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 crisis, 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 interesting 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 somewhat ataxia 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 scleroses or chronic inflammation 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 scleroses 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 coördination, 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