BONDURANT (E.D.)

REPORT OF THIRTEEN CASES OF MULTIPLE NEURITIS OCCURRING AMONG INSANE PATIENTS.

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REPORT OF THIRTEEN CASES OF MULTIPLE NEURITIS OCCURRING AMONG INSANE PATIENTS.[1]

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THE inflammatory-degenerative changes, which, occurring in the peripheral nerves, are generally described under the name of "neuritis," may be especially active in the nerve-sheaths and connective-tissue framework—"interstitial neuritis,"—or have their chief location in the nerve-fibers—"parenchymatous neuritis"—or, as is most often the case, may involve both the neural and connective-tissue elements—"diffuse neuritis." The causes of neuritis are many and varied, and, used as a basis of classification, give rise to a number of more or less easily recognizable clinical varieties of the disease, the most important being:

(1) The neuritis occurring as a result of direct injury to the nerve-trunk—wounds, blows, pressure, as from sleeping on the arm, from dislocation of bones, from tumors. (2) That form resulting from exposure to cold. When the trunk of the seventh cranial nerve is involved, as is often the case, we have here a familiar form of facial paralysis. (3) The forms resulting from direct extension to adjacent nerves of the infection of bacterial diseases, as pneumonia, diphtheria, tuberculosis. (4) Those forms due to the presence in

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the blood of the poisons of these infectious diseases, especially syphilis, diphtheria, typhoid fever, malaria, variola, and tuberculosis. (5) The varieties resulting from introduction into the blood of toxic agents from without, as alcohol, arsenic, lead, opium. (6) The endemic or epidemic forms frequent in Asia and the islands of the Pacific, as the "kakke" of the Japanese and the "beriberi" of the Malay peninsula. (7) Certain forms affecting especially the cutaneous nerves and accompanied by trophic disorders of the skin in area supplied by the diseased nerve, of which herpes zoster is a familiar example.

Whatever the cause of the neuritis, or its pathological variety, if it involves a single nerve or a small group of adjacent nerve-trunks, it is called "simple neuritis"; if a number of nerves in different portions of the body are simultaneously affected, "multiple neuritis." The varieties referred to in 1, 2, 3, and 7 above are usually "simple"; those in 4, 5, and 6, are apt to be "multiple." To these rules, however, there are exceptions. Thus, the writer has seen one case of typical peripheral neuritis (or pseudo-tabes) due to pressure upon the lumbar and sacral nerves of a large aneurism of the abdominal aorta; on the other hand, general toxemia may cause localized effects, as has been observed in one case here, where, in consequence of the therapeutic employment of arsenic, a herpes zoster occurred. A simple neuritis is also apt to be interstitial; a multiple neuritis, parenchymatous.

Since some confusion exists in the minds of

many as to the relation borne by the different forms of neuritis to one another, and as to the proper naming of the several forms, it may be well to state that "multiple neuritis," "peripheral neuritis," and "polyneuritis" are synonymous terms, and that "alcoholic neuritis," "malarial neuritis," "syphilitic neuritis," "pseudotabes," etc., are all forms of multiple neuritis.

At the Insane Hospital, during a period of ten years, ending February, 1895, there occurred not more than a half-dozen cases of multiple neuritis, and all of these, with two exceptions, were of the alcoholic or syphilitic variety, the exceptions being the polyneuritis in the lower extremities, due to compression of aortic aneurism, to which reference was made above, and a case occurring in a negro man, who, while much excited, stamped his bare feet upon the floor until both were much inflamed, a neuritis following as a probable result of the injury.

In February, 1895, a case of multiple neuritis developed in a white female patient without assignable cause, and in the past autumn and winter —from November, 1895, to February, 1896, twelve other cases of multiple neuritis occurred. These cases were distributed irregularly through the several wards and departments of the institution, four of them occurring in white men, eight of them in white women, one in a colored woman, the population of the Hospital numbering about twelve hundred.

