INTERMITTENT SPINAL PARALYSIS OF MALARIAL ORIGIN.

BY

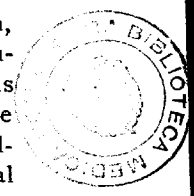
V. P. GIBNEY, M.D.,

New York.

CASE I.—First attack with nearly complete recovery under quinine in one week; second attack a month later and recovery not complete at end of three months, quinine not being regularly administered; recovery complete within four months after removal to non-malarial locality; third attack five weeks after return to malarial locality, and recovery complete in three weeks under quinine and after removal; fourth attack four and a half months after return home; in hospital again three weeks later, and at end of six months recovery not quite completed; fifth attack six months after discharge from hospital, and recovery nearly complete at end of two months; electricity employed during the last four attacks.

Frank P., aged seven years, was brought to the Out-door Department of the Hospital for Ruptured and Crippled, Sept. 28th, 1878, for a hydrocele. There was observed at that visit a peculiarity in his gait that excited our interest, though no observations were recorded. Indeed, no history was obtained until some months later, when it was learned that he was one of three children, that phthisis and Bright's disease existed in his maternal grandparents, and that his mother was of a nervous temperament. I have quite recently learned from Dr. C. Peck Smith, the family physician, that, for several years, she has suffered quite severely at times with various neuralgias. The father is intemperate.

Our little patient was living in the summer and early fall at 206



W. 32d street, on the first floor of a rear building, and the mother always reported, when asked about hygienic surroundings, that the house was damp and that malarial fevers prevailed in the immediate neighborhood.

In the early part of September, he complained of being chilly, and this feeling was followed immediately by heat and vomiting and general indisposition. He had what she termed a "bilious attack." On the following evening, he seemed to have high fever and passed a very restless night, crying aloud repeatedly, and acting like one delirious. There were no convulsions; his bowels were constipated; he referred the pain to his limbs; in fact he "seemed sore all over," and cried if he were handled. On getting out of bed next morning, he was unable to walk, but could take a few steps after being rubbed. The second night following this, he was feverish again, and in the morning he could scarcely use his hands and arms, crying at the same time from muscular pain. During the succeeding week, he was confined to his bed with a fever; had much cutaneous hyperæsthesia; was at times bordering on delirium; was obstinately constipated, but had no opisthotonos and no symptoms pointing to disturbance of the bladder. He took much quinine at this time. After a week, there was temporary improvement, and he was up for three days, when he fell sick again, and was worse, it seemed, than during the first attack. On the subsidence of the fever, nearly a week later, paralysis of the four extremities was, to all appearances complete. Power returned very slowly. The family physician finally referred him to Dr. Seguin, and the case was exhibited at the clinic early in January. Dr. L. Emmett Holt was then a student at the College of Physicians and Surgeons, and he gave me the following notes which were taken verbatim from Dr. Seguin's lecture: "A peculiar case of infantile paralysis. History: went to bed four months ago well; no symptoms during the night. In the morning, the legs were palsied to the knee. On the following morning, the arms palsied; no other palsy, no head symptoms, bladder normal. Progressive increase in the paralysis and wasting in the paralyzed parts. No anæsthesia (?). Examination: extensors of legs good; extensors of feet show a little power—he slightly moves the toes of each foot; extensors of wrist good, [though] on left side some effort [required]. Paralysis of flexors [anterior tibials] of feet. Some faradic reaction in gastrocnemius, none in anterior tibial [those on left side not mentioned]. No reaction in thenar or hypothenar eminences [galvanic-test not mentioned].

Diagnosis: myelitis of the anterior horns." Dr. S. did not see the patient again.

He was admitted to the hospital Jan. 14th, 1879, and our examination then was as follows: A fairly nourished boy standing unsteadily, and when not supported, with limbs in marked genu-valgum; walks very oddly, balancing himself for a step with apparent effort; limbs widely separated; one foot is then brought forward, coming down to the floor with a flop, after which, as the weight of the body is thrown upon the limb, a genu-recurvatum is produced. The other foot and limb then go through with a like performance. There is a diminution in power in the supra-scapular muscles, in the flexors of the fingers, and possibly in the extensors of the fingers. The thenar and hypothenar eminences are undoubtedly atrophied to a moderate degree, at least. The muscles already mentioned and those to be mentioned are very flabby as one feels them.

