ranged between 180 and 210, the patient suffering surprisingly little dyspnœa most of the time, and finally making a complete recovery. In most of the cases there was moderate tachycardia, with murmurs and venous pulsation, without much dyspnœa. In the cases showing general anasarca the dyspnœa, anxiety, and mental distress were often most pronounced. Cyanosis was occasional only. Some fourteen of the twenty-one fatal cases died directly from heart failure, at times varying from three days to six weeks after the first symptoms of neuritis appeared.

Many of the patients, in particular those suffering from heart irregularity, with dyspnœa and general anasarca, were blanched and pale, presenting an appearance of great anæmia, which, however, examination of the blood in about twenty cases failed to confirm. With Fleischl hæmometer no case showed a lower percentage than sixty, and the majority ranged from seventy-five to ninety, which is fair average for supposedly healthy persons in this part of the world. The number of red corpuscles, counted by Thoma-Zeiss instrument, was also not noticeably diminished. In cover-glass preparations, taken during the acute stage, a leucocytosis was in several cases shown, and in two instances there was a marked

increase in the eosinophile cells, which increase subsided after a few days. A large number of both fresh and Chenzinsky-stained cover preparations were examined in acute and chronic stages, with especial reference to the presence of the malarial or other plasmodium; such organisms, however, were not discovered in any instance.

While some—a half score, say—of the cases were mild and of short duration, the disease, taken collectively, is the most serious, fatal, and distressing with which we have ever had to contend. Of the seventy-one patients attacked, twenty-one died, and all save ten or twelve, which may be classed as mild, were unable to walk for from two to four months, and were for weeks confined to bed with digestive and cardiac disorder. At the expiration of eight months there are, as above mentioned, still two patients unable to walk and with the danger of permanent contraction and loss of motility in legs.

In the fatal cases, one patient died of pulmonary tuberculosis, one of pneumonia, two in the *status epilepticus*, fourteen directly from heart failure, and the remaining three from a combination of causes. The complications hastened or insured a fatal termination in six or seven cases.

There is no specific treatment for the disease. The obvious indication in this as in other forms of multiple neuritis is removal of the cause, although, unfortunately, in our outbreak the causes were of a general nature and not immediately removable, so that our therapeutic efforts were chiefly directed toward the relief of the more distressing symptoms in each individual case. Cathartics, especially calomel and magnesium sulphate, gave in many cases undoubted relief, diminishing the intoxication symp-

toms as well as the œdematous effusion. For the relief of the pain morphine and coal-tar derivatives were employed, the former being the most efficacious. Hot applications occasionally gave some relief. Quinine produced no visible effect in any instance. Heart stimulants—digitalis, strophanthus, strychnine, etc.—were seemingly powerless to modify the cardiac weakness or distress, as were also the hypodermic injections of camphor used in a few cases. After the acute stage was passed tonics were used, and to aid in restoration of function to paralyzed muscles electricity and massage were given quite generally, with probably some benefit. The disease is very intractable and uncertain in course, termination, and reaction to drugs.

For orientation it should be remembered that multiple neuritis in most of its forms and varieties is a result of the action of some toxic substance upon the peripheral neurones, a long list of chemical salts, vegetable alkaloids, autogenous poisons, and bacterial toxins possessing the power of causing the disease. The symptoms of all forms of neuritis are largely those of abolition or perversion of function of the diseased nerves; and they vary with the severity of the disease and its distribution rather than with the nature of the poison, although the latter has some influence in determining the clinical features of the affection. Thus, the forms of neuritis due to alcohol, lead, syphilis, etc., are of slow and insidious onset and afebrile; the forms accompanying acute bacterial infection, on the contrary, are of rapid onset and show the fever and other systemic disturbance usual in acute germ diseases. Clinically, beriberi belongs to the last-named group; its distinguishing feature is its endemic and seeming epidemic occurrence—*i. e.*, its occur-

rence in numbers of persons about the same time in the same locality.

Entering now upon the question of the causation of beriberi, we find that much uncertainty and diversity of opinion prevail. There seems, in the mode of onset and clinical history of the disease, good ground for the growing belief that its exciting cause is a specific micro-organism.