A brief memorandum of the thirteen cases is appended for reference:

I.—A white woman, aged thirty-two, imbecile, healthy, well-nourished. Without discoverable cause, a neuritis developed in nerves of lower extremities, extending to middle of thigh, the symptoms being pain, sensory perversions, muscular weakness and incoördination, becoming in ten days a complete motor paralysis of affected part, with inability to walk or stand and characteristic "foot-drop," trophic disturbances—some edema and a scattered herpetic eruption on legs—absence of patella reflex. There was in the beginning slight rise of temperature, restlessness, furred tongue, offensive breath, and some gastro-intestinal disturbance. After a month or two, muscular atrophy became marked, and in course of time the calves of the legs diminished to about half their former size. Electrical reactions at this time: Markedly diminished excitability to galvanic as well as to faradic current, without a typical reaction of degeneration. After remaining at a standstill for four months, patient began improving, and now (six months after attack developed) has partially regained power over muscles in the legs, although still unable to walk or to stand. She is slowly improving, and will probably recover entirely.

II.—White woman, aged forty-six, demented many years, well-nourished and in good bodily health, developed what was at first regarded as a malarial attack—chill followed by rise of temperature. A day or two later began complaining of pains in legs, with weakness, and became unable to walk. She showed the usual sensory disorders, absence of patella reflexes, muscular paresis, incoördination, etc., followed after a few weeks by atrophy of muscles of legs. She remained helplessly in bed for two months, then slowly regained use of the limbs, and now (five months after on-

set of attack) is practically well, although the legs are still smaller than formerly, and she is somewhat unsteady in her gait. Reflexes have not returned.

III.—White woman, aged forty-three, terminal dementia, physical health good. Had a "bilious attack" (or auto-infection fever) lasting a week, with nausea, anorexia, furred tongue, headache, offensive breath. She recovered entirely from this and remained out of bed a week; then began complaining of pains in her legs, of weakness, and of inability to walk. Soon grew unable to stand or to use limbs at all; feet and legs became edematous and intensely painful and hyperesthetic; patella reflex vanished; no fever nor constitutional disturbance. Some muscular atrophy supervened, and a typical reaction of degeneration could be obtained in affected muscles within a month from the time the disease appeared. After two months she began improving, and now (four months from onset of attack) is able to walk and suffers no pain. Still has some muscular incoördination and reflexes have not returned.

IV.—White man, aged thirty-five; epileptic since infancy, and mental faculties disordered for many years; anemic, emaciated, and nephritic. He took to bed with gastro-intestinal symptoms and a slight rise of temperature, followed by pain, sensory perversions and muscular weakness in legs, with loss of patella reflex. He was never entirely paralyzed, muscular atrophy was not marked, and after two months he was able to walk again. Reflexes are still absent after five months.

V.—White man, aged thirty-three; epileptic since childhood, imbecile and demented, but healthy and well-nourished. Without assignable cause, and without any pronounced constitutional disorder, legs became weak and unsteady, pain-

ful, edematous, and patella reflexes disappeared. He was unable to walk for several weeks, then slowly improved, and after two months was able to get about as usual. Muscular atrophy slight.

VI.—White woman, aged twenty-six; hysterical mania, with dementia. Health good, body well-nourished. Beginning with a mild auto-infection there was rise of temperature to 101° F. on second day, she developed pain and tingling sensations in legs, with muscular weakness and absence of reflexes. She remained in bed one week only, then became able to walk again, improved steadily, and in two months had practically recovered.

VII.—White man, aged thirty-eight. Chronic melancholia with dementia; anemic, nephritic, and poorly nourished. Neuritis developed in lower extremities without visible cause, the symptoms being pain, sensory disorders, weakness, absence of reflexes, etc., as in the other cases, together with some gastro-intestinal disorder. At this time (three months after onset) patient is partially recovered, but is in bed a part of the time and legs are weak and smaller than before.

VIII.—White woman, aged forty-five. Chronic mania engrafted upon a paranoiac disposition. Previous health good. After three or four days of malaise, rise of temperature to 100° F., gastro-intestinal disorder, etc., local symptoms of neuritis in nerves of legs and lower half of thighs appeared, with burning pain, sensation as of "ants crawling on legs," "as if legs were asleep," edema, tenderness, muscular weakness, etc. She was never entirely paralyzed, pain grew less after a few days, and she retained power of locomotion throughout, although showing a markedly ataxic gait. Patella reflex abolished. Later, distinct muscular atrophy and a partial reaction of degeneration were noted. Gradual recovery of power

in legs and restoration of reflexes in five months.