The flexors of the thighs are subnormal in form, while the extensors are normal; the adductors seem subnormal, the abductors normal. There is decided relaxation of the posterior ligaments of the knees. Extensors of the legs are paretic, flexors normal in power. The knee reflexes are normal. He is unable to flex the foot, and the power to extend is far from normal; he flexes and extends the toes quite well. The Achilles tendons are contracted so that the feet cannot be passively flexed (dorsally) beyond 90°. The feet are cold. There is no difference in the comparative measurements of the limbs; the circulation is poor. The symptoms in fact are quite symmetrical throughout. The electrical reactions were not recorded. A diagnosis of infantile paralysis with an interrogation point was made, and the treatment consisted in electricity, and apparatus to overcome the contracted tendines Achillis. During the latter half of February, he had a naso-facial erysipelas, and on *April 23d*, it is recorded that decided improvement in the paralysis has taken place. The apparatus has been discontinued since he walks very well without any support. He is now able to button his clothing; this his mother thinks a decided improvement. No medicines have been employed save those used for the erysipelas. *June 4th.*—Within the past six weeks, the progress toward recovery has been unmistakable. His gait is nearly perfect, and he executes all the movements of the feet without apparent effort and with nearly normal power. He uses his hands well, and the degree of improvement casts a shadow of doubt over the diagnosis, at least from a pathological stand-point. The electrical reactions, both currents,

in all the muscles, are about normal. *July 11th.*—He was discharged and a very thorough examination was made, comparative measurements being taken of both upper and lower extremities without any difference whatever in size. The thenar and hypothenar eminences had become fuller. The individual groups of muscles were submitted to all the usual tests, including both currents, and full notes made, although negative in character. I do not propose to transcribe them here, but simply mean to say that the boy left the hospital *cured*, and went back to his home in 32d street.

Aug. 25th, 1879.—Readmitted this date. Since his discharge, he has continued well until three days ago. On the evening of the 21st, he went to bed complaining of being cold, and extra bed-clothing did not succeed in keeping him warm; he was restless throughout the night, and towards morning he was feverish, keeping his bed until this morning. Every night he has complained of being cold, though seemingly feverish. He has likewise complained of pain in his legs, has had no appetite, has had nausea, but no vomiting, and his bowels did not move from the *21st* to the *24th*. He had an action yesterday evening, and a remission of his symptoms followed almost immediately. The patient is very irritable, and is so weak in his limbs that he has to be carried into the hospital. This attack closely resembled that in September last, the mother reports, the difference being that this is less severe.

It is learned that, for several months before his first attack, he lived in a house on the same street, between Eighth and Ninth avenues, in which a sewer leakage was afterwards discovered, the family moving out as a consequence. It is learned, also, that a woman in the same house had what was called malarial fever, and recovered only after she had moved away. There were no other cases in the tenement.

The muscles of the arms, *Aug. 27th*, respond vigorously to a strong faradic current, equally well on both sides; the muscles of the forearm scarcely perceptibly to the same current slowly interrupted, the flexors responding a little better than the extensors. It is doubtful whether the muscles of the hand respond at all. When the positive pole is placed over cervical spine and negative over dorsum of forearm, the flexors react, while the extensors do not. The galvanic current gets the reaction in same muscles not quite so vigorously, with poles similarly placed. The electro-sensibility is about normal. The muscles of the lower extremities react in the same way as those of the upper extremities, with this

exception: a strong galvanic current gets no responses in the peroneals, while the faradic gets response barely perceptible. He was ordered quinine and faradism.

Sept. 17*th.*—To all appearances completely restored. Cod-liver oil ordered in addition to the quinine, which has been given in about gr. iv. doses twice a day. This was discontinued two weeks later, and he would have been discharged but for the occurrence of a glandular enlargement in the axilla. This disappeared spontaneously, and on *Nov.* 14*th*, at noon, he had a slight chill followed by headache. Unfortunately his temperature was not taken. He was not in a good condition of health, and he remained in the hospital until *Feb'y 6th*, when he was discharged, there having been no return of the chill and constitutional disturbance of any kind. He was examined with much care, and found to have made a complete recovery.

June 25th, '80.—During the past three weeks, he has been getting weak again; he falls easily, is very weak at the knees, and has not power this morning to flex either foot to 90°, though they can be forcibly flexed to the full extent. He is living at the same place, and about a month ago he was sick for a week, having a chill every other day. He is now ordered quinine gr. iiss. t. i. d. There does not seem to be any elevation of temperature, and the thermometer is not employed.

