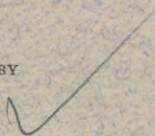


Seguin (E. C.)

MYELITIS FOLLOWING ACUTE ARSENICAL
POISONING (BY PARIS OR SCHWEIN-
FURTH GREEN.)

BY

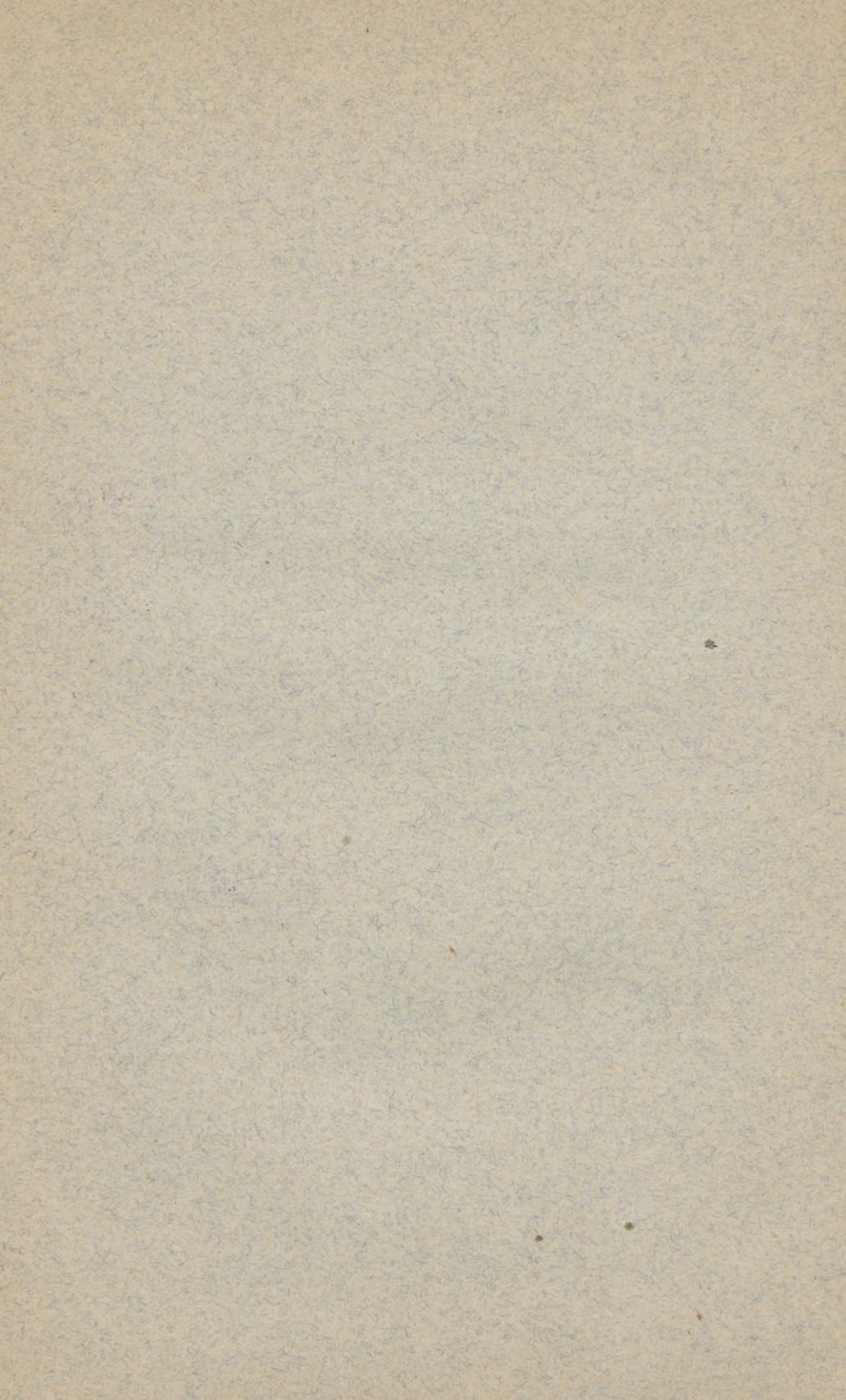


E. C. SEGUIN, M.D.

CORRESPONDING MEMBER OF THE VEREINS FÜR INNERE MEDICIN OF BERLIN, ETC.



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MYELITIS FOLLOWING ACUTE ARSENICAL
POISONING (BY PARIS OR SCHWEIN-
FURTH GREEN.)*

By E. C. SEGUIN, M.D.,

CORRESPONDING MEMBER OF THE VEREINS FÜR INNERE MEDICIN OF BERLIN, ETC.