IX.—White woman, aged thirty-three. Epileptic since childhood and demented for ten years. Previous health good. Underwent the "Flechsig opium treatment" for epilepsy—opium in slowly increasing quantity given for a month, suddenly withdrawn, and potassium bromid in large doses substituted. Fits stopped, but about two weeks after withdrawal of the opium, patient having for some days shown gastro-intestinal and auto-infection symptoms, a neuritis developed in lower limbs, all symptoms more marked on one side than on the other. The pain was a prominent feature for several weeks, and, later, muscular atrophy became pronounced. After a month symptoms of neuritis developed in both forearms, and after this both foot- and wrist-drop were present. Reflexes abolished. Patient grew steadily weaker, emaciated, became stupid, lay in a semi-comatose state for days, had persistent constipation, furred tongue, sordes on lips and teeth, offensive breath, anorexia, no rise of temperature, but toward close (six weeks after onset) temperature became subnormal; no difficulties of respiration nor cardiac irregularities noted. Died seven weeks after beginning of the attack.

X.—Colored woman, aged thirty-six, imbecile; previous health good. Had an attack of "continued fever" (which was probably tubercular) lasting five weeks. Recovered and was up and able to move about for a week; then developed a violent neuralgic (?) pain in chest, and in a day or two exhibited typical symptoms of neuritis in lower extremities, and a few days later in nerves of arms and body (intercostals) also. Temperature became subnormal. She grew weaker, ex-

perienced great difficulty in respiration, and died of asphyxia, without cardiac irregularities, three weeks after disease first appeared. Autopsy showed general miliary tuberculosis, involving pia mater, as well as all internal organs.

XI.—White man, aged thirty-four. Epileptic convulsions for five years past; mental weakness noticed during four years. Health good; muscular and well-nourished. With fever and the frequently noted gastro-intestinal disturbance, the pains, sensory abnormalities, and muscular paresis characteristic of neuritis developed in the arms first, slowly thereafter in the legs, rendering patient nearly helpless. Wrist- and foot-drop marked. The pain was severe from "top of head to sole of foot," as he said; hyperesthesia and absence of reflexes also noted; some edema; later, some muscular atrophy. After four weeks he began suffering from dyspnea and cardiac irregularities, temperature became subnormal, and he died two months after the onset of the disease, of cardiac failure and asphyxia (involvement of pneumogastric, probably).

XII.—White woman, aged forty, utterly demented for many years. Previous health good. The nurse noticed that she seemed indisposed to move from place to place, and that when necessary to move she would get down on the floor and crawl like an infant. Examination showed absence of patella reflexes, inability to stand or walk steadily, sensory perversions, edema, incoördination of movement. Patient was too demented to complain of pain if she experienced any. She showed no constitutional disorder, and refused to remain in bed. She regained the use of her legs after two months. Reflexes were absent after five months, and some atrophy is still apparent, although she walks fairly well.

XIII.—White woman, aged fifty-six. Chronic mania, with great excitement and turbulence. Nephritic, emaciated, and in feeble health, with frequent diarrhea, progressive emaciation, and exhaustion from excitement. After being excited for two weeks she became helpless, and upon examination the usual evidences of neuritis were discovered in the lower extremities—muscular weakness, absence of reflexes, pain and tenderness, inability to stand. Trophic disorders were more prominent than in the other cases—edema and a herpetic eruption, and later a crop of small boils. The neuritis was confined to the legs. Patient died of exhaustion and nephritis about two weeks after disease developed.

It will be noted that the neuritis affected the lower extremities in every one of the thirteen cases, and the lower extremities alone in ten of the cases. The other three cases, in which the nerves of the arms and body became involved, all proved fatal although only one of these fatal results could be attributed directly to the neuritis (Case XI). One of the other cases died of miliary tuberculosis, the third of auto-infection, although death in each case was doubtless hastened by the neuritis. In one case the neuritis developed in course of a miliary tuberculosis, in one it followed a "bilious attack," in one the "Flechsig opium treatment" for epilepsy, in one a doubtful malarial infection; in none of the other cases could a reasonable guess at a cause be made.

