becomes, and in consequence the more easily it yields to any injurious influence. The same may be said of the sensory fibres which are nourished by the ganglia of the posterior roots. This also explains the parenchymatous nature of the affection, since there is no reason why the connective tissue should suffer most at the periphery, and we find it accordingly most frequently in the course of the nerve, as is seen in many cases of sciatica.

The motor symptoms, the wasting and the changes in the electrical reactions, in this disease are easily explained by the implication of the motor nerves; the exact site of the lesion while in the trunk or the actual nerve ending not making any difference. The ataxy of this disease is interesting, and is explained by the affection of the sensory muscle nerves, which, as was shown by Tschirjew, end not in the muscular fibre itself, but in the interstitial connective tissue between the fibres. Derangement of the function of these sensory fibres would naturally obstruct the connection of the muscle with the higher centres and ataxia must be the result. The pains and hyperæsthia are due to the molecular changes going on in the periphery of the sensory nerves, which are still connected with their centres by healthy tissue, these changes inducing a state of over excitability in the nerve itself, which condition continues until complete restoration takes place.

It has been well said that the discovery that certain combinations of symptoms, which were thought to be due to a disease of the spinal cord, are really due to disease of the peripheral nerves, is one of the most important steps in modern pathology. This assertion offers a ready explanation for the fact that until quite recently a diagnosis of multiple neuritis was so seldom made. The older observers devoted their attention exclusively to the central nervous organs, disease of the peripheral nerves being quite overlooked. Although first described by Duménil in France in 1864, it is only since the works of Joffroy (1879), Leyden (1880), and since then those of Buzzard, and especially Déjerine, have become known that multiple neuritis has been given its true place in neurology. This also helps to explain why multiple neuritis was so frequently diagnosed as anterior poliomyelitis, Landry's disease, locomotor ataxia, etc., by the older authors. Although it usually happens that both the motor and sensory nerves are involved together, cases are met with in which either the motor or the sensory nerves are alone affected. If we suppose the motor nerves only to be affected, we can understand at once how the acute onset, the muscular wasting with the reaction of degeneration, the rheumatic pains, and the absence of sensory symptoms from the skin may lead to a diagnosis of poliomyelitis.

In certain rapid forms of multiple neuritis the resemblance to Landry's disease is very great. The causes of both are alike in many respects, and the symptoms have much in common. In Landry's disease, however, the symptoms ascend the trunk from the legs, and it is nearly always rapidly fatal. In Germany, Eichhorst recognized by autopsy one of his cases, diagnosed Landry's disease, to be really multiple neuritis; and in France Déjerine met with the same discovery. The latter expressed to me his opinion that Landry's disease was always rapidly fatal. Very interesting in this particular are the cases published by Pal of Vienna. One of these, a fatal case, lasted twenty days in all, dying one week after entry into the hospital. The symptoms showed paralysis of all the extremities, pulse at the last quick, paralysis of abduceus of left eye, sensibility normal or scarcely lowered, bladder and rectum intact, and some diminution of Faradic irritability. At the *post-mortem* lesions were found in the peripheral nerves, and also in the cord of which I happen to have some microscopic sections. This case would, he says, have undoubtedly been described as Landry's