

## Lecture.

## LOCOMOTOR ATAXIA—AMYOTROPHIC LATERAL SCLEROSIS—LATERAL SCLEROSIS.

A CLINICAL LECTURE DELIVERED AT THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA.

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GENTLEMEN, this patient comes to us with the statement that every two or three weeks, or sometimes at longer intervals, he has frightful attacks of abdominal pain accompanied with vomiting. Under these circumstances, attention is naturally directed to the condition of the stomach. We learn that these attacks are not provoked by any error in diet, that they are apparently spontaneous, and that between the attacks there are no symptoms of dyspepsia or indigestion. The attacks are exceedingly violent. The patient came to the hospital two weeks ago. When I first saw him, he appeared to be suffering from no severe symptoms. A day or two later, I found him in bed, groaning and moaning as though in agony. There was frequent vomiting of mucus tinged with bile, or of a liquid so thin as scarcely to be called mucus. These attacks of pain with persistent vomiting lasted three or four days and kept the man from sleeping, in spite of the free use of opium. Such attacks are evidently not dependent on irritation of the mucous membrane of the stomach.

The pain would suggest the passage of gall stones. Careful examination, however, shows that the pain lacks the sudden cessation which is characteristic of biliary colic. It is a steady, unbearable pain, lasting for hours and days and unaccompanied with jaundice, disturbed digestion, or any other manifestation of the passage of biliary calculi. You will call to mind the case of the woman with attacks of pain, similar to these, occurring in the rectum, which I showed you last week. When you have brought to your notice a case of horrible, recurring, violent, unaccountable pain, remember the possibility of its being one form of crisis occurring in locomotor ataxia. Sometimes these paroxysms of intense, shooting, darting, tearing, boring pain attacks the genital organs.

When I found this man sitting at his bedside, my attention was at once directed to his pupils. I found that they were very small, in other words, he had a distinctly myotic or contracted pupil. When I shut off the light with my hand, I found that the pupil did not dilate. It was, indeed, insensible to light. I then tried the pupillary reflex, but there was no dilatation of the pupil produced by pinching the skin of the neck. Then I asked him to look at my finger held close to his face and a moment later to look at a distant object, and found that the pupil which was immovable to light, and responded not to peripheral irritation, reacted normally to the movements of accommodation. Our patient has, therefore, that comparatively rare pupil known as the Argyle Robinson pupil, because first described by that gentleman.

Before going further with this case, I wish to say a few words in regard to conditions of the pupil as seen in nervous affections. We have as the afferent nerve, so to speak, going from the eye to the centre within the brain, the optic nerve. We have two centres, connected as motor centres with the pupil, the oculo-motor centre and the spinal dilating centre, situated high

in the neck. What happens when a person's neck is pinched? An impulse is sent through the sensitive nerves which reaches the cervical centres, lying in the upper part of the spinal cord. As a result, there goes out from the cervical sympathetic ganglia, an impulse causing dilatation of the pupil.

Again, the pupil contracts with exposure to light, and dilates when the light is withdrawn. This is especially accomplished through the oculo-motor centres. The optic nerve is the afferent nerve. Its fibres run through certain centres in the neighborhood of the thalamus opticus and then pass down to the corpora quadrigemina and oculo-motor centre. As a result of exposure to light, there is oculo-motor stimulation and the pupil contracts.

Then as to the movement of accommodation. When a near object is looked at, the eyes are brought convergent as to their axes, and at the same time, the pupil contracts and the shape of the lens is altered for the purposes of distinct vision. These are so-called consentaneous or associated movements, that is, movements which habit or the original construction of the nervous system has brought about as always being performed together. They apparently take place through the oculo-motor centre. An impulse from the upper cortical region of the brain is sent down to the oculo-motor centre for the act of accommodation, and the centre sends out an impulse which contracts the pupil, and at the same time, converges the eyes.

Besides these various movements of the pupil, there are others associated with emotional conditions, but we have been unable to study these in this case.