Simmons * suggested some years ago that this germ "bears many striking resemblances in mode of production to paludal or marsh miasm, though entirely distinct and separate from it," and more recently Scheube and Glogner † publish the results of some observations leading to a similar conclusion—*i. e.*, that beriberi is a protozoal disease, resembling malaria in ætiology—and also claim the discovery of an organism in the blood which bears some resemblance to the *Plasmodium malarix*. A commission appointed by the Dutch Government to investigate the nature of the disease reported in 1886 the discovery in the blood and tissues of a micro-organism resembling that of splenic fever, and which could be cultivated outside of the body. Further reports, confirmatory or otherwise, as to the nature and causative influence of this bacterium have not come to the notice of the writer.

Among a long list of predisposing causes the following are of most importance: Insufficient, unwholesome, or unsuitable food, especially excess of carbohydrates in the dietary (rice diet); unhygienic and unsanitary surroundings and mode of life—such as dampness, defective ventilation, overcrowding, etc.—the causes, in

* Article Beriberi, in Pepper's *System of Medicine*.

† Virchow's *Archiv*, vol. cxxxii.

short, favoring the development of scurvy and other in-
nition diseases; to which may be added unfavorable cli-
matic conditions, as excessive or long-continued heat.

Turning to a consideration of the circumstances un-
der which the disease developed at this institution, and
omitting unnecessary detail, it may be stated that at the
time the outbreak occurred the food supply was amply
sufficient in quantity and fulfilled all of the requirements
of a liberal dietary; there was no excess of starchy food.
There had, during some years, been a gradual improve-
ment in the quality of the supplies purchased, and in the
cooking and serving of the food, and the dietary of
1895-'96 showed a distinct improvement over that of
any preceding year in the institution's history.

The general sanitary condition of the hospital was
also good. In common with many other State insane hos-
pitals we have suffered somewhat from overcrowding, but
this has never been excessive, and was not greater dur-
ing the year or two preceding the appearance of the
first cases than it had been during fifteen years previous;
and at the time of the second and most severe outbreak
it was less—in fact, there was no overcrowding to be
complained of; for in February, 1896, a new building,
capable of accommodating a hundred patients, was
opened, entirely relieving whatever congestion previous-
ly existed. Furthermore, in this latitude there are
comparatively few weeks in the year during which win-
dows and doors can not be kept open, insuring sufficient
ventilation. In addition, all of the patients, save those
sick in bed, work out, walk out, or sit in the yards and
airing courts daily, except when rains and cold prevent,
which is not often.

The summers in this portion of Alabama are long

and hot, and there is much warm weather in every month of the year, except about two months in winter. Most of the rainfall occurs in winter and spring, the summer and autumn being the dry season. The summers of 1895 and 1896 are commonly recognized as having been the hottest and most enervating experienced here in many years, and that of 1896 the driest, opinions fully corroborated by the weather bureau, the report of which shows that the mean temperature for the months of July, August, and September, 1895, was higher than that of a similar period during any previous year since a record has been kept, and that of 1896 a degree higher still, the figures being:

TABLE II.

Showing Mean Temperature for Months of July, August, and September, at Tuscaloosa, Ala., in Fahrenheit Degrees.

1882.....	75·6	1890.....	No record.
1883.....	No record.	1891.....	No record.
1884.....	79·7	1892.....	No record.
1885.....	78·3	1893.....	No record.
1886.....	80·0	1894.....	78·5
1887.....	No record.	1895.....	80·5
1888.....	76·4	1896.....	81·5
1889.....	76·9		

Note, however, that there is no direct relationship between excessive heat and our beriberi outbreak; the first thirteen cases appeared in February, November, and December, 1895, *not* during the summer season, and the second group of cases occurred in September and October, 1896, after the period of extreme heat had passed, although, in this instance, the depressing influence of the previous two months of intense heat probably acted as a contributory cause.