The patients were young or middle-aged, and had been insane and in the institution some time; four were epileptic; all were demented more or

less; none had used alcohol, and in only one case was a (doubtful) history of syphilis obtained. In most of the cases there was some evidence of an acute general toxemia, usually noted a day or two before the local symptoms attracted attention. In at least three cases, however, symptoms of general disease were lacking. No marked effect upon mental state nor upon the course of the insanity was noted in any instance. An examination of the urine of about three-fourths of the cases was made after the acute stage was passed, without discovering any indication of increased renal irritation, the urinalyses, as a whole, giving the same result as had been obtained at examinations made before the neuritis developed.

Why, after so many years of immunity, we should have a dozen cases of peripheral neuritis within a few months, is not clear, unless we assume the existence of some endemic cause, absent from our locality up to last year, such as is supposed to be active in beriberi—some germ or other source of infection. Excess of starchy food has been assigned as a cause of endemic neuritis, but here the food of the patients does not show an excess of carbohydrates, and the quality and kind of food has been practically the same for years. Infection also is not clear. While four of the cases occurred in one ward, they all developed about the same time, and no other cases appeared; the remaining nine cases were widely scattered. The summer and autumn of 1895 was, it is known, a sickly season, malarial diseases, among others, abounding. While our neuritis cases, with one

exception, obviously did not follow malarial infection, the causes favoring the growth of the malarial germ in the South may have also been favorable to the development of the etiological basis of endemic multiple neuritis. The blood of several of the neuritis patients was examined for the malarial plasmodium, without success. Although cases of multiple neuritis among the general population of the State have not come to my notice, I am informed by Dr. P. T. Vaughan, assistant physician at the Arkansas State Asylum for the insane, at Little Rock, that a number of cases of multiple neuritis occurred among the insane patients at Little Rock about the same time we were experiencing the epidemic at Tuscaloosa, indicating that the cause, whatever it might be, was widely diffused, rather than local or peculiar to the Alabama State Hospital. Several of our cases showed the auto-infection symptoms—dryness of tongue, offensive breath, emaciation, etc., seen in the dry or atrophic form of kakke or beriberi.

A fairly representative composite clinical picture of the disease, drawn from the cases we have seen, is the following:

After a period of malaise, lasting from a few hours to several days, with the discomfort common in the prodromal stages of acute disease, sometimes with a distinct chill, usually with a rise of temperature, together with anorexia, dryness of mouth and throat, furred tongue, offensive breath and constipation, the patient begins complaining of pain in the affected part, the legs most often. The pain is often quite severe, is darting

and shooting, or burning, boring, aching, stinging, or "like ants biting," as one patient expressed it. Muscular weakness and incoördination is then noticed, and other sensory disorders appear, as diffuse or irregularly distributed areas of hyperesthesia or (rarer) anesthesia; tenderness on pressure is common, sensations as of "bugs crawling on the legs," "as if the limbs were asleep," are also mentioned. The tendon or deep reflexes are also abolished (noted in all of our cases). The muscular weakness increases in severity, the gait becomes ataxic, patient is unable to get up and down steps, unable to stand steady with eyes closed; the weakness may become only a paresis, but in many cases there is almost complete paralysis at the height of the attack, with the characteristic wrist-drop or foot-drop. The sensory disturbances subside after a longer or shorter time, the motor weakness is more persistent. Trophic derangements, appearing at a later stage than do the sensory and motor disturbances, are also frequent, as edema, eruptions, sores, discoloration, etc., of the skin, or, more rarely, a herpetic eruption, or even a typical herpes zoster. When the motor paralysis becomes well-marked, there is always atrophy of the muscles supplied by the affected nerves, this becoming, in some instances, very marked. Contractures may result. There are then also changes in electrical reaction, either a diminution in galvanic, as well as faradic excitability, or a partial or complete reaction of degeneration.

