ference with the function of the so-called trophic centres for the joints in the spinal cord. It is claimed that the degenerative process in tabes and syringomyelia involves such parts in the spinal cord. The other view, and one that is steadily gaining ground, is that owing to the lessened or disturbed sensation so frequently met with in both tabes and syringomyelia, traumatic influences have much to do with setting up the inflammatory action, and according to this view it is not necessary to assume the presence in the cord of centres having a trophic influence over the joints, the destruction of which brings about the changes. In the great majority of cases of both tabetic and syringomyelic arthritis, a history of a fall or injury is obtainable. There is nothing special in the joint changes that could not be explained by an inflammatory action excited by an injury. The clinical difference is accounted for by the sensory disturbance in the joints, and all things considered, it appears more consistent with observed fact to explain the arthritis on the assumption of an injury than that it is brought about by the involvement of certain definite parts of the spinal cord.

The question of the cause and nature of the joint changes in tabes, syringomyelia, etc., is still a matter of doubt. It is unwise to speak too positively on this matter. There is, however, very strong ground for taking the view that the joint changes in rheumatoid arthritis are not due to disease of the spinal cord. Should such changes be brought about in that way, it is hardly conceivable that they should not present evidence of not only microscopic, but macroscopic changes in the spinal cord. In several cases the spinal cord has been examined in rheumatoid arthritis after death, and no abnormal appearances have been discovered Folli in two cases saw some wasting of the cells of the anterior cornua, but elsewhere nothing. Changes in the peripheral nerves have also been met with in a few cases, but neither the slight changes described by Folli or the nerve changes are constant, and, therefore, cannot be considered as sufficient causes of the joint changes in rheumatoid arthritis.

It is difficult to explain the marked and comparatively early atrophy of the muscles that occurs in rheumatoid arthritis. It does not correspond clinically to that met with in anterior polio-myelitis for we do not meet with any marked reaction of degeneration. The electric reaction is often normal, and is rarely more than slightly lowered. The view commonly held at present is that the wasting is brought about in a reflex manner. This theory receives support from the experiment of Raymond that division of the posterior spinal roots prevents wasting from taking place in joint disease. It must