July 19*th*.—Again admitted to the hospital, helpless and crying with pain in his calf muscles and in the soles of the feet. There is complete palsy in the anterior tibial groups, and no reaction to a very strong faradic current. It takes quite a strong current to get any reaction in the posterior tibials even. The galvanic test is not employed, battery not being in working order. The extensors and flexors both of the thighs and legs seem not this time to be at all affected. He cries all the afternoon with pains in the lower limbs, and asks that they be rubbed, which being done, gives him relief. His tongue is coated, and his vital signs are 120, 26, and 102°. Next day, in the morning, his temperature was 101¼°, and in the evening 101½°. Quinine is ordered gr. iv. t. i. d. On the next day, 21*st*, he keeps his bed, lying on his left side, with thighs flexed on abdomen and legs on thighs. Face is flushed, and he passed a bad night, *i. e.*, he cried much with pain in his limbs. His temperature in the morning was 101°, in the evening 101°. No relief came next night until 3 i. of the U. S. sol. of morphia was administered. On the following day, continuing quite helpless, the fluid extract of ergot, 3 ss. three times a day, was added to the quinine. In the evening, his temperature was

99½°. On *July 23d*, it is recorded that he is crying with pain in his limbs, and on the *24th* he was more comfortable, though constipated. There was no elevation of temperature worth recording from this time forth, and the quinine was discontinued. He soon began walking about again, feebly, however; the knee phenomena could not be obtained, and the reactions to faradism were very unsatisfactory. Indeed, at times, no reaction could be had at all. A month later, one day in September, I had a call from my friend Dr. Putzel, and he tried faithfully to get reaction in the anterior tibials, failing most signally to get any response to either a strong faradic or a strong galvanic. It was not until *Jan. 5th*, '81, that the above muscles reacted to the two currents. The strength was such, too, as to make the application wellnigh intolerable. On the *10th of March*, a much milder current produced reaction. He was discharged this date, and the restoration was complete, with this exception: the small toes of each foot remained in flexion, the tips of the nails touching the floor—"crumpled."

Sept. 28th.—He is walking badly again, typical drop-foot existing. The small toes are still "crumpled," as when he left the hospital, only the deformity is a little more marked. He did not return to the same place, but has been living on Ninth avenue, in a locality presumably free from malarial influences. He has not been sick any of late, and nothing would seem to indicate a return of malarial poisoning, unless the following could be so construed: on the *5th of October* he appeared feverish, there was vomiting, had no appetite, his bowels had not moved for two or three days, his tongue was coated, he had no chill, yet his symptoms led the mother to pronounce this a "bilious attack." He was better on the *6th* and *7th;* on the *8th*, his limbs became weak, and on the *9th* he was not only paretic, but he had much pain in the calves, the pain keeping him awake nearly all night. On the *10th*, he was unable to walk. On the *11th*, he walked without assistance, though experienced much difficulty in clearing the floor with his toes. There is inability to flex either foot. Quinine and ergot are ordered. Dr. Putnam-Jacobi saw the case on the *17th*, and could get "no response to faradic current in the extensor communis, peroneal, or anterior tibial groups, but did [get a response] in the extensor of the great toe, and in the calf muscles." The extensors of the wrist responded vigorously to the faradic current. The doctor excluded anterior poliomyelitis, and after getting a complete history, favored the view I was entertaining, viz., a spinal hyperæmia, probably malarial. The patient was admitted to the hospital for the fourth time on *Oct.*

20*th*, and his condition was similar to that recorded in the other attacks. The paralysis did not involve so many muscles as on former occasions, yet the anterior tibial groups were quite as powerless, and the reactions were quite as difficult to obtain. The small toes presented the same peculiar appearance as when he was last discharged. I have repeatedly examined his gums for the blue line of lead poisoning with absolutely negative results. His anterior tibial muscles on *Nov. 24th* responded to a strong galvanic current, and the peroneal responded to a strong foradic. At present writing, *Dec. 2d*, he has recovered nearly all the power in the muscles that were paralyzed. There is for the past fortnight a desquamation of the epidermis of feet and legs, exactly like that which occurs after scarlatina, and yet the boy has not had any symptoms pointing to scarlatinal poisoning. This desquamation does not occur on any other part of the body. His knee reflexes are normal.

Erb, in Ziemssen,[1] remarks that "the occurrence of purely intermittent attacks of paraplegia and paralysis which, according to all appearance, are of spinal origin, and which make a most striking impression as contrasted with the usual stability of spinal paralysis of similar severity, is undoubtedly one of the most remarkable forms of manifestation of malarial infection," and I agree fully with him when he says "literature contains but very few instances of this form of spinal paralysis."