In the Argyle Robinson pupil, there is want of response to light and to reflex irritation from the skin, but the pupil does respond to alterations of accommodation. Wherever this pupil is found, there is almost of necessity serious organic nervous trouble, and the probabilities are always in favor of the idea that the patient is suffering from locomotor ataxia. The Argyle Robinson pupil has, however, been found in a certain number of cases of general paralysis of the insane, and perhaps, in a few other diseases, but it marks especially the presence of locomotor ataxia. Where in any given case, as here, there exists violent gastric crises, along with the presence of the Argyle Robinson pupil, you have even without further examination, sufficient grounds for the diagnosis of locomotor ataxia.

The explanation of this peculiar condition of the pupil is not difficult. We know that the optic or afferent nerve is perfect because the patient sees. There is no evidence of paralysis of the oculo-motor nerve and the pupil is contracted. Supposing the man to be suffering with locomotor ataxia, it is plain that the reason why there is no response to light is that the fibres which connect the optic centre with the oculo-motor centre are involved in the diseased structures.

There is interruption of the pathway and there can be no passage of the impulse from the centre of the optic nerve to the oculo-motor centre. The reason that the pupil does not respond to peripheral irritation is because the sensitive nerve connected with the upper spinal region is also involved in its passage through the cord. You will remember that in locomotor ataxia, there is a chronic inflammation and sclerosis or hardening of the posterior column of the spinal cord, and hence these sensitive fibres are cramped and squeezed and their function abolished. When the skin is irritated, no impulse reaches the centre. It is interest-

ing to observe in connection with the gastric crises, these other signs that the disease is high up in the spinal cord, even in the medulla, for the medulla, although placed within the skull for purposes of protection is nothing more than the upper part of the spinal cord.

When the man is examined further, other evidences of locomotor ataxia are found. In the first place, he has lost the patella reflex. He has darting and shooting pains through the legs, unaccompanied with soreness, or with pain on motion. Remember that bilateral, darting shooting pains, without soreness and without pain on motion, are in nine cases out of ten, if they are persistent and not due to gout, the result of locomotor ataxia. When with this there is loss of the patella reflex, the diagnosis is almost positive. I have also found some disorder of coördination in this case. The man has a somewhat ataxic gait. He walks with the feet spread wide apart so as to give a firm base of support. The movement of the legs are irregular. With some difficulty he can stand on both legs with the eyes shut, but is unable to stand on one foot with the eyes shut.

We are therefore able in this case to arrive at a positive diagnosis of locomotor ataxia.

Leaving this case for the present, let me briefly call attention to the various forms of local inflammation of the spinal cord with which we meet in practice. In the so-called system diseases of the cord, the sclerosis or chronic inflammations involve certain tracts of the cord, running up and down, but do not invade widely scattered foci. In the centre of the cord is the gray matter. Then we have the lateral tracts. In the centre of the anterior portion of the cord is a small tract which corresponds to these lateral tracts physiologically. Then we have the posterior median columns or the columns of Goll. So far as system diseases are concerned, we know of two sclerosis especially, which produce definite symptoms. In the first place the posterior region may be involved, especially the region where the posterior nerve roots emerge, constituting *locomotor ataxia*. In the second place, the lateral columns of the cord may be affected, constituting *lateral sclerosis*. There are one or two cases in which the symptoms have been said to have been due to sclerosis confined to the columns of Goll. This is, however, rare, and I have never met with such a case. In the anterior portion of the gray matter there are certain groups of large cells. These are the motor cells whose function it is to convey the nervous impulses which shall cause contraction of the muscle, and it is also their function to preserve the nutrition of the muscle. When a muscle is cut loose from these cells it wastes and its electrical reactions change. When this portion is diseased, we have, if the affection is acute, infantile paralysis or acute muscular atrophy; if it is chronic, we have progressive muscular atrophy. When in spinal affections, there is rapid wasting of the muscles and rapid changes in the electrical reactions, there is disease of these cells; whatever else may be present in the spinal cord these cells are involved.

There is an affection of the spinal cord, in which there is disease of the lateral columns associated with disease of these motor cells. This is known as *amyotrophic lateral sclerosis*.

In the consideration of the case before us, I have called attention to most of the symptoms of locomotor ataxia. They are disorders of locomotion and coördi-

nation and pain without loss of motor power or wasting of the muscles.

