Case IV.—A. S., male, aged 36, a shoemaker, admitted to the hospital under Dr. Martin, December 30, 1909, complaining of inability to walk, and lightning pains.

For three or four years the patient has been in the habit of congratulating himself on his ability to hold his water, even making it a matter of boasting that he could go from the time he got up until he went to bed again, without emptying his bladder. In April, 1908, after selling his place of business, he started out with what was left to him in a bullock waggon, and walked for eight days and nights across country, in cold and rainy weather, becoming terribly fatigued and was "snow blind" for weeks after this; from that time he has been unable to walk without support, not being able to control the movements of the legs; he cannot attempt to walk in the dark. In May, 1908, he found that he could not urinate when he wanted to, and he began to have incontinence. He has had the lightning pains for the past seven months. There is a history of a chancroid 10 years ago, but there were no secondary symptons.

On Examination:—The fundi are pale and atrophic looking, there is the typical Argyl-Robertson pupil, and internal strabismus due to a paresis of the left external rectus. There is marked loss of tone and extreme ataxia of the lower extremities, so much so, that the patient cannot walk; he has impairment of sensibility over the feet and about the mammary regions. The knee and ankle jerks are absent.

Following the other cases, the explanation of this one is obvious. Of course the snow blindness was simply a conjunctivitis, but the glare of the snow on the eyes was quite sufficient to cause a partial optic atrophy,[1] and the journey, exposed as he was to wet, cold and extreme fatigue, sufficient to produce exhaustion and determine the symptoms in the lower limbs.

We know that there are certain changes which take place in the functioning nerve cell; these may be demonstrated under the microscope; they consist in a breaking up of the Nissl bodies, and further a swelling of the ganglion cell with an emigration of the nucleus towards the periphery, and finally, if the hyperactivity of the cell be continued, and extrusion of the nucleus and the death of the cell. If, however, the activity of the cell cease before the extrusion of the nucleus we know that the cell will recover, under normal conditions, its original appearance.

Now, in Tabes, "function determines the symptom complex." There is, in this disease, the presence of a toxine, usually syphilitic, which renders the nutritional papulum served to the nerve cell not sufficient

[1] Edinger reports a similar case of a naval officer who developed optic atrophy from being exposed to the glare of the sun off the water while superintending target practice at sea.