The disease progresses in intensity for a period

ranging from a few days to some weeks, then remains stationary for a shorter or longer time, then slowly disappears, pain first, motor weakness next, reflexes last returning. There are all grades of severity and duration, from a mild paresis, disappearing in a few weeks (Case VI) to paralysis of some months' duration (Case I), or the disease may cause death, as in Case XI. Usually the peripheral extremities only of the nerves are affected, the disease extending to the middle of the arm or to the middle of the thigh, as a rule. In the severer cases, and possibly in a few of the milder grade, the inflammation extends quite to the cord. While the great majority of cases recover, the disease often proves a serious one, as is shown by our three fatal cases among thirteen. Death, when directly owing to the neuritis, as in Case XI, is generally due to participation of the pneumogastric and other respiratory nerves in the disease, death occurring from asphyxia or from failure of the heart.

The diagnosis of peripheral neuritis, save in the cases pursuing an atypic or unusual course, is not a matter of difficulty, the rapidly developing motor weakness, incoördination, loss of tendon-reflexes, together with the pain, perversions of sensation, and trophic phenomena offering a characteristic clinical picture. Very mild cases might be overlooked, the slight pain and muscular weakness not attracting sufficient attention to suggest an examination of the affected limbs, or being attributed to "neuralgia" or "muscular rheumatism," etc. In the severer cases also, the local

manifestations may be overshadowed by the marked constitutional disturbance—fever, general prostration and weakness, auto-infection symptoms, etc., but in this, as in the first instance, if an examination of the nerve-reactions is made, there will be little opportunity for error.

The symptoms of poliomyelitis anterior, or infantile spinal paralysis, resemble somewhat those of multiple neuritis. Poliomyelitis, however, is preëminently a disease of infancy and early childhood, and is usually a purely motor paralysis, lacking the sensory disorders of neuritis.

Acute ascending or Landry's paralysis resembles a polyneuritis in many particulars; in fact, many of the symptoms of Landry's paralysis are due to involvement of the peripheral nerves, and some of the reported cases of Landry's paralysis are most probably cases of multiple neuritis; in fact, some (Ross) have insisted upon the identity of the two diseases. The chief differences noted in typical cases are: Landry's paralysis is usually a motor one, the pain, trophic disorders, etc., of neuritis being absent; it advances more rapidly than neuritis, the muscles do not atrophy, and the reaction of degeneration is not present.

When a polyneuritis involves the lower limbs alone, and is of slow onset and progress, as is often seen in cases due to alcohol or syphilis, the disease bears a close resemblance to tabes dorsalis or locomotor ataxia, and well deserves the name of pseudo-tabes, which has been applied to it. The symptoms common to the two diseases are: The pains in the legs, the sensory perver-

sions, muscular incoördination, ataxic gait, swaying with closed eyes, absence of tendon-reflexes. The differences are: The girdle-pain of tabes is not present in neuritis; the lancinating, stabbing pains of tabes are not common in neuritis; the pain in the last-named being continuous, burning, stinging; the pupillary-light reflex, absent in tabes, is not changed in neuritis. There is loss of coördinating power in both diseases, but in tabes there is, at least in early stages, no muscular weakness, while this weakness is marked in neuritis; the "crises" of tabes are absent in neuritis. Thus, in typical cases the differences are sufficiently prominent. In some atypic instances, however, it becomes a matter of no small difficulty to decide between a neuritic pseudo-tabes and a true tabes—a fact not surprising when the pathology of the two is borne in mind. The nerves are usually affected in tabes, and many of the symptoms of tabes are obviously those of the accompanying neuritis; in fact, it has been claimed that locomotor ataxia is a primary affection of the peripheral sensory neuron.

The treatment of multiple neuritis is, first and most important, removal of the cause, if such is discoverable and removable—withdrawal of the alcohol, opium, arsenic, lead, etc., treatment of the syphilitic, malarial, or other infection.

For the neuritis itself, there is no specific. Of drugs, quinin and the salicylates are most often recommended. For the relief of the pain, the most distressing single symptom, coal-tar derivatives or, preferably, opium, may be given. The

steady galvanic current is sometimes of service; hot applications give much relief in some cases—hot water, hot flannels, hot poultices. After the acute stage is passed, the aim is to minimize the muscular atrophy and prevent contractures, and to restore muscular power as soon as possible. This is best done by the systematic use of electricity (faradic current) and massage. In the large majority of cases recovery is complete, although from several months to a year, or even longer, may be needed