Hertz[2] makes an abstract of Macario's case[3]

"in which a woman was seized, two days after confinement, without any known case, with formication in the feet, which then spread to the thighs, the trunk, and the upper extremities, these becoming, at the same time, paralyzed and anæsthetized, and her tongue also becoming paralyzed. These manifestations were repeated three times after the quotidian type, and were then arrested by quinine."

Hertz himself has never seen a case. Romberg[4] de-

[1] Wm. Wood & Co., New York, vol. xii., p. 814.
[2] Wm. Wood & Co., New York, vol. ii., 601.
[3] Gaz. méd. de Paris, 1857, No. 6.
[4] Lehrbuch d. Nervenkrankh., i., p. 752.

scribed a case which is quoted by Erb in his articles, to which reference has already been made.

"A woman sixty-four years of age, after having been quite well the day before, was suddenly attacked with paralysis of the lower extremities and the sphincters. Sensibility was unchanged, consciousness clear, the temperature cool, pulse 80, small and empty, no pain in the spinal cord. The next day there was an astonishing change in the condition. The patient can walk again and void urine voluntarily, and only complains of weakness in the legs. The next morning there was paraplegia again, which had set in at the same hour as two days before. A third paroxysm was awaited, which also set in at the appointed time, although without paralysis of the sphincters. Quinine effected a rapid cure."

Hartwig,[1] also quoted by Erb, relates the case of a vigorous laborer, aged twenty-three, who, five years before, had suffered from a tertian intermittent for a few weeks, but from that time till *Nov.*, 1873, had remained perfectly well and strong. Then he first felt weariness in the legs, this gradually increasing, the arms soon participating. On the third day, he was obliged to take to his bed, and the night following there was complete paralysis of legs, trunk, arms, and even the movements of the head. The muscles of the face escaped, while speaking, breathing, and swallowing were somewhat hindered. The sphincters were uninvolved, sensibility was intact, no head symptoms, no pain. Perspiration was excessive. At the end of twenty-four hours, these symptoms subsided, and in half an hour, generally with an increased perspiration, all the muscles again became movable. For the next twenty-four hours, he had no sign of paralysis, a weariness and heaviness of limbs being all that he complained of. Then followed an attack of paralysis again, and then there followed regular successive free intervals and attacks of about twenty-four hours' duration. The time occupied by the attack gradually extended to forty hours, the interval being much shorter. Under arsenic, the intervals were lengthened to about forty hours; quinine lengthened them at first to four days, then strychnine, hypodermically, was followed by a re-establishment of the tertian type. A few times the sphincter ani was affected. In one of his attacks, at the end of March, he lay completely paraplegic, only the facial muscles acting normally, the flexors of hands and of feet showed a minimum amount of motion, sensibility of the

[1] Diss. Halle, 1874.

skin and muscles normal; pulse, 72; respiration, 20; temperature 99½°. Reflex action entirely wanting, electrical excitability of the muscles almost entirely extinguished (during the intervals only diminished). Greater and lesser irregularities occurred in the succeeding month; quinine lost its power, yet the disease retained its strongly remittent type, and gradually the cachexia became marked, the muscles showed marked emaciation. The final result of the case is not reported.

Dr. Wilkes, in Guy's Hospital Reports,[1] reports two cases of intermittent tetany, in which paralysis, from the history, seems to have occurred, though he does not attribute the attack to malarial poisoning. One patient was thirteen years of age, and the other sixteen. In both of these, however, there were severe muscular spasms.

The literature of malarial poisoning is full of neuroses; and if one attempted to collect all the convulsions, the muscular spasms, the neuralgias dependent on its influence, a paper would swell into a volume. Trousseau, indeed, after discussing the neuroses of marsh fevers, says:[2]

"We may in this way pretty easily explain the very varied symptoms of both simple or pernicious marsh fevers; in this way, confirmation is given to an opinion already held by others, and in which I completely concur, viz., that intermittent fevers, under whatever forms they may present themselves, ought to be classed with the neuses."

There is nothing then to prevent a patient who suffers from malarial fever getting a paralysis almost identical with infantile spinal paralysis, or, more properly speaking, anterior poliomyelitis. The anatomical diagnosis appears to be the same, yet, pathologically, we have, I think, a very different condition. In lead palsy, too, the same region of the cord is affected, and perhaps, also, the same regions are affected in diphtheritic paralysis. The prog-

[1] Vol. xvii., 1872, p. 181.

[2] Clinical Medicine. English Translation. Phila., 1873, vol. ii., p. 695.

nosis, however, differs materially in these toxic paralyses from that in the true anterior poliomyelitis.