THE physician who, meeting with a case of arsenical paralysis, would seek for information on the subject in the accessible and contemporary treatises upon disease of the nervous system, would be grievously disappointed. Such writers as Grasset, Ross, Wilks, Bauduy, Hamilton, do not mention the affection at all; the illustrious Romberg, and Erb merely give it a passing reference. Prof. Hammond (1881), in the last edition of his treatise, says nothing of paralysis following acute arsenical poisoning, and refers to paralysis and anæsthesia as results of slow poisoning. Apparently, he has seen no cases of arsenical paralysis. Rosenthal (1875) devotes only a short paragraph to arsenical nervous symptoms; refers to paralysis in the course of chronic poisoning. In a case which he saw there were paralysis, partial anæsthesia, and diminished elec-

* Read at the meeting of the Medical Section of the New York Academy of Medicine, October 17, 1882. (The original cases alone had already been read before the American Neurological Association, at its Eighth Annual Meeting, June 21, 1882.)

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tro-muscular contractility. Leyden (1875), in his classical work on diseases of the spinal cord,¹ gives a *résumé* chiefly after Leroy D'Étiolles. He does not appear to have had cases of his own, and considers the disease a neuritis.

A little more extended research in older books, and in periodicals brings to light numerous observations and some valuable experimental studies upon the subject.

Indeed, arsenical paralysis seems to have been very early noticed, and to have attracted considerable attention until within the last twenty years. As early as the thirteenth century, P. Abano² refers to paralysis and contractures after arsenical poisoning. These symptoms are also mentioned by Forestus³ (about 1560-70); and Zacchias⁴ (1630) mentions paralysis, spasms, contractures, and anæsthesia as following poisoning. From that time arsenical paralysis is frequently mentioned by medical writers. Hahnemann,⁵ in one of his earlier works (1786), relates several cases.

In 1812, Sir Benjamin Brodie, in an interesting communication to the London Royal Society, entitled "Observations and experiments on the actions of poisons on the animal system," devotes a section to the effects of arsenic, and relates how in several of his animals (rabbits and dogs) the hinder extremities became paralyzed. He considered the brain to be affected in these cases.

The following interesting case was published, in 1809, by Dr. G. Thilenius.⁶

A young lady having observed a hard lump in her left breast, neglected it until the ensuing spring, when it became very painful. A miserably ignorant barber who was consulted, applied a prepa-

¹ Klinik der Rückenmarks-Krankheiten, Bd. ii, p. 296.

² De venenis eorumque remediis, cited by Imbert-Gourbeyre, *l. c.*

³ Cited by Imbert-Gourbeyre, *l. c.*

⁴ Quæstiones medico-legales, Romæ, 1621-50, cited by Imbert-Gourbeyre.

⁵ Ueber das Arsenik-Vergiftung, Leipzig, 1786, cited by Imbert-Gourbeyre.

⁶ Medic.-chirurgische Bemerkungen, Frankfurt, 1809, in Leroy D'Étiolles, p. 63.

ration of arsenic. This was followed by ulceration and increased pain, and, according to the father's statement, three days later her arms and legs became insensible, and so much paralyzed that she could neither walk nor feed herself. The limbs were also cold. In the course of two months the arms recovered, and the legs improved steadily. Electricity was used; the tumor removed by the knife. At various times there occurred prickling and jerking in the legs. Anæsthesia and atrophy not mentioned.

In about a year after the attack the patient was able to walk without a cane; her limbs were warm, and the wound in the breast well healed.

In 1793 (three years after the attack) patient was perfectly well, married, and had a child.—(Obs. No. 80 of Leroy D' Étioilles.)

Orfila,¹ the great French chemist and toxicologist, in experiments upon dogs, made prior to 1840, noticed paralysis of the hinder extremities in dogs which survived arsenical poisoning (also in fatal experiments).

Prof. Christison,² of Edinburgh, in his classical work on poisons, treats of symptoms of arsenical poisoning in a masterly way. He makes three categories of cases of arsenical poisoning. In a first class of cases, in which, with symptoms of violent inflammation of the gastro-intestinal tract, death results in from twenty-four hours to three days; nervous symptoms not present. In a second class of cases, with little evidence of inflammation, extreme prostration and syncope are the chief symptoms, death occurring within six hours; no paralysis observed. Convulsions may close the scene. In the third category, that of subacute cases, there is moderate gastro-intestinal inflammation; symptoms are same as in other classes, but milder. In the later stage these cases are apt to show marked nervous symptoms: coma, epileptoid attacks, mania, tetanus, hysterical seizures, partial paralysis resembling lead paralysis in affecting the extremities; contractures may exist. In speaking of symptoms con-

¹ *Traité de Toxicologie*, Paris, 1852.