The town of Tuscaloosa is situated on the Warrior

River, two hundred miles from the Gulf of Mexico, at an elevation above the sea of about three hundred and thirty feet. The insane hospital is located two miles above the town, near the river, which forms one of the boundaries of the hospital tract. Save during periods of low water the river is navigable for small steamers as far as Tuscaloosa, but here the stream has a fall of twenty-seven feet in the two miles between the hospital and the bridge at Tuscaloosa, and above this point there are numerous "shoals," or rapids, separated by stretches of deep water in which the current is sluggish. In opening the river to navigation above Tuscaloosa three locks and the necessary dams have recently been constructed on the rapid opposite the town, converting this rapid into a series of deep, still pools, the uppermost of which extends to the next "shoal," a distance of some nine miles.

The dam which created this uppermost and longest pool was partially completed in 1894 and the level of the water above it raised a few feet; it was finished in the summer of 1895, the water level being then permanently elevated ten to twelve feet, backing up the water for long distances into the small brooks and creeks which empty into the river, killing the fringe of shrubs, trees, and other vegetation along either bank, and filling the water with dead and decaying vegetable matter.

This dam is at the lower boundary of the hospital tract; the principal group of hospital buildings is a half mile from the pool it creates. The main hospital sewer discharges its contents into the river three hundred yards *above* the dam. Three quarters of a mile above the main sewer a second small sewer empties in, carrying chiefly the waste water from the hospital laundry, but also the sewage from several water-closets. A short dis-

tance above this the water company which supplies the town of Tuscaloosa has its pump house, the suction pipe entering the river not more than one hundred feet above the sewer. There is a group of buildings at the hospital farm, above the water company's pump house; the sewage from these buildings flows in a small brook, which, after a course of three quarters of a mile, finds its way into the river on the same side as and three quarters of a mile above the pump house.

There are about fourteen hundred persons at the main hospital group, and a hundred and ten at the farm. There are, approximately, one hundred and fifty thousand gallons of sewage a day emptied into the river above the dam.

Since the erection of the dam there has been, except during a freshet or rise, no perceptible current in the river. During the unusually dry summer and autumn of 1896 the pond above the dam became almost stagnant, evaporation and the leakage through the stone dam more than counterbalancing the flow of water down stream, the result being that the water altogether ceased to run over the dam, and the level in the pond slowly fell. The water then acquired an abundant scum for some distance above the dam, and at the points of entrance of the three sewers a distinctly unpleasant odor was perceptible. It was even alleged that the water as used in Tuscaloosa had an offensive odor; but this was not noted at the hospital. The condition in the late fall of 1895 was approximately as above described, only not so bad.

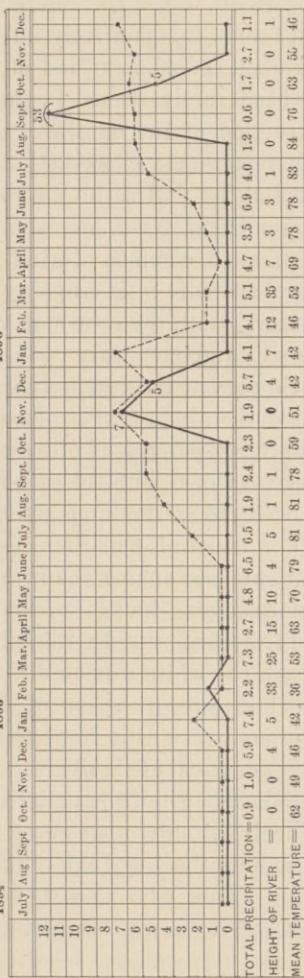
Now, from the opening of the hospital until 1895, a period of thirty-four years, the sole source of the water used in the institution was a large spring. With the gradual growth of the institution the supply of water

by degrees became insufficient, and after a dry autumn season, which probably diminished its flow somewhat, it was found necessary to supplement the spring by water purchased from the Tuscaloosa Water Company. The mains of the two systems were connected, and the water was pumped directly from the river into the hospital tank and was more or less diluted by such water as the spring continued to furnish.