It will be seen by an analysis of the case I have just placed on record that his *first* attack occurred in September, 1878, a few days after an intermittent fever, and that partial recovery took place under the administration of quinine within one week. Indeed, when I first saw the patient for a hydrocele, it was on the twenty-eighth of September, and there was nothing noticeable except a peculiarity of gait. The *second* attack occurred the next month, and the recovery was not complete in January, at the time he was first admitted to the hospital. Quinine was not employed, and he did not recover until four months after admission. He was simply removed from malarial influences.

The *third* attack was five weeks after his return home, and he was readmitted three days after its invasion, put upon quinine and faradism, recovery being complete this time in three weeks.

The *fourth* attack, four and a half months after going home, was preceded one month by the usual "bilious spell," and he did not come into the hospital again until three weeks later, when there was considerable sensory disturbance, requiring morphia for several days. It was six months this time before he recovered, although quinine was at first used, and then ergot. Be it noted too that the recovery was not perfect, inasmuch as the interossii were left over, and have not as yet recovered their tone.

The *fifth* attack came on six months after his discharge from the hospital, coming on gradually, and attended, like the fourth, with pain in the four limbs, and improvement was more marked after he came into the hospital, and now nearly two months have passed, and the cure is not complete.

There is this peculiar feature, viz., the epidermal desquamation.

The next case I have to report had three attacks:

CASE II.—Wm. Johnson, aged six years, was admitted to the hospital May 13th, 1880, poorly nourished, head large, and walking with much difficulty. As he walked, the limbs were widely separated, and the toes scarcely cleared the floor. He could not flex (dorso-flexion I mean) either foot, and both unsupported hung in talipes equinus; the thigh flexors were weak, and he made quite an effort to get up from the floor. There was a hyperæsthesia in the inferior extremities, and the muscles felt flabby as one handled them. The grasp of both hands was weak, the faradic reactions in extensors and flexors of wrist being good, yet not vigorous. In the lower extremities, especially in the anterior tibial group, a very strong faradic current got a very feeble response. There was no comparative atrophy, and apparently no real atrophy, of the limbs. The family history was not good, the father being intemperate, the mother being of delicate build, and often out of health. No distinctly transmissible diseases, however, were traceable.

Two years ago, during the summer of 1878, the family were living in 118th street, between First and Second avenues, when this boy was taken suddenly ill one day, complaining of pain in back and limbs, vomited, and his skin was excessively hot. The pain in the limbs passed off next day, but the fever continued, and he was sick for nearly six weeks. During convalescence, his limbs, upper and lower, became quite powerless, and he had no treatment for this but rubbing of the limbs, and bathing them in salt water. He recovered completely within a few weeks, and had no return of malarial symptoms or paralysis until April, 1880. At that time he lived at 336 E. 115th street, and much street-repairing and street-opening goes on constantly in that locality. He began with a feeling of malaise; he had dull pains in his limbs by day, but towards one or two o'clock at night he would awake out of sleep crying aloud and complaining of pains in the limbs. These would leave him towards morning, when, on rising, he would be comparatively well. Febrile symptoms accompanied these pains by night, and his mother insisted that they occurred every night during the latter part of April and the first of May. He gradually lost power in his limbs until he was admitted to the hospital. The treatment was electrical, and apparatus to support feet at right angles. The reactions improved,

he gained power, the apparatus was soon discontinued, and on Sept. 24th he was discharged cured.

In tracing the case out recently, I have learned from both father and mother that he continued well and free from paralysis until last spring, when he was taken in the same manner as in the former attacks, the constitutional symptoms being more severe. He became paralyzed in all four extremities during the first day or two, his bowels were very constipated, and during the second week he had convulsions, but, in the intervals, his mind was clear, and he died simply from exhaustion, he being conscious almost up to the hour of death. The attending physician states that he did not have tubercular meningitis. No autopsy; and hence a good cause pathologically lost to science.