² *A Treatise on Poisons*, Phila., 1845, p. 244, *et seq.*

nected with irritation of the *primæ vivæ*, Christison makes this shrewd remark, which applies critically to many of the older cases of arsenical paralysis: "Cramps in the legs and arms (occur in arsenical poisoning), a possible concomitant of every kind of diarrhœa."

The father of modern clinical medicine, Graves¹ (1842), after speaking of paraplegia from inflammation of the bowels, refers to Orfila's experiments in which all (?) the dogs which survived arsenical poisoning were paralyzed in their hinder limbs, and states that in his opinion in cases of arsenical as well as of lead poisoning, the poison acts directly on the central nervous system (spinal cord), and that the palsy is not due to the intestinal irritation.

Huss,² of Stockholm, in his work on alcoholism (1852), mentions several cases of arsenical poisoning with severe nervous symptoms. He gives one which is instructive as regards its etiology.

For the cure of intermittent fever, a large teaspoonful of Fowler's solution was given at one dose (equivalent to $\frac{7}{12}$ grain, or .035 gramme, of arsenious acid). After the usual symptoms of acute intoxication, there gradually ensued an almost complete paralysis of the extremities, with anæsthesia of the hands and feet, severe pains and cramps in the lumbar region and lower extremities.

In 1857 we meet with quite an important contribution to this subject. Leroy D'Étiolles,³ in his work on paralysis of the lower limbs, devotes a chapter to arsenical paralysis, and relates the following three cases (in addition to the case of Thilenius already quoted).

Obs. 79.—Poisoning from external application of arsenious acid; general paralysis; recovery of upper extremities first.

¹ Clinical Lectures, Gerhard's edition, Phila., 1842, p. 94.

² Cited by Imbert-Gourbeyre.

³ Des paralysies des membres inférieurs, deuxième partie, p. 28, *et seq.*, Paris, 1857.

Male patient . . . age. Dr. Trochon, of the hospital at Pornic, amputated his leg for cancer. In the cicatrix cancerous buds appeared. Arsenical paste, made one hundred times too strong by druggist's error, was applied, and very soon symptoms of acute intoxication appeared ; life saved with difficulty. At the end of ten days patient convalescent, but with well-marked paraplegia and paresis of the arms.

Seen five months later by Leroy : Arms weak and not adroit ; tendency to drop-wrist. Marked paralysis in remaining lower extremity, with emaciation, but not positive atrophy of muscles ; contracture in semi-flexion ; foot hyperextended (pes equinus) ; toes flexed. Sensibility to touch and pain much impaired on limbs. Electrical tests not used.

Gradual improvement of paralysis in spite of progressive cancerous infection.

This case bears a certain resemblance to my own cases. Although it is stated that the muscles were not atrophied as in lead paralysis, yet from the contractures and the degree of emaciation present, it seems to me highly probable that there was atrophy, widely distributed, as in mild cases of poliomyelitis. It is a pity that electricity was not used, although at the time when this observation was recorded, 1855, only the bare fact of diminution or loss of faradic contractility could have been determined.

The contracture in flexion with pes equinus is strikingly like what existed in my own Case 3.

Obs. 81.—Poisoning by the ingestion of arsenious acid : paresis of arms ; paraplegia lasting fourteen months.

A female patient aged thirty-seven years, was admitted to the service of Dr. Bouvier, Hospital Beaujon, January 22, 1850, suffering from severe toxic symptoms produced by eating cakes charged with arsenious acid.

As soon as the urgent symptoms had subsided (time not noted), it was discovered that the patient was paralyzed in her lower limbs, and that they were the seat of painful jerking (reflex movements?) Her arms were weak. On 18th February, on leaving the hospital, she was unable to stand, and said that she could not feel the floor under her feet. In September of the same year she

was readmitted with pleurisy; and it was noted that while her arms had recovered, her legs were just as weak, and as insensible to touch. At no time was there interference with the functions of the rectum or bladder. Later some improvement took place, but the patient finally died of exhaustion caused by a profuse diarrhoea. No autopsy.

Obs. 82.—Case of Aran in *Union Médicale*, July 6, 1852. On June 9, M. Aran presented to the Société Médicale des Hôpitaux, one of two young men who had, two months previously, been poisoned by arseniate of sodium. The victims had swallowed this salt, supposing it to be tartrate of sodium. One died in twenty-four hours; and a lady to whom they had given some of the poison, is not yet perfectly well.