This river water—the tank twice full—was first taken in January, 1895, at the close of a season of drought, the river being still near low-water mark. Our first case of beriberi occurred two or three weeks later, in February. No more river water was used until July, 1895, the river in the mean time having been continuously at from five to thirty-five feet above “low water.” After July the water was used in increasing amount during several months. The dry season coming on, the water in the river reached low-water mark in October, and remained low during November and half of December. In November and December twelve cases of endemic neuritis developed, the last one about the 1st of January, two weeks after the river had begun to rise. The river water was used now and then during the winter and spring of 1896, but the stream was well above low water most of the time. As the dry summer season came on the water was used more liberally. The river fell to low-water mark in August, and about September 1st our worst epidemic of beriberi began, the last case occurring on October 21st, although the water in the river remained low and was continuously used. For a concise statement of the facts of the case the reader is referred to Chart VI.

While some years ago malaria prevailed extensively

Chart VI.
1894



The black line shows the number of cases of beriberi. The dotted line shows the number of times the hospital tank was filled with river water (each tankful being fifty thousand gallons). Total precipitation in inches and tenths. Height of river above low-water mark (average for the month) in feet. Mean temperature in Fahrenheit degrees (the nearest whole number).

along the Warrior River, during a decade or two preceding 1895 it had been a comparatively infrequent form of disease in and about Tuscaloosa; and at the hospital especially it had been rarely seen. Beginning at the time of the construction of the dam above referred to, there was a quite remarkable exacerbation of malarial disease at the hospital, as well as in the town, cases of intermittent and remittent fever and some pernicious forms becoming only too common during the summer and fall of 1895, and sporadic cases cropping out through the succeeding winter. With the return of warm weather in 1896 there was still a greater wave of malarial disease inaugurated, which persisted through the autumn and early winter. The outbreaks of beriberi have occurred at the time or just after the malarial diseases have been at their worst, but with one doubtful exception the two diseases have not occurred in the same patient. In 1895 the majority of our cases of malaria occurred among the white male patients and among the negroes; the majority of beriberi cases among the white females, who were suffering but little from malaria. In 1896 the conditions were nearly reversed, the white women developing many cases of malaria; but only thirteen of beriberi; the white men, who suffered very little from malarial disease, furnishing forty-four cases of beriberi. The colored women, who suffered extremely from malaria during both 1895 and 1896, furnished only one case of beriberi during the two years, and the hundred colored male patients at Graystone Farm, who also were attacked by malaria both years, had no case of beriberi appear among them.

Of course, the question suggests itself, Was the epidemic one of malarial neuritis, such as is known to

occur in the island of Jamaica and some other parts of the world? This seems to us improbable. We had, as stated, much malaria at the same time; some patients had malaria, some had neuritis—no patient had both. The malarial organism was not detected in any instance, although it was especially sought for. A number of the blood-slides were also submitted to Dr. George S. Brown and Dr. Cunningham Wilson, of Birmingham, who concurred in the opinion that nothing resembling a malarial plasmodium existed in any one of them. And, lastly, quinine exerted absolutely no influence over the course of the disease.

Along with this accession of malarial disorders there was a notable increase in the amount of dysentery and diarrhoeal disease, and both at the hospital and in Tuscaloosa there was a rise in the death-rate. At the hospital the death-rate during 1896 was nearly double what it had previously been.

The seemingly pertinent circumstances under which the outbreak of beriberi occurred are given without comment, leaving the reader to formulate his own opinions as to the probable cause of the disease. Passing the facts in review, the following conclusions seem, however, not unwarranted:

1. That the dietary and the sanitary condition of the institution were not such as predispose to the occurrence of beriberi or other inanition disease.

2. That the feeble-minded and degenerate insane are especially susceptible to the disease.

3. That the sedentary life led by a portion of the patients was a further predisposing cause.

4. That the exciting cause of the disease developed in the recently dammed up river with and under the cir-

cumstances favoring the growth of the malarial germ and was transmitted in miasmatic exhalations or in the water used for drinking.

The fact that the disease did not appear in Tuscaloosa, where the river water was extensively used, is explained by the fact that there were not in Tuscaloosa a number of inactive epileptics and other degenerates, who alone supplied the material for the development of the malady. It may be assumed that the activity of the toxic agent was only sufficient to cause the disease in those most predisposed to it. Intensify the poison and other classes at the hospital and the population of Tuscaloosa might not escape.