I very much regret that I am unable to find any records of autopsies, and that I must content myself in speculating on the pathology of intermittent spinal paralysis. I cannot help but think that the pathology must be essentially different from that in the ordinary spinal paralysis of infancy. While studying these two cases clinically, I was very much impressed with the symmetrical development of the symptoms, with the symmetrical returns of the same, and even in the study of the cases of Macario and of Romberg and of Hartwig I am still more impressed with the symmetry of the paralysis; in fact, I do not now recall such cases of intermittent poliomyelitis, unless it be Case XX. in Dr. Seguin's monograph on Myelitis of the Anterior Horns.[1] This patient did not make a complete recovery, however, as his left hand remained numb and weak up to the date of his second attack.[1] At one time, too, when I could get no reaction to either current in muscles not much atrophied, I could not explain the nature of the lesion in the anterior cornua. In no instance have I found a typical degeneration reaction, and yet the toxic influence on the nerve-centres at one time in Case I. was so profound that neither current, as above mentioned, would bring about any reaction, and yet we had a good

[1] G. P. Putnam's Sons, New York, 1877, page 25.

recovery. Now, can this be an acute myelitis? While my friend Dr. Seguin is sanguine, to a limited extent, in the acute and subacute anterior myelitis of the adult, he expresses himself, in 1877, in his monograph, to which reference has just been made, on page 117: "I am at present inclined to the opinion that unless cured in two or three months, children with myelitis anterior have very little prospect of perfect recovery," and a few lines further, generally speaking, he says: "In other words, I believe that the prognosis in adults and children depends upon a factor which we can only indirectly determine—I mean the amount of injury done to ganglionic cells of the anterior horns. We have not as yet, and probably a long time will elapse before we shall have, the elements for saying that ganglion cells may be regenerated, or even that they may be purged of accumulations of granular matter." Now, whatever views Dr. Seguin may entertain at present, I must say that my own study of cases, not a few from a clinical stand-point, and from my study of them as to ultimate results under circumstances, often laborious, compels me to say that I am unable to adduce a single case of what I believe to be idiopathic anterior poliomyelitis of childhood in which complete recovery has taken place. I know that Erb speaks lightly of Kennedy's "temporary paralysis," saying that "scarcely any of his cases belong under this general head [poliomyelitis anterior acuta], and those which do belong under it were not 'temporary.'" Speaking of prognosis, he says additionally: "In fact, the best we can do, almost always, is to make a prognosis of *recovery, with greater or lesser remaining defects.*"[1]

I am aware, too, that Frey and some others have reported cases of complete cure, yet I am among those who question their diagnosis or the completeness of their records.

[1] Ziemssen's Encyclopædia, vol. xiii., pp. 702, 706.

In Erb's article on Intermittent Spinal Paralysis, he objects to Hartwig's views, that the changes which take place are transitory hyperæmia and serous transudations into the substance of the cord, on the ground that the disturbance is always confined to the motor portions of the cord, leaving the sensory portions entirely free. This Erb calls a fact, but I am unwilling to grant that the sensory functions are always entirely free. Those violent pains certainly prevent us from excluding the sensory tracts, except on the hypothesis of a neuritis migrans. For my own part, I do not see why a hyperæmia of the cornua in the level of the sixth, seventh, and eighth dorsal, and of the first, second, and third sacral nerves, when the anterior cornua are so large, may not produce motor disturbances, taking such precedence over the sensory that the latter becomes insignificant.

In pernicious intermittent fever, in which coma and other grave nervous disturbances have existed, very slight changes are found in the cerebro-spinal axis. "In the comatose form," says Hertz, "a brownish or slatish discoloration of the cortical portion of the brain, and sometimes also of the ganglia, has occasionally been discovered, which was due to accumulation of pigment matter, as well as numerous punctiform extravasations, which have been thought to depend on the blocking up of the capillaries by means of pigment, but which Heschl says may exist independent of the formation of pigment matter, and represent capillary aneurisms. Instead of this, however, it is more common to find only more or less hyperæmia and œdema of the brain-substance and its membranes . . . indeed, hyperæmia and œdema of the brain are the principal conditions present after all the disturbances of the central nervous system connected herewith."[1]

I simply am led to the belief that a congestion of the

[1] Ziemssen, vol. xii., 626.

cord, an active hyperæmia followed by a passive hyperæmia, producing *œdema* of the cord, is the pathological condition in the affection which forms the subject of my paper. I have no personal data, so far as the temperature goes, for denying the inflammatory condition of the cornua. In other neuroses, even of a grave form, Hertz observes that he has never found the temperature elevated. "The pulse is normal or a little rapid, or, in many cases, a little below the average frequency."[1]