In the surviving male patient interesting nervous symptoms have appeared. In about fifteen days after the ingestion of the drug, symptoms of paralysis appeared in the lower limbs, more marked in the right leg. The upper extremities have also been weak. The paralysis has remained very much in *statu quo*. The paralyzed parts are somewhat anæsthetic. The lower limbs are the seat of tingling below the knees; and the upper extremities in the finger-tips. At one time the parietic extremities showed diminished calorification. General health good.

M. Duchenne examined the young man and found slight diminution of electrical irritability, and the skin showed diminished sensibility to the current.

Later, on 8th September, M. Aran reported to the Society that the patient had recovered, apparently in consequence of forty-six baths and forty-six douches at Bagnères de Luchon (hot sulphur springs). Improvement showed itself distinctly after the thirty-sixth bath.

Leroy makes these general statements: In lead paralysis the forearms are usually affected (sometimes only one); arsenical paralysis tends to involve all the limbs; the lower limbs are more affected; often there is well marked paraplegia; the action of the bladder remains normal. Sensibility is usually much impaired (nearly as much as motility). He refers to wasting of muscles, but states that it contrasts with the positive atrophy of lead paralysis. Electro-muscular contractility persists, but is diminished. Treatment is

efficacious, and the duration of the paralysis is usually less than one year.

Shortly after the appearance of Leroy D'Étiolles' work, a learned French physician, Imbert-Gourbeyre,¹ professor at the medical school of Clermont-Ferrand, published a series of articles in the *Gazette Médicale* (1858), in which he gave an elaborate account of our previous knowledge of arsenical paralysis. I am indebted to this essay for bibliographical data. The articles contain nothing original. In 1863, Smoler² published a case of paralysis after acute arsenical paralysis, which is referred to by Rosenthal (1875).

Jaccoud³ (1864) devotes several paragraphs to arsenical paralysis (paraplegia), and expresses his belief that the palsy is caused by the direct action of the metal or its compounds upon the tissue of the spinal cord. He does not, however, appear to have seen a case.

In 1881, Seeligmüller⁴ placed on record four cases; two after acute poisoning, and two after chronic intoxication. In his acute cases he noted paralysis, numbness, and anæsthesia (in toes), contractures, wasting of the extensors especially. The paretic and wasted muscles showed fibrillary contractions; the nails were gradually lost. Electro-muscular contractility was diminished or even lost.

He gives the following points for differential diagnosis from lead palsy: the acute origin of the paralysis, disorder of sensation as well as of motion, rapid muscular wasting, absence of blue line on the gums, and of cachexia.

In the same year appeared the essay of Popow,⁵ of St.

¹ Études sur la paralysie arsénicale. *Gazette Médicale*, 1858, pp. 5, 19, 59, 94.

² Lähmung nach Arsenikvergiftung. *Österreich Zeitschr. für pract. Heilkunde*, 1863.

³ Les paraplégies et l'ataxie du mouvement, Paris, 1864, p. 323, *et seq.*

⁴ Ueber Arseniklähmung. *Deutsche med. Wochenschrift*, 1881, No. 14, *et seq.*

⁵ Ueber die Veränderungen im Rückenmarke nach Vergiftung mit Arsen und Blei. *St. Petersburger med. Wochenschrift*, 1881, No. 36.

Petersburg, upon the pathological anatomy of arsenical paralysis as produced artificially in animals. Popow carried on his experiments under the guidance of Prof. Mierzejewski; giving arsenious acid to dogs in doses ranging from $\frac{1}{10}$ to 2 grains at a dose, producing acute and chronic intoxication.

In cases where death ensued in four to five hours after ingestion of the poison, the spinal cord showed both macroscopic and microscopic lesions. The gray matter appeared swollen, intensely red, more especially in its two enlargements. Microscopic examination revealed enlargement and congestion of the small blood-vessels, and accumulations of lymph corpuscles in the lymph-spaces.

There were also abundant extravasations of blood-corpuscles and plasma around the vessels, especially in the central portions of the gray matter. The walls of the blood-vessels were in a state of fatty degeneration.

The ganglion cells exhibited three degrees of change. A first degree of alteration showed cells well stained by carmine, and containing vacuoles of variable sizes, some of which could be traced into the cell-processes. A second form of cells had no processes, were feebly colored by carmine and exhibited a punctate granular infiltration. Lastly, here and there were cells in a third state of change, consisting only of a nucleus surrounded by dark brick-red pigment. The white substance only showed pigment masses here and there, more especially about the blood-vessels.

In cases of acute intoxication in which a fatal result ensued in the course of three, five, or six days, the spinal cord presented very much the same appearances. The distinction between the white and gray substances was less defined. The vascular injection and the exudation of plasma were less marked, but on the other hand the changes in the ganglion cells were more distinct, the

vacuoles larger, and the granular state more pronounced. There were more cells, or properly remains of cells, of the third category above described. The white substance was normal, except some enlargement of blood-vessels, and considerable accumulations of pigment.

