

under similar circumstances. In the next stage the thick layer of connective tissue in the intima renders the vessel fully able to withstand momentary increments of pressure from within.

It seems probable, therefore, that the arterio-sclerotic aneurism develops during the first stage of arterio-sclerosis. Thoma tells us that at this time the wall of the artery is so far weakened that it is perfectly easy to produce artificial aneurisms in the vessel removed from the body during life or after death by a pressure of from twelve to twenty-four centimetres of mercury. This cannot be done either with normal arteries or with such as present an advanced degree of arterio-sclerosis.

It is of interest to know that the early stage of this lesion, the time when the arteries are easily dilatable, is supposed to last only a year. As a result of experiment and calculation, the inference has been drawn that a year after the beginning of the disease in a vessel, the intima is to such an extent re-enforced by newly formed connective-tissue that liability to formation of aneurism may be excluded.

It should be remarked that this period of time applies, not to the whole arterial system (in Thoma's calculations), but to each diseased vessel. That is to say, the various arteries may become diseased in succession throughout the body; and from the beginning of the development of weakness in the muscular coat in the case of each vessel, a year must be allowed to elapse before liability to aneurism in that particular artery has passed.

"Every individual who is the subject of arterio-sclerosis is thus for a year exposed to the danger of aneurism in all parts of his arterial system. This period occurs chiefly between the ages of thirty-five and forty-five years." We should probably want stronger evidence of the truth of such exact inferences than seems to be forthcoming before we accepted them.

It is of great clinical interest that this weakness of the arterial wall which marks<sup>21</sup> the early stages of arterio-sclerosis can be diagnosticated by ophthalmoscopic examination. The evidences of its presence are said to be tortuosity of the arteries, pulsation and locomotion visible in them (without other explanation, such as chlorosis, anæmia, neurasthenia), opacities in their walls, often a diminished lumen, hyaline degeneration of some vessels of papilla, occasionally aneurism of the central artery, and oftener varicose veins. Complete obliteration of an artery has been seen, and thickening of the walls of veins with partial obliteration of their lumina.

It is altogether likely that changes like these in the central artery of the retina, and in the ophthalmic artery, are associated with changes in the internal carotid. It is believed that the rest of the arterial system may be intact and yet serious changes show themselves in the carotid and its branches.

(To be continued.)

**QUARANTINE IN THE SUEZ CANAL.**—An international conference is to assemble shortly at Venice, in order to deliberate, at the instigation of Austria-Hungary, on the subject of the passage of the Suez Canal by vessels in so-called "quarantine."

<sup>21</sup> Thoma: *Deutsche med. Wochenschrift*, 1889, No. 18, p. 362, and *Archiv für ophthalmoscopie*, xxv, 1889. Ruchmann: *Zelstsch. f. kl. med.*, xvi, 1889, 506.

## LEAD PARALYSIS.

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IN 1882,<sup>1</sup> I published several cases of lead-poisoning in which the paralysis was limited to or most marked in the legs, the symptoms being due probably to a peripheral neuritis. During the last few years several other similar cases have come under my notice. In that article I reviewed at length the results of autopsies and the views of observers in regard to the seat of the lesion in such cases, as published up to that time.

The view that the muscles are chiefly affected has found but little support, Friedländer being the principal advocate of this localization.

Erb, E. Remak, Eulenberg, Monakow, Bernhardt, de Watteville, Birdsall, favor the view that the spinal cord or brain are the parts primarily affected. Westphal, Leyden, Zunker, Gombault, Charcot favor the view that the lesion is primarily a neuritis. Not all these authors have been able to examine the nerves and nerve-centres in cases of lead paralysis.

Since the publication of the previous paper on this subject there have been several autopsies published.

Wynter<sup>2</sup> found lead in the brain, in case of lead-poisoning.

Oppenheim<sup>3</sup> found the ganglion cells of the spinal cord diseased. While the white substance was normal the gray substance of the anterior columns (only in a doubtful degree of the posterior column) was diseased throughout nearly the entire cord. The chief centre of the diseased changes was the cervical and lumbar enlargements, while above and below these the intensity of the changes diminished. He draws the inference that these cells must have been disturbed in their function long before they showed any material changes which could be recognized.

Schultze<sup>4</sup> found the cord normal. He found, on the other hand, an atrophy and destruction of most of the nerve fibres in the trunk of the radial, below where the branch for the supinator longus is given off. Above, the change was less, so that just below the plexus there were no clearly abnormal fibres.