The spleen is usually enlarged, when it is enlarged, after repeated attacks of intermittent fever or at the close of a remittent fever. It sometimes regains its size when the patient moves to a non-malarial district. May not the spinal cord in like manner remain engorged so long as the patient is in a malarial atmosphere? There are many cases of paralysis depending on lesion in the anterior cornua, I am convinced, that recover completely. I have histories of several such cases with an undoubted malarial history, wherein the connection is close enough to fully warrant the relationship of cause and effect. A little while ago I spoke of intermittent paralysis being symmetrical. The cases I have just referred to are likewise symmetrical, but as the paralysis has occurred only once, I could not introduce them into this paper. Then, again, there are some cases when only a single member is affected, and I cannot better illustrate this than by reporting the following case of *spinal hemiplegia:*

Through the kindness of my friend, Dr. H. P. Geib, of Stamford, Ct., I saw, Nov. 20th, 1881, a child, Rosie C., aged twenty-one months, living within fifty yards of "the mill pond" of Stamford—a pond which has become quite notorious in the sanitary history of that town. During the drought of the past autumn, the doctor informs me, this pond was nearly dry, no water flowing over the dam, and out of forty houses within a radius of one

[1] Ziemssen, vol. ii., p. 622.

hundred yards of the stagnant water in question, thirty-eight contained unmistakable cases of malarial poisoning. In this family are three children, every one of whom, with the mother, suffered during the whole summer with malarial fever. For four years they have lived within this atmosphere, and the mother herself, during her last pregnancy—the one in which our patient took a conspicuous part—was suffering from the tertian variety. The babe suffered during its first year with symptoms always relieved by antiperiodics. In June of present year, it began to have distinct chills with fever following. These continued every second day until July 28th, when the left arm was found to hang limp by the side. The left lower extremity was powerless at the same time, and there was no facial disturbance. The child was not constipated, but, on the contrary, had a diarrhœa. Two days later, the fever assumed a remittent type, and the patient was very ill for two or three weeks, there being no apparent return of power in the superior or inferior extremity. The mother tells me that the child was able to use the fingers feebly, yet was not able to hold anything in the hand. She does not recollect whether there was any power in the toes or not. Dr. Geib saw the child only once, making a diagnosis of malarial paralysis. The case passed into other hands, and hence my history of its progress I obtained from the mother, who reports that during this period the child was excessively cross, but did not make any signs, such as using the other hand to rub the left arm or leg, which gave her the impression that the crying was due to pains in the affected members. About the first week in September, or nearly six weeks from the date when the arm first fell powerless by the side, it raised the arm and stood up, the limb trembling all the while. About this time the right hand was observed to tremble markedly when the baby attempted to use it, for instance, in holding a cup. The febrile symptoms have all abated, and convalescence from the paralysis is taking place slowly. When I examined the case to-day, I found a fairly-nourished child, unable to stand alone, but able to stand with very slight assistance, and able to walk across the floor holding on to the mother's hand, tottering like one drunk; both limbs trembled, yet this sign was more marked in the left, and there was a dragging of this foot. The little patient could execute all the movements of the shoulder quite readily, yet with feeble power, and the hand trembled markedly in attempting to hold a light weight. I could discover very little difference between the two hands, for the right one trembled likewise in the same test. The left inferior extremity

responded to all the voluntary tests of muscular functions. I could find no atrophy and no hyperæsthesia either of the four limbs or the spine. There was no splenic enlargement. I did not have an opportunity of making any electrical examination.

The family history is interesting.

The father has been for years an epileptic, and a victim to alcoholism. The mother is free from neuroses, though her mother was for several years before death hemiplegic. The eldest sister is now under treatment for vertebral caries.

At the meeting of the Neurological Society, December 6th, where I had the privilege of presenting the above cases for discussion, my object was gained, and my only regret was that on account of the lateness of the hour, I had not an opportunity of closing the discussion as is usual. Hence I supplement my paper now with a résumé, bearing in mind the points raised by the speakers on that occasion, and for the remarks then made, I must refer my readers to the *Medical Record* of Dec. 17th, 1881, pp. 694, 695.

For one I am not prepared to admit a poliomyelitis anterior acuta in either of the two cases I have reported, although topographically this seems to have been the lesion. In view of all the evidence which the literature of the disease furnishes to the contrary, it would be an astounding assumption, I think, in the absence of any post-mortem data.

That it was not an hysterical paralysis, as first occurred to my friend, Dr. Putnam-Jacobi, I need only quote Jolly[1] who, writing of this affection, says: "*The behavior of the paralyzed muscles to the electrical current is normal;* the excitability both for the induced and for the constant remains unaltered, even after a persistence of the paralysis for years."