In the chronic cases, those in which death occurred in the course of three months (one animal had paresis of the hind legs not long before death), the spinal cord appeared less firm, and the microscopic appearances differed noticeably from those observed in the acute cases.

The walls of the blood-vessels were much thickened, and showed a distinct fibrillary structure, with diminution of the calibre of the vessels, and exudation of blood in the perivascular spaces. In the meshes of the perivascular spaces were extensive hyaloid masses. The number of ganglion cells was much diminished; those remaining showed large vacuoles, and belonged to the first group described. In these cases the white substance was much more affected, especially in the postero-lateral columns. The cylinder-axes exhibited points of swelling here and there; they were granular; in many preparations they were merely represented by groups of fine granulations. The septa of the white substance likewise exhibited a granular change; and the periphery of the white and gray substances was thickly strewn with small masses of black pigment.

The spinal nerves, carefully examined at their origin, and at various points of their course and distribution, presented no pathological alterations.

From these *post-mortem* observations Dr. Popow concludes that:

1. Arsenic, even in a few hours after its ingestion, may cause distinct lesions of the spinal cord, of the type known as acute central myelitis, or acute poliomyelitis.

2. In the more chronic cases the pathological changes are found in the white as well as in the gray substance, constituting a diffused myelitis.

3. The peripheral nerves remain normal, even three months after intoxication.

4. The paralysis of arsenical poisoning is of central origin.

It might be added that in three guinea-pigs poisoned by lead, and dying on the sixth, seventh, and tenth days, similar lesions were found, *i. e.*, evidences of more or less diffused myelitis, and no lesions of peripheral nerves.

This essay, issued under the supervision of so distinguished a neurologist and microscopist as Prof. Mierzejewski, is in many respects the most important contribution to the subject. Connecting its conclusions with inductions which can be legitimately drawn from the cases of Leroy, Seeligmüller, Smoler, and Rosenthal, and my own, we are able, I think, to form a definite conception of the true nature and relations of arsenical paralysis.

In last year's *Philadelphia Medical Times* Prof. J. M. Da Costa¹ relates a case of subacute myelitis which occurred in a man who had been taking "small pinches" of arsenic (arsenious acid?) for three months. The general features of this case and the paralytic phenomena are so unlike what has been observed in the other cases referred to in this paper, that I entertain a doubt as to its having been an "arsenical paralysis." The rapid improvement under very large doses of iodide of potassium, and the history of a venereal sore one year before admission, would seem to furnish a better clue to the nature of the myelitis.

The cases which have fallen under my own observation are three in number. The subjects were all would-be

¹ Clinical Lecture on Arsenical Paralysis, *Phila. Med Times*, March, 1881.

suicides with Paris green,¹ and they presented remarkably similar symptoms. In many respects the cases resembled those already related.

CASE I.—Samuel L., hostler, seen March 21, 1879, in consultation with Dr. M. Burke.

At the end of January, while in good health, swallowed a large quantity of pulverized Paris green. Had much difficulty in swallowing it, and very soon was led to a drug store, where emetics were given; and later he was taken to Bellevue Hospital, where the stomach-pump was thoroughly used. Vomiting, gastric pain and irritation, extreme prostration, lasted four or five days.

Soon after he began to go about his room, he noticed numbness in his fingers and hands, followed in two or three days by similar sensations in his feet. Paresis appeared about the same time in all the extremities, and had steadily progressed to extreme paralysis below the knees, with wasting of the muscles there. Has had much burning, gnawing pain in soles and insteps; a little in the hands. Ten days ago became unable to stand. No cerebral symptoms, or palsy of bladder, or jerking of legs.

Examination.—Hands and forearms only weak; no positive paralysis or atrophy; no anæsthesia.

Legs completely paralyzed below the knees; cannot move feet or toes. Thigh muscles are weak. Marked atrophy of calves and of anterior tibial muscles. No anæsthesia of soles, unless it be a slight tactile dulness.

Test with faradic current; no reaction in right leg, nerves, or muscles. In left leg no reaction in anterior tibial muscles or nerves, but a feeble contraction can be produced in the calf.

Patient is at times hysterical.

At fifteen had a chancre, not followed by secondary symptoms.

I have no further notes of the case; but some sort of galvanic treatment was carried out. A few months afterward I learned that the patient was well, and some time in the winter of 1880-81 he came to my office and exhibited a vigorous pair of legs. He

¹ Paris or Schweinfurth green is a compound substance which is best designated as aceto-arsenite of copper.