Schultze thinks that the pathological changes are to be found in the nerves primarily, that in generalized lead paralysis a central lesion in the anterior cornua may be added to this.

Osw. Vierordt<sup>5</sup> found in one case no change in the spinal cord nor nerve roots; especially was there no change in the cervical portion of the cord.

"An undoubted pathological change is nowhere found in the spinal cord and its roots."

There was extensive degeneration of the right radial and median nerves, less marked of the ulnar nerve.

He considers that there is no analogy between the pathological changes found in lead paralysis and those found in infantile paralysis.

Alexander Westphal<sup>6</sup> reports thirteen cases of saturnine encephalopathy with two autopsies. His conclusions are:

(1) That there is a direct influence of lead upon the brain. This toxic influence is manifested by general cerebral symptoms, as well as by focal symptoms. Especially are to be counted among these that peculiar neurosis, which is accompanied with hemianæsthesia

<sup>1</sup> *Archives of Medicine*, vol. viii, August, 1882.

<sup>2</sup> *Journal of Mental Science*, January 1888, p. 483.

<sup>3</sup> *Arch. f. Psych. und Nervenk.*, xvi, 1886, p. 476.

<sup>4</sup> *Ibid.*, xvi, 1886, p. 791.

<sup>5</sup> *Ibid.*, xviii, 1887, p. 48.

<sup>6</sup> *Ibid.*, xix, 1888, p. 620.

and psychic disturbances. At the same time single cerebral nerves (especially the optic) may be subject to anatomical changes from the poison.

(2) There is an influence of lead upon the cerebral vessels, and thereby is produced arteritis and its consequences (hæmorrhage, encephalomalacia).

(3) Influences of lead upon the kidneys with uræmic cerebral symptoms.

(4) Finally these causes may be combined together.

It would seem from the results found in the majority of autopsies that neuritis is the most common lesion in these cases, but it is not the only change due to lead. Yet it is not in accordance with what we know of neuritis to have in it exaggerated deep reflexes and ankle clonus. The cases in which the symptoms of lateral sclerosis were prominent could not have been cases of neuritis. Thus:

Cases I and III were clinically typical cases of sclerosis of the lateral columns except the anæsthesia in Case I.

Case II had some of the symptoms of lateral sclerosis, but was less simple, and with these there were cerebral symptoms.

Case IV had the exaggerated deep reflexes, but otherwise did not resemble lateral sclerosis.

Cases V and VII were typical neuritis.

Case VI had some spasmodic action of the legs, which is less commonly seen in neuritis, and the cerebral symptoms are rare in neuritis. Otherwise the symptoms pointed to the nerves as the seat of the lesion.

Clinically, then, the symptoms, in some of these cases, would indicate that the lesion was in the nerves; in other cases, that the lateral columns of the cord were chiefly affected. In others it would seem as though the nerves or cord and the brain were both affected together. These might be considered mixed cases.

As might be expected, judging from the differences of opinion as formed from the results of post-mortem examinations, the analysis of the clinical features of these few cases would lead to the conclusion that no one nervous tissue is exclusively affected, but that all may be subject to changes due to the poison of lead.

*Case I. Anæsthesia, partial analgesia of legs, exaggerated reflexes, loss of motion, contracting of legs, lead in urine, cerebral symptoms, — recovery, relapse.*

Miss A. G., age nineteen, student, was admitted to the Adams Nervine Asylum, October, 1885; for nearly a year she had had a tired feeling, after that a numbness in her limbs and a feeling of weakness, no headache nor pain. She was obliged to leave school in April on account of this general loss of strength and feebleness in her legs. There were no other symptoms at that time.

There was found marked anæsthesia on both sides as high as the umbilicus; partial analgesia to the same height; muscular sense or the sense of position much diminished, especially on the right. Plantar reflex more marked on left; patellar tendon reflex strong on both sides; ankle clonus strongly marked on both sides; a light patellar clonus on the left, not on the right.

The legs were stiff; it was not easy to move them passively. During the night the knees were drawn up, and in the morning the patient had considerable difficulty in straightening the limbs on account of the stiffness, worse on the right. The knees and ankles had been swollen; the hands and fingers had not been. There was a slight uncertainty in the motion of the

hands with the eyes shut. The patient was unable to stand; she could sit in a chair if well supported; she could not move her toes, but could slowly extend the legs or draw them up.

