in the cord. While the cerebrum is at rest during sleep, or when we are not moving, the anterior horn cells have an opportunity of resting and recuperating, and when the stimuli from the spinal sensory cells are cut off owing to their degeneration in this disease, the anterior horn cells have still further opportunity for resting.

This theory also accounts for the exacerbation of symptoms after any acute infection, accident or shock, the additional toxines in the system impairing still further the recuperative power of the cells. It accounts for the number of cases of tabes that develop in soldiers newly sent on active service. The 4th case referred to is an example of this. It accounts, also, for those few cases where there is even on careful and exacting investigation no evidence of primary syphilitic infection, some other slowly acting toxine may be present impairing the recuperation of the cells.

Will this theory account for the pathological picture we find in Tabes? We think it will. There is the degeneration and sclerosis of the posterior columns, usually most marked in the lumbar region, but in cases of cervical Tabes, such as the three first cases shown to-night, the sclerosis is most marked in the cervical region. At the same time there are relatively few changes to be found in the posterior ganglia from which these degenerated axis cylinders arise. Just as in a plant that has not had sufficient nourishment it is the leaves and branches which die first, the roots still retaining their vitality, so it is with the nerve cell when its nutrient pabulum is insufficient, the parts farthest away from the nutritional centre are the first to die and their place is taken by connective tissue, giving rise to the sclerosis of the posterior columns of the cord. None of the other theories of Tabes based on the pathological picture of the disease will account for the clinical findings in so satisfactory a manner, they do not account for the preponderance of male tabetics over women, nor for the improvement of the ataxia when optic atrophy develops, nor for the development of the optic atrophy itself, nor for the Argyl-Robertson pupil, nor do they account for the cervical region being affected in one case and the lumbar region in another. Again, if we accept this theory as the true one, it immediately puts into our hands most obvious and definite indications for treatment at least prophylactic, and the results of this treatment bear out the truth of this theory. This is especially seen in the re-educational treatment of the ataxia. The patient must avoid fatigue of any part.