In looking up this point I was astonished to find that such a popular and so constantly used a term as Paris green, was not to be found in the indexes of any of our dispensatories, treatises on materia medica, and, stranger still, not in works on toxicology.

Prof. Chas. F. Chandler, in reply to a note, very kindly gave me all necessary chemical information on the subject.

had completely recovered and was at work again as a hostler at Jerome Park.

CASE 2.—Mary N., aged sixteen years, was admitted to the New York Hospital on December 11, 1878, in the service of Dr. Woolsey Johnson. To Dr. R. W. Amidon, then house physician of the hospital, I am indebted for notes of the case and for the opportunity of studying the case in its later stages. Dr. Johnson has kindly given me permission to use the case.

The patient was a strong, rosy-cheeked girl of German parentage. She had never suffered from rheumatism or malaria. Thirteen days before admission she swallowed five cents' worth of Paris green. In five minutes she vomited, and after an emetic had been given she vomited again, rejecting all (?) that had been swallowed. Probably had some gastro-enteritis, as she vomited and was purged for two or three days.

It is reported (by patient and her friends) that on the first night she had fever. Second day, no fever or pain. Third day, at 3 P.M., had fever for one hour and a half; burning pain in toes; hands felt stiff. On the fifth day, at 9 A.M., fever returned, with slight headache, but no chill. The burning pain extended up to the knees. One week after taking the poison her legs became stiff, and she lost power in her arms; had "cramps" in her hands. These symptoms continued, but the headache ceased; tried to walk, but found that she was partially paralyzed in her legs; needed help to walk, and suffered pain in her knees (in the attempt). Three days later (tenth day) loss of power increased; had cramps in hands; had tightening sensations in hands and feet, and they began to peel and showed a mottled red and white appearance. Bowels and bladder normal.

Has not been unwell for twelve weeks; previously regular.

Condition on admission, thirteen days after taking poison: Patient complains only of headache, and of inability to walk, because "cords of knees are stiff." Appetite, bowels, bladder, and eyes normal.

Hands are cold and moist. The extensor muscles of both hands are weak, those of right hand weaker. Some twitching of long flexors and of interossei.

The skin is lax. The small muscles of the region of the right little finger are completely paralyzed and wasted. The right thenar eminence is smaller than it should be. The hypothenar group on the left side is in the same condition. Grasp very weak. On dynamometer each hand shows about 20° (on outer circle).

There is hyperidrosis. No anæsthesia is present, but she complains of a burning pain when pricked with a pin.

The legs are semi-flexed, showing mostly palsy of the extensors of both feet. The left foot is more inverted than the right. Legs and thighs smaller and colder than normal. Toes are red; the circulation is sluggish. The muscles of the legs are not flabby, but the anterior tibial regions are flattened. Hamstrings rigid on both sides.

Circumference of right thigh, 30.5; right leg, 23.5.
 " " left " 30.5; left " 24.5.

There is complete paralysis of the anterior tibial muscles. The peroneal and posterior tibial groups are somewhat atrophied and paretic. Great toes are motionless. Flexion of thighs is moderately good; extension complete. No increased reflex actions. Is rather hyperæsthetic (in legs).

Dec. 14th. A tendency to retention of urine is noted (but is not again referred to). Examination with the faradic current showed good contractions in left thenar and hypothenar eminences, but none in the right. In the right leg there is slight reaction in the anterior tibial muscles; none in the peronei.

Dec. 26th. It is noted that there is no faradic reaction in the anterior tibials and peronei. Sensibility is good. Hamstrings less rigid. Patient has plaster apparatus for legs, and the application of the faradic current.

Jan. 11, 1879. Walks with some support, and has done so for a week. Left leg nearly straight.

Jan. 15th. Circumference of right thigh, 34.0; right leg, 24.
 " " left " 34.5; left " 26.

Toes always cold and moist; tender to slight pressure. Less contraction of hamstrings. No reflex actions in legs. Interossei of hands do not improve, and remain as flaccid, atrophied, and weak as on admission (faradism not used on upper extremities).

Jan. 24th. Galvanism tried for first time; ten cells cause contraction of tibialis and peronei; eight cells (Stohrer battery) cause contraction of quadriceps, sartorius, and muscles of calf. Some atrophy of extensors of the right forearm and hand; good reactions (current not stated) in ulnar distribution, but not in wasted extensors. On the left side good reaction in forearm and hand, except abductor pollicis, to both galvanism and faradism.

Jan. 27th. Menses appeared with great pain.

Feb. 4th. Electrical applications omitted because of *malaise*. Patient doing nicely; muscles react with small amounts of electricity.

Feb. 9th. Some trophic changes in feet; nail of big toe coming off. The skin is rough, and there is vaso-motor disturbance. Reactions improving (in legs).