There had been no colic, no lead line on the gums. Except the above mentioned slight inco-ordination of the hands, there were no symptoms above the waist. After she had taken iodide of potassium for a week, lead was found in the urine.

She gained somewhat before leaving, had more sensation in her limbs; after about two months could tell whether her feet were hot or cold, and sensation was more natural half-way up to the knees. In March she wrote that she could sometimes move her toes, but the legs moved when they got ready, not when she wished to have them.

Later she was taken to a "Christian Scientist," and was much injured by excitement. She became so bad that there was a question, whether she had not lost her reason; but she subsequently went West, where she had more judicious treatment, slowly recovered the use of her limbs, and could walk well without difficulty. Four or five years later she again was partially disabled; whether she was again exposed to lead I have not learned.

When first seen she was not especially nervous, but the hysterical condition was developed during the excitement caused by visiting the "Christian Scientist," showing that the nervous system was in a condition to be easily upset.

*Case II. Cerebral symptoms, anæsthesia, severe pain, exaggerated reflexes, loss of motion, lead in urine, — great improvement.*

Mrs. H. entered the asylum early in 1888, age twenty-five. She had been sick at times for three years since a miscarriage. She gradually lost strength, and was finally obliged to give up her housework. She felt tired out, the left side ached and was numb and cold, sometimes with a sinking feeling. The right leg gave some trouble, but less than the left. For about three weeks she had been dizzy; she had twice fallen at least partially unconscious; then her head felt badly; she was numb and unable to move for a while. She said she had a nervous attack the previous summer, in which it took several persons to hold her. She had a poor appetite, but nutrition seemed to be fairly good; the least exertion, however, tired her and exhausted her strength.

The pupils were rather large and reacted well; co-ordination of hands was good and sensation normal in hands; she lay with her feet crossed and extended; the great toes were strongly flexed; sensation to a light touch and to pressure was absent below the knees on the left, diminished on the right, diminished on the thighs in some areas.

On striking the ligamentum patellæ on the right to test for reflex, a continuous tremor of the leg and foot was excited. Ankle clonus of short duration was present on both sides. Pressing the left foot up to excite this caused severe pain in the left knee, followed by spasm and rigidity of the legs, especially of the left. The back was sore and tender to the touch. She could not walk; was confined to the bed; she slept poorly; the catamenial flow was profuse, attended with much pain and nausea. Her manner of talking and acting was decidedly hysterical; but she seemed really to suffer much in her back and the ovarian region. Lead was found in the urine.

The galvanic current was used to the spine, the legs and over the ovaries with relief to the pain. After several days she slept better, and finally she slept all night when the electricity was used. She eat more, and sat up.

May 1, it is recorded: "No ankle clonus; patella tendon reflex apparent, but obscured by muscular rigidity: sensation about both knees acute and painful."

She gained, and the last of October could walk about her room without assistance except such as she got from leaning on the furniture. All the muscles reacted naturally to the electric current. After leaving the asylum she relapsed, and did not do as well as I hoped she would.

*Case III. Loss of motion, contraction in legs, exaggerated reflexes, lead in urine, — no gain.*

Mrs. B., age forty-four, admitted June, 1888, was quite well till four years previously, then she first noticed numbness in the right leg, and that leg felt heavy; the gait was unsteady. Three years before entrance she had a child, and was somewhat better afterwards, so that she could walk about and do some housework. For about three months her legs felt strong, then they began to drag as before. She fell, striking her back, without loss of consciousness; since then has had pain in the small of the back and between the shoulders; otherwise she has had no abnormal sensations.

There was a swelling of the feet. After staying in bed a few days this improved, but she could not walk. The bowels were constipated. The legs drew up, flexing at the knees, and then in a short time extended again. On waking in the morning she found the legs flexed at the knees, especially if she was constipated. Sometimes the legs got crossed. Appetite and digestion were fair, and she got a fair amount of broken sleep. The skin was dry; there was œdema of the legs below the knees; sensation was about normal in the legs. The motion was much impaired in the legs; she could not rise without assistance. On moving the legs passively, they were found to be stiff; patellar tendon reflex was exaggerated; there was marked ankle clonus; plantar reflex was stronger on the right; the abdomen was full and tympanitic; the hands and arms were all right. A large amount of lead was found in the urine after she had taken iodide of potassium.

She made no material gain, and the last that was heard from her she could not walk, and was in essentially the same condition as when she left the asylum.