Let me now call attention to this second case which represents another form of spinal affection, namely, lateral sclerosis. The symptoms of locomotor ataxia are sensory and afferent. The lack of the power of coördination is due to the failure of afferent impulses to reach the brain. In lateral sclerosis, the symptoms are disorders of motion, but not of nutrition of the muscle, nor of sensation. There is no wasting of the muscle and no lack of coördinating power. There is simply disorder of the motor function of the muscle. These are chiefly the result of irritation, so that there is more or less permanent spastic muscular contraction. With this there is excitability of the reflexes with a certain amount of loss of power, because whilst the fibres are irritated, there is also interference with the passage of currents down from above.

This case exhibits the symptoms of lateral sclerosis. Although I lift the leg and support it at the thigh, there is no bending at the knee, and it requires considerable force to flex the leg. Tapping the tendon of the patella, I find that the patellar reflex is abnormally increased. I cannot at this time develop ankle-clonus, although it has been noted in this case. There is, then, excessive rigidity, increased reflex activity, and, in addition, a peculiar gait. When the disease is fully formed, the gait is characteristic. The patient cannot get his toes from the floor, owing to the spasm of the calf muscles. Examining the legs, no muscular wasting is found. The electrical reactions are normal. The arms are sometimes affected in lateral sclerosis, but in this patient the stiffness is very slight at the arm. There is, however, great loss of power in the arms. On exploring the arms, fibrillar contractions are noted as abundant. These are never seen in pure lateral sclerosis. Moreover, there is great wasting of the shoulder and arm muscles. These symptoms, we are told, came on gradually, they are evidently due to disease of the motor or anterior cells of the gray matter of the cord, and we have an instance of the chronic spinal disease, known as *amyotrophic lateral sclerosis*.

Turning now to the third patient, we find the following history. This girl is twenty-six years of age. She was in good health until three and a half years ago, when she developed the symptoms with which she now suffers. The affection has come on gradually. There was, she stated, at one time partial loss of power in the left arm and leg, and she was unable to work for four weeks. She then obtained an easy place and returned to work.

You observe the same stiffness of the legs, seen in the previous case. When I raise the thigh from the bed, there is no flexion at the knee. The muscles of the calf are contracted. The patellar reflex is increased. There is some rigidity of the arms, most marked on the left side.

We have then, in this case, either lateral sclerosis or something simulating it. There is an affection known as hysterical contracture in which there is loss of power, with heightened reflex activity, rigidity, or more or less permanent contraction of the muscles, a disease which very closely simulates lateral sclerosis, and in some cases it is almost impossible to make the diagnosis. Two years ago I had a case in the Philadelphia Hospital which well illustrates the difficulty in diagnosis. This woman suffered with Pott's disease of the vertebrae which had produced angular curvature.

It is not uncommon to have secondary sclerosis following the transverse myelitis of this affection. With a pronounced history of disease of the vertebrae, there were the typical symptoms of lateral sclerosis and an almost entire absence of hysterical indications. The diagnosis of lateral sclerosis was made. On one occasion, she was given some powders of bismuth for a slight derangement of the stomach, and she began to rapidly improve, so that in a few days, instead of being confined to a rolling chair, she was able to walk about. That was largely a case of hysterical contractures.

This woman before us is not distinctly hysterical. If in a decidedly hysterical patient you have symptoms like these, especially if they have developed suddenly, the probabilities are that you are dealing with a case of hysterical contracture. Although this patient is somewhat nervous, she is not distinctly hysterical. The disease has come on slowly. Hysterical contractures are more apt to come on suddenly, but not necessarily so. This woman has another symptom which I believe is characteristic of organic disease, and at present, I should always make the diagnosis when I found it distinctly present. During the past two months she has complained of having the feeling of a bandage around the waist. This she spoke of herself. Of course if you ask an hysterical patient if she has a band-like feeling around the waist, she will be very apt to say that she has, when she had never dreamt of it. Another point I wish to allude to, and that is, that chronic nervous diseases, especially in women, are often associated with hysteria. If any one of us were shut in a room for months and months, over-shadowed by a great cloud of approaching troubles, it is probable that we would develop hysterical symptoms. The point is always to be borne in mind, that underlying the hysterical manifestations, there may be a real organic trouble.