March 10th. Walks quite well. Some remaining weakness of anterior tibial muscles.

Discharged improved.

This patient at once began to be treated as an out-patient at the Manhattan Hospital, and after several weeks of treatment by galvanism mostly was completely cured.

The notes taken at this time have been misplaced, but our recollection is clear that her upper extremities were about well, though perspiring, and that the lower limbs exhibited paresis, a sluggish circulation, and a peculiar sensitiveness and tenderness. She was able to walk alone, but lame. Her general health was quite good.

It seems certain that this was a case of subacute polio-myelitis chiefly. The inflammatory action must have extended to deeper parts of the gray matter, as shown by continued hyperalgesia and by the contractures.

CASE 3.—Ellen R., aged twenty-six years, admitted to Manhattan Hospital May 10, 1881. In September, 1880, took a large dose of Paris green. Was exceedingly ill; vomiting and diarrhœa. In a week nearly complete paralysis developed. Legs completely paralyzed; forearms the same.

A gradual recovery began in the course of a few weeks (no treatment).

Three weeks before admission to the hospital, Dr. J. B. Emerson, who visited her in the country, found her fingers and the soles of her feet nearly insensible to pricking. I examined the patient May 9, 1881, and the following notes were taken. Can walk with a little aid, impeded by moderate contracture of right knee, and tenderness of feet. No voluntary power (motion) below ankles. Complete anæsthesia to contact on soles of feet and on finger-tips. Feels cold and heat, however, and pricking quite well. The upper extremities simply present a slight paresis with moderate wasting of the hand muscles, some interossei quite

wasted, and some large fibrillary movements in the same. No cutaneous trophic changes. Thighs moderately wasted, with some contracture of right hamstrings. No patellar tendon reflex. Calves and anterior tibial muscles are much wasted; legs and feet bluish and cold; slight tactile anæsthesia of feet.

Patient is thin and in poor health; has been using an unknown quantity of morphia.

She was ordered a mixture containing diminishing amounts of morphia; and Dr. Adam, assistant physician of the hospital, applied galvanism and faradism very faithfully to her for weeks. He also gave her passive movements and massage. The improvement was steady, and in a few weeks patient left the hospital almost perfectly cured as regards paralysis, and in good general health. She was forty-eight days in the hospital. The day after admission Dr. Adam made a thorough testing of the affected muscles with the galvanic and faradic currents, which may be summed up by saying that most of the paralyzed parts exhibited the degeneration reactions, viz.: 1, diminished or wholly lost faradic reaction in muscles and nerves; 2, sluggish contractions to galvanism, with $\text{ancc} = \text{cacc}$ in many muscles, and $\text{ancc} > \text{cacc}$ in some. For example, in the muscles of the legs below knees a very strong faradic current caused no reaction. In the right gastrocnemius $\text{ancc} > \text{cacc}$. In other muscles $\text{cacc} = \text{ancc}$. In some interossei of hands $\text{ancc} > \text{cacc}$.

This patient is again under my care at the Manhattan Eye and Ear Hospital (October, 1882), for the cure of the only remaining weakness, viz.: paralysis of both anterior tibial muscles causing pes valgus. This is the only muscle which does not respond to the will, but all the muscles of the legs show a most astonishing quantitative reduction in electrical reactions: no reaction in muscles or nerves to full strength of faradic secondary current, and few small reactions in nerves and muscles to fifty good Leclanché elements. Reactions obtained are of normal quality.

In this case, besides the contracture of the hamstrings, as in Case 2, we have distinct though slight anæsthesia to indicate a certain extension backward of the lesion, in the spinal gray matter.