There was less nervousness and instability than in the two preceding cases.

*Case IV. Loss of motion, anæsthesia in legs, exaggerated reflexes, lead in urine, herpes zoster, cerebral symptoms, — improvement, relapse, recovery.*

Miss C., age sixteen, was seen in October, 1881; ailing since March; gave up school then. Going to school she walked rather over a mile, going that distance four times a day. She walked it easily in twenty-five minutes. Two years before she rode much of the time. At Christmas she was tired, but it seemed to be only natural fatigue. There seems to have been no serious trouble before March. She stayed at home from school three days on account of being tired and feeling poorly; was on the bed most of the time, slept much, was not feverish, had no sore-throat, had a dull headache. She then went to school

for half a day, and on coming home could not walk, was very nervous, burst into tears; said that she could not get home, her limbs were numb and weighty. After that she lay down most of the time, slept much during the day all summer, could not walk much, was easily tired, going up stairs made her limbs ache and caused short breath. She cried much and easily.

During the summer she was at the seashore, and after her return her back ached when she rode. When tired her legs felt heavy, and sometimes ached as high as the knees, also she had a sleepy feeling in them. She had a dull headache most of the time.

When first sick there was twitching of the legs in sleep, but this had ceased when first seen. Patellar tendon reflex was strong; a slight ankle clonus was present.

Lead was found in the urine after she had taken iodide of potassium; lead was also found in the drinking-water.

About June she had a very painful attack of herpes zoster. She gained much after this, and was in a fair way to get about. The family moved into another house, and soon after she was less well. The symptoms took a more hysterical character: she was nervous, tremulous, easily moved to tears, and her general nervous condition was much worse. She was more easily tired and upset by company or by little vexations. It was found that the drinking-water was again liable to be contaminated by lead; this was corrected, and there was a slow but constant improvement until she fully regained her health.

*Case V. Cerebral symptoms, loss of motion in arms and legs, pain, reflexes absent, lead in urine — great improvement.*

Mrs. L., age forty-five, had been ill six or seven months, at first having strange, disagreeable feelings and general malaise, with dyspepsia. She had severe nausea and was fed by the rectum, was delirious, and mind was impaired afterwards for four or five weeks. She had had considerable discomfort in legs and hands, and was unable to grasp objects well. At times she had pain in her joints, her legs were weak, and she could not stand without support. The left leg could not be fully extended, the right could be. There was diminished muscular sense in the left leg; a light pressure on the calves caused pain. Rubbing hands and toes caused a feeling like electrical shocks. A light touch was not painful. There was diminished sensation on both sides.

The pupils reacted and the eyes moved naturally; tongue was protruded straight; motions of the face were good. There was a slight loss or coördination in the hands; no tremor of hands. Sensation of hands, feet and legs was natural. Patellar tendon reflex was absent on both sides; there was no ankle clonus. The left leg was somewhat contracted at the knee, and extension caused pain at the hip. To the faradic current the vastus internus on both sides almost failed to respond. Action of the peronei and the interossei of the fingers reacted very poorly. The flexors in the forearms did not react readily; the other muscles of arms and legs responded very well. An unusually large amount of lead was found in the urine.

There was a peculiar childishness in the patient's mental condition; it seemed as though the brain's action was enfeebled as the muscular power had been. She could not read, could listen to reading only for a

short time. There was, however, a firm determination to get well, and an unusual perseverance in the use of exercises to strengthen the muscles. She steadily gained, and finally was able to walk with only a cane for support, and could use her hands fairly well.

*Case VI. Spasmodic action and loss of power in legs, deep reflexes absent, cerebral symptoms, lead in urine, — improvement.*

Mr. B., age fifty-one, called to see me in 1890. He had been ailing for a year or more. He had exercised much in rowing and in other sports. He first began to grow weak in his legs and lost control of his rectum. He felt as if he had an iron on his back tied around tight. There had been hemorrhoids seven years before and at that time he had a mucus discharge from the rectum. He grew weaker in his legs, there was numbness of rectum and back, a girdle sensation about the abdomen. His legs had a sleepy, prickly feeling. Once in a while there was pain in the back during the previous two months, sometimes pain in the legs, no lancinating pain. Once, while riding, he had a peculiar sensation in his rectum, and had to get out and walk. With his eyes shut he walked poorly. Patellar tendon reflex was absent.