The disease occurred at the hospital only when water taken from the river at its lowest stage was used—taken at the time the contamination from sewage and decaying vegetation was greatest. The disappearance of the disease after October 21, 1896, while the use of the water from the low river was continued, was due in all probability to the fact that all susceptible to the disease had taken it by this time; and the coming of cooler weather may also have caused a rise in vital tone and resisting power.

The only instances of the occurrence of beriberi in the United States which have found their way into medical literature are: First, sundry sporadic cases brought on shipboard from Asia and tropical America to New York, Philadelphia, San Francisco, and other seaports, reported by Seguin,* Hebersmith,† Roosevelt,‡ Boenning,§ Wertenbaken,|| and others. Second, an epi-

* *Medical News*, December 11, 1886.

† *United States Marine Hospital Report*, 1881.

‡ *Medical Record*, February 19, 1887.

§ *American Journal of the Medical Sciences*, May, 1894.

|| *United States Marine Hospital Report*, 1895.

demic among the New England fishermen of the Grand Banks, which occurred some years ago and was studied and reported by J. J. Putnam.*

A number of letters of inquiry addressed to practitioners of medicine in Alabama and other Southern States, to some of the prominent neurologists and teachers, and to the superintendents of insane hospitals in all portions of the country resulted in the discovery of one other epidemic of beriberi. This occurred at the Arkansas State Insane Hospital, Little Rock, in 1895, and having never been reported its chief features are briefly given below, and as far as possible in the words of Dr. W. B. Barner, assistant physician, who kindly wrote me an account of the outbreak:

“The cases numbered between twenty and thirty in all, and occurred during the months of July, August, and September, 1895. The prominent symptoms were malaise, with rise of temperature to 100° to 102° F. in the afternoon (a degree less in the morning), œdema of ankles, motor paralysis of legs, loss of patellar reflex, and foot-drop. In some cases the limbs were hyperæsthetic, in others partial anæsthesia was observed; some suffered much pain, others almost none. The gravity of the cases varied, the milder showing œdema of ankles with ill-defined paralytic symptoms, the severe ones complete motor paralysis of legs. Only one case proved fatal—a young negro man, already reduced to the lowest degree of health by chronic maniacal excitement.

“All except three or four of the cases occurred among the male patients. At the time, however, that this neuritis was so prevalent on the male side of the hospital,

* *Journal of Nervous and Mental Disease*, August, 1890.

almost every patient on the female side was suffering from pronounced malarial symptoms, several deaths occurring from the classic pernicious intermittent malarial fever. It was very noticeable that whereas almost all of the women suffered from malaria the men supplied nearly all of the neuritis cases. At the time of the outbreak our entire rear campus was excavated for the purpose of laying a sewer system, much damp earth being exposed to the sun's rays. There were frequent rains, followed by very hot days, and all of the conditions seemed favorable to the development of the malarial plasmodium."

It will be observed that in its general character and clinical features the outbreak was very similar to that which is made the subject of this paper, save that the cases were of milder type. Note the interchange with malaria, and compare our experience on this point as given above.

It may with reason be assumed that the above enumeration embraces all of the beriberi which has thus far occurred in this country. While rare among Europeans and their descendants in all parts of the world, the malady in epidemic form has several times appeared in Europe, the first instance being an outbreak in France in 1828, referred to by Starr,* and the last, and in this connection most interesting, the epidemics at the Richmond District Asylum, Dublin, which have been recently mentioned incidentally in the medical press, but of which no detailed account has yet appeared.

In reply to a letter of inquiry Dr. Donelan, the chief medical officer, has communicated the following facts:

* Multiple Neuritis and its Relation to Certain Peripheral Neuroses. *Medical Record*, February 5, 1887 (Middleton Goldsmith Lectures).

The first outbreak occurred in 1894, beginning early in June, no case occurring after September. About one hundred and fifty patients were attacked, and twenty-five deaths occurred. A second epidemic occurred during the summer of 1896, first appearing in July and attacking in all about a hundred patients and six nurses. A larger number of females than males have been affected.