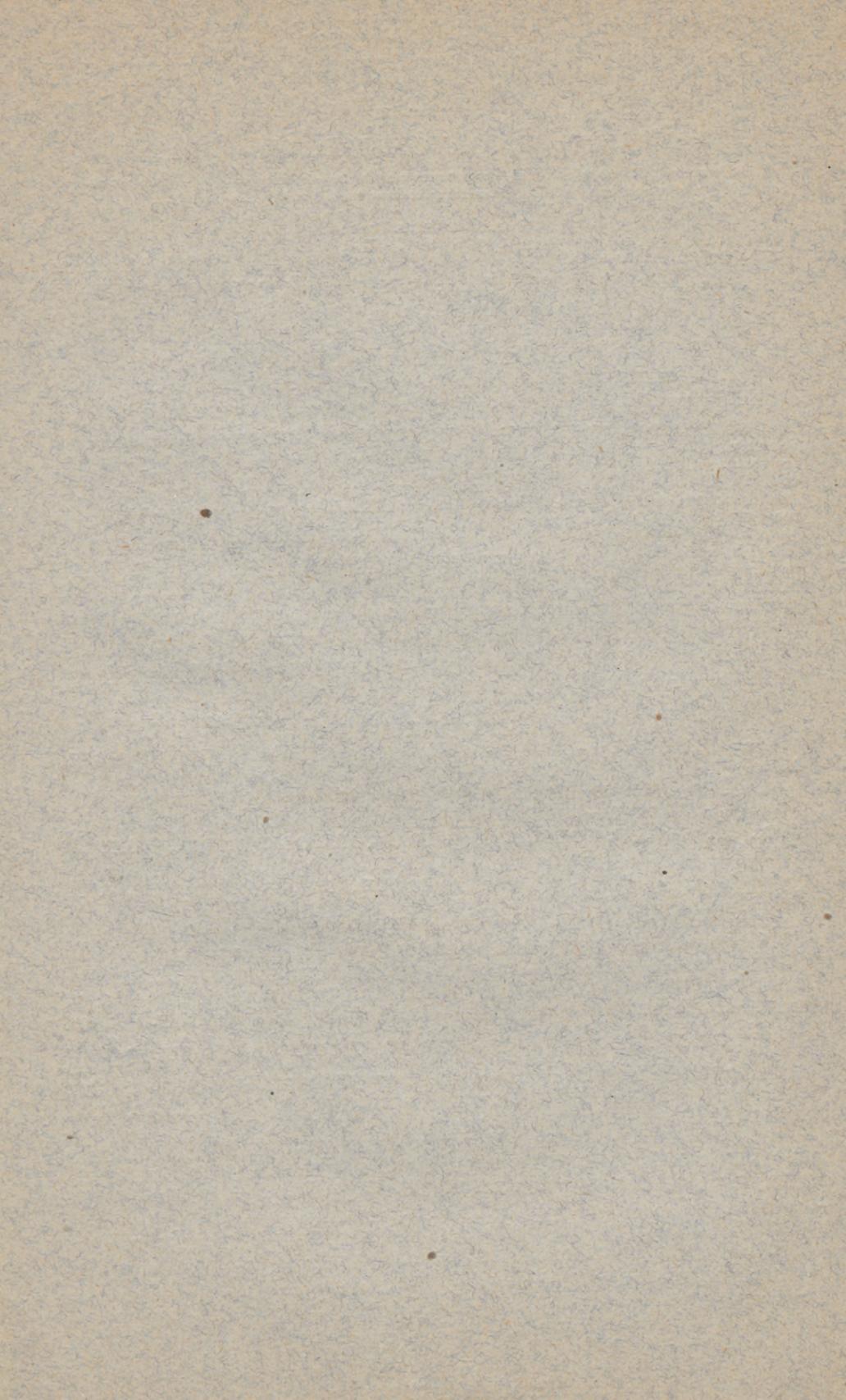
To sum up, these three cases presented evidences of slight subacute, diffused myelitis, more distributed in the

anterior cornua. In Case 1 the symptoms were more purely those of poliomyelitis.

In all cases the symptoms of myelitis followed within a week after the ingestion of the poison.

If we compare the symptoms present in the various human cases related and quoted, and the pathological appearances found by Popow in his animals, it is, it seems to me, legitimate to reach the following conclusions:

1. Arsenical paralysis is the expression of a myelitis.
2. This myelitis approximates the type known as poliomyelitis in so far as the symptoms are chiefly motor; that the paralyzed muscles undergo some atrophy, and exhibit the degeneration reactions to electrical currents; that the bladder is never palsied; and that in animals the ganglion cells of the anterior horns are extensively diseased.
3. There is usually more than poliomyelitis, as shown by Popow's *post-mortem* findings, and by the presence in living human subjects of pains in the nerves and muscles of the affected limbs, and by the occurrence of actual anæsthesia.
4. Consequently it might be better to speak of arsenical paralysis as due to diffused central myelitis with special involvement of the anterior gray matter.
5. Whether this myelitis is strictly arsenical, *i. e.*, caused by the direct effect of the arsenic on the tissue of the spinal cord, or whether it is produced (as are many forms of myelitis) by the irritation of peripheral nerves (cutaneous, intestinal and gastric nerve-endings), is a question which cannot at present be definitely solved, but which presents an interesting field for future research and speculation.



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EDITED BY

WILLIAM J. MORTON, M.D., NEW YORK.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM, UNIVERSITY OF VERMONT; PHYSICIAN TO
DEPARTMENT OF NERVOUS DISEASES, METROPOLITAN THROAT HOSPITAL, N. Y.; ETC., ETC.

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