He lived for four years where the drinking-water came through a quarter of a mile of lead pipe from a spring. After taking iodide of potassium, lead was found in his urine.

I did not see him again until February, 1891. He had had hemorrhoids and these had been operated upon in the interval. He was no better, rather worse. There was the same girdle sensation about the body at the level of the ensiform cartilage extending nearly the width of the abdomen.

The legs twitch when he goes up stairs, so that it is sometimes easier to take two steps at a time. When sitting the legs are strongly adducted. He sometimes staggers in walking, legs twitch when he lies down. There is little or no pain. Numbness about rectum and genitals. Patellar tendon reflex was gone. There was no inco-ordination of hands, no disturbance of eyes, tongue was protruded straight.

There was a very marked hypochondriacal or hysterical element noticeable in the patient. He was extremely anxious about himself and inclined to dwell upon his disability. When he went out to his business he was better than when he staid at home. He would declare in the morning that he was worse, could not walk, could not go down town. With a little encouragement he would start off walking with difficulty with the aid of a cane, after getting a short distance from the house the cane would be put under his arm, and he would walk without it; or he would meet a friend and stand talking with him, forgetting all about his infirmity.

When I gave him a prescription for iodide of potassium, five grains to a dose, I cautioned him that it might be too much; he had unpleasant sensations from it but not such as are caused by the iodide. On reducing the dose he went on quite well. One Saturday he did not go in town to business. Sunday morning, after a very good night's rest, he had a very nervous spell, said he was in terrible agony, but after a little while he did not mention this again but said his family did not understand that he was continually losing the use of his limbs; he tottered and made terrible work in walking. In a short time a friend came whom he was pleased to see. Nothing more was

heard about the walking and on Monday morning he went to town as usual.

When last seen he was better, having less of the girdle sensation, less bad feeling in his fingers and less numbness in the rectum and genitals, and was looking better in the face.

*Case VII. Numbness, loss of power in legs, lead in urine, cerebral symptoms, — rapid recovery.*

Mr. M. had been ailing two months previous to my seeing him. He had a pricking feeling in his fingertips and toes. He had had syphilis for which he had been treated for two years. He had had no skin affection, but four months after the first lesion he had a sore throat. He had formerly used spirits and tobacco.

About a month before he came under my notice he had had a fissure of the rectum for which he had used a large amount of lead as an injection. He had used canned food, his drinking-water had come from many sources; so it could not be learned how much lead he may have obtained from that.

Two months before the visit the numbness began, then his legs got heavy and he could hardly walk, then he was in bed two weeks with pneumonia. It was necessary for him to travel after this attack of pneumonia and the nervous symptoms were aggravated; the numbness was worse, the heaviness of the legs became worse, his knees and ankles became weak and it was difficult for him to walk. There was no numbness above the middle of the legs nor above the wrists, the arms were weak as well as the legs. There was no pain except twice in the middle of the forearms. He dragged both legs, the left the more. The pupils reacted to light, the left was slightly the larger; the tongue and the facial muscles acted naturally, there was a slight inco-ordination in the left hand, less in the right; patellar tendon reflex was absent. Sensation was blunted in the legs, higher it seemed normal, it was perceptibly delayed in the tips of the fingers, slightly delayed in the palm and much so on the palmar surface of the fingers, both hands alike.

After taking iodide of potassium, lead was found in his urine. He was given the iodide and made an unusually rapid recovery.

He was very nervous, almost hysterical, and anxious about himself, and probably appeared worse than he really was.

In April I saw him again: he said he was perfectly well and seemed so to be. The patellar tendon reflex was normal, he walked naturally, sensation was normal.

Syphilis might be suspected, but the dose of iodide was not more than seven grains, and it would be quite as unusual to have syphilitic neuritis recover so speedily with so small a dose as to have lead neuritis recover so in so short a time. Though he seemed very badly off when first seen, much of the unfavorable appearance was probably due to his extreme nervousness.

Dr. Minot reports a case of lead paralysis with rapid recovery.<sup>7</sup>

The prognosis in these cases of lead paralysis even when they seem very severe, is generally favorable to judge by these and similar cases. By a persistent use of iodide of potassium, with massage and electricity, even seemingly hopeless cases have improved or recovered. In a few cases, especially if not seen early enough, the termination is unfavorable. Serious cerebral symptoms are probably more unfavorable than the spinal and peripheral symptoms.

<sup>7</sup> Boston Medical and Surgical Journal, August 16, 1882, p. 155.