There is in this case, a slight inequality of the pupils. Everything, therefore, points to the existence of lateral sclerosis and the prognosis is unfavorable. We should, however, not pronounce a too positive prognosis, but should leave a way of escape in case the condition should prove to be largely hysterical.

## Original Articles.

### A CASE OF INFANTICIDE.<sup>1</sup>

BY W. H. TAYLOR, M.D., NEW BEDFORD, *Medical Examiner.*

M. R., aged a few years over thirty, gave birth to her fourth illegitimate child at some time during the night succeeding April 21st, 1884. She was employed as a domestic in a gentleman's family, and as she did not appear at her customary duties on the morning of April 22d, search was instituted, and she was found in bed suffering from the effects of her delivery. The physician to the poor being summoned, found a dead child lying upon the floor, closely wrapped in a shawl, together with the results and debris of the labor. Not being satisfied that the child was still-born he referred the case to me, and gave a very full account of the appearances of the room, mother, and child, which caused in his mind the suspicion that all was not as it should be. Upon arrival at the house I found the woman in bed in a room at

the farther end of the attic from the stairway, which stairway was shut off from the story below by a door. She occupied this attic alone, and had neither medical nor other attendance during her labor.

Upon the floor in front of the bed, and a foot or a foot and a half from it, was a waterproof overgarment of india rubber, the edges of which had been carefully turned over to make a sort of trough three feet by two perhaps. Upon this garment was a chamber-vessel filled completely with bloody fluid and a placenta with membranes attached. Four inches of the umbilical cord remained upon the placenta, and the extremity of the cord was ragged. Beside the chamber-vessel the garment had upon it the body of a new-born child (wrapped in a red and black checked shawl), a blue flannel skirt, and a large quantity of human faeces, meconium and bloody fluid.

The mother on being questioned concerning her labor, stated that she went to bed as usual on the night of the 21st, not expecting to be confined. At eleven o'clock, P.M., she had a desire to urinate, and rose to perform this act. Upon resuming the bed, she had a few slight pains not sufficient to prevent sleep. At midnight, as near as she could judge, she had a desire to stool, and, rising for the purpose, while in a squatting posture by the bed, she gave birth to her child, then fainted and fell to the floor. At one o'clock, A.M., she regained consciousness, and, throwing a shawl over the child, she went to bed, where she remained until found in the morning. She did not know how the cord became ruptured, or how the placenta came to be in the urinal, and the child upon the floor. She did not hear the child cry or manifest any sign of life.

Examination of the child's body revealed the following appearances. The body was well smeared with meconium, and a considerable quantity had escaped from the child. The face, hands and feet were cyanotic. The body was cold with hardly any rigor mortis. The portion of umbilical cord attached to the child was twenty-five inches long and had one turn about the child's neck, leaving a free end fifteen inches long. The extremity of the cord was ragged and bulbous, and had clot behind the incurved extremities of the vessels. The cord had not compressed the neck to leave any depression or mark, and could not have compressed it except by strong traction of the maternal end. A little whitish froth was noted about the left nostril. There was a slight caput succedaneum over the occiput and a little to the left. No marks of bodily injury were seen on inspection. The cavities of the body opening externally, contained no solids or fluids except what might naturally occupy them.

Autopsy showed a fat male child at term, twenty inches long and weighing seven and three-quarters pounds. Both testicles were in the scrotum, and the nails had grown to the ends of the fingers. No fluids escaped on inverting the child, and none were found in the respiratory passages.

On opening the body, the diaphragm was found at the level of the sixth rib. The lungs removed with the heart and thymus gland, floated high above the surface of water. Sections of all parts of the lungs floated after being subjected to heavy pressure. Sections made from the edges of the lungs with the freezing microtome by my friend, Dr. Wm. N. Swift, who kindly assisted me, showed the air-cells to be everywhere fully dilated. The lungs were rosy red anteriorly, darker posteriorly. They contained much dark

<sup>1</sup> Read before the Massachusetts Medico-Legal Society.