Rheumatic paralysis, or paralysis *a frigore* was suggested by Dr. Putnam-Jacobi, and Dr. Seguin was "inclined to take the same view." The former, however, freely admitted that "the point against such a diagnosis was the elec-

[1] Ziemssen: Encyclopædia, N. Y., vol. xiv., p. 519.

trical reactions, which were not those of a functional paralysis." Niemeyer states that rheumatic palsy "does not present any peculiarity with regard to its extension, and for a differential diagnosis we can only avail ourselves of the fact that it is not apt to assume the form of hemiplegia or paraplegia."[1] While the peripheric nerves are the nerves usually attacked by cold, we do not have any law of symmetry observed. A double facial palsy, for instance, is a very rare occurrence, and the facial nerve is so frequently affected that I know I shall be pardoned for italicizing in the following quotation I make from Erb: "Rheumatic paralysis may arise in all parts of the nerves, and in all nerve regions, but, perhaps, most frequently attacks the peripheric nerves, some of them, indeed, as the *facial nerve*, with a marked preference."[2]

A distinguished medical friend, to whose opinion I always attach much weight, has suggested that my first patient may be one of those peculiarly susceptible to the influence of lead. The facts that he never had any symptoms pointing to lead colic, had no blue line on the gums, and had no arthropathies, furnish strong circumstantial evidence, at least, against such a theory.

Authorities say, Erb[3] and Niemeyer,[4] for instance, that this paralysis may be recognized with the greatest facility by the fact that very definite groups of muscles are successively attacked, the extensor muscles of the forearm, those supplied by the radial nerve, being first attacked. And then, too, sensory disturbances are as a rule entirely absent. Niemeyer[5] says that paralysis of the lower extremities when it does occur, takes place long after the paralysis of the hand and fingers. In my own cases re-

[1] Text-book of Practical Medicine, vol. ii., N. Y., 1876, p. 326.
[2] Ziemssen, vol. xi., p. 398.
[3] Ziemssen, vol. xi., p. 450.
[4] Loc. cit., vol. ii., p. 326.
[5] Ziemssen, vol. xvii., p. 576.

ported in this paper, and in those reported by Macario, Romberg, and Hartwig, the paralysis began first *in the lower extremities.* So that I think we have very little difficulty in excluding lead-paralysis.

I am willing, for the sake of argument, to admit (although such was not the fact) that the paralysis did not come on suddenly, to admit that it lasted for some time after *one or two distinctly tertian types* in the early history of the case, that it (the paralysis) did not terminate in sweating so far as my history goes (by some oversight I did not question the parents on this point), that the attacks were marked by sensory disturbances, inasmuch as sensory disturbances are recognized the world over as being the most common manifestations of malarial poisoning, and that the paralysis was not complete, in the sense of general; I am willing, I say, to admit all these five points made, and yet I am unwilling to admit that these attacks presented a single feature that would exclude them from the category of malarial attacks. Because an attack does not follow any distinct law of periodicity, it does not prove that it is not intermittent. In chronic malarial poisoning, the exacerbations may be such and their force may be so spent upon a special organ as to leave results that may persist for indefinite periods. The spleen may be subject to active hyperæmia so often that passive hyperæmia may be induced, the organ may remain engorged and crippled as to function long after the exacerbations have passed away, and a complete change of residence to a non-malarial locality may be necessary to restore the organ to its original size; and to object to calling an affection malarial because a pathological condition is induced, the nature of which we don't know, on the ground that this unknown condition somehow *produces* the symptoms in question, seems to me to be the limit of pathological refinement. We claim to look for causes of disease, and if a disease

can be proven to be malarial in origin, for instance, what a point, therapeutically, have we gained!

Even in the simplest forms of intermittent fever, the sweating stage is often absent or reduced to a minimum,[1] and in young children Hertz[2] says that this is never so general and profuse as in older children and adults. Masked intermittents prevail about NewYork; these have very irregular types and are above all others neurotic in their phenomena.

In conclusion, then, let me call attention to the necessity of differentiating between the curable paralyses of children and the incurable paralyses. Let me state, too, on the strength of cases that I have not reported in this paper, because they were not intermittent, or relapsing, if this term be preferable, that there may be spinal paralyses affecting one side or a single member, clearly traceable to malarial poisoning. They are not numerous, however, and the history will generally enable one to exclude a poliomyelitis anterior. If nervous diseases are increasing, as many authorities claim, may we not expect to find the cerebro-spinal axis the more readily and the more frequently influenced by malarial poisoning, just as we find now the nerves so commonly affected?

---

[1] Saint-Vel, Gazette hebdom., 1863, No. 13.

[2] Ziemssen, vol. ii., p. 597.