This outbreak has quite generally been attributed to overcrowding, faulty sanitation, and dampness, conditions entirely absent at Tuscaloosa.

In view of the close ætiological and pathological relationship existing between endemic multiple neuritis and acute anterior poliomyelitis, it will be interesting to briefly consider in this connection an epidemic of the latter disease which occurred in Rutland County, Vermont, during the summer of 1894, and which was investigated by Dr. C. S. Caverly, from whose published accounts of the epidemic the following facts are taken:

The epidemic began in the latter part of June, reached its height in August, and subsided by October 1st. There were "upward of a hundred and thirty cases," almost all of them occurring in the valley of Otter Creek, "a sluggish stream, dammed at several points," and "below Rutland carrying a large amount of sewage." The majority of the cases occurred at and below Rutland. The cases were not more numerous in dwellings situated immediately on the stream, but most of them occurred near Otter Creek or its tributary brooks. The summer of 1894 was unusually hot and dry, and the water in the stream at a very low stage. Most of the cases occurred in children, although some adults were attacked. Many domestic animals—horses, dogs, fowls—were also affected.

The chief symptoms were motor paralysis in arms or legs, with minor sensory disorders and much cerebral disturbance, leading in some cases to the diagnosis "meningitis." Dr. Starr, Dr. Dana, and Dr. Jacoby concurred in the opinion that the disease was acute anterior poliomyelitis, although many of the cases were recognized as atypic. In reply to my inquiry as to whether it was possible that there might have been cases of neuritis interspersed among those of poliomyelitis, Dr. Caverly writes: "There is no doubt some of the cases in our epidemic of 1894 were neuritis, but, occurring with so many of undoubted poliomyelitis, there is no doubt the essential disease was of the latter character."

Some of the circumstances attending this epidemic of poliomyelitis in Vermont are very similar to those prevailing at this hospital during the last epidemic of beriberi—the proximity of a sluggish stream, dammed at several points and containing sewage, together with unusual heat and dryness.

A somewhat similar outbreak of poliomyelitis on a smaller scale occurred during the summer of 1896 in Greene County, Alabama, about forty miles south of Tuscaloosa. I owe to Dr. G. A. Moore, of Clinton, most of the information obtained concerning the cases, which were fifteen or more in number, scattered over an area of country ten to fifteen miles in diameter. Adults as well as children were attacked; the disease began with fever and general systemic disorder, which was generally diagnosed as "malaria," there being much malarial disease in the community at the same time. A few days to a week or two after the fever appeared some paralysis developed, involving sometimes one upper or one lower extremity, sometimes both extremities on one side, some-

times both upper or both lower extremities, and in one or two instances both lower and one upper extremity. Most of the patients recovered entirely and all improved, but in some there is still, seven to nine months after attack, some paralysis and muscular atrophy remaining. Dr. Moore mentions that some of the negroes who had the disease thought they were "conjured," and refused to submit to medical treatment. One of the cases, a white boy, twelve years old, was brought to me for consultation and diagnosis, giving opportunity for a thorough examination, which left no doubt that the disease in this case was acute anterior poliomyelitis. The histories of the other cases furnished me by Dr. Moore point conclusively to poliomyelitis as the correct diagnosis of them all.

Two or three cases of infantile paralysis have occurred in and near Tuscaloosa during the past twelve months, but there has been nothing approaching an epidemic of the disease.

Of our fatal cases of beriberi seven were brought to the autopsy table. The post-mortem appearances and the results of subsequent study of the tissues will be made the subject of a future report.

Suffice it in this connection to say that in at least one case there was evident degenerative change in the motor cell bodies in the anterior gray horn of the cord; that in no case were there redness, hæmorrhages into, or gross inflammatory changes in the nerve trunks, the lesion, so far as yet determined, being a degeneration in the nerve filaments themselves, and there were the usual degenerative changes in the muscles supplied by the affected nerves, including the heart muscle in those cases in which the vagus participated in the lesion.

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