changes with increasing distinctness, and at the level of the first dorsal segment was quite soft, the normal markings being obliterated. Microscopically was seen parenchymatous myelitis and neuritis, with degeneration of nerve fibers and ganglion cells in both the white and the gray matter.

Taft, A. E. ANTERIOR HORN CELLS. [Am. Arch. of Neur. and Psych., Jan., 1920.]

The problem of spinal segment localization. Three of the cases in which cell counts were made on the anterior horns of the spinal cord were those following amputation of an extremity. The other twenty-five had signs of a cord lesion. Variations in the counts between the two sides in the first three was not more than two or three cells in the final average. In two, the greater number was on the side corresponding to the amputation. In the cases without amputation variations in the counts was at times greater than with amputation. In sections where identification was made of right and left side, no uniform difference between the two corresponding counts appeared.

Crouzon and Bouttier. FAMILIAL AMYOTROPHY. [Bull. de la Soc. Méd. des Hôp., Dec. 19, 1919.]

That which the authors give as a new variety of amyotrophy was seen in three sisters between 25 and 32. The first symptoms appeared at 14, 22 and 12 years of age, the older sister is unable to stand on account of the motor and atrophic disturbances. Speech is also spasmodic and panting, hardly above a whisper; reflexes are abnormal, and choreiform movements present.

Herzog, Franz. REFLEX TIME OF THE TENDON AND THE MUSCLE TONUS. [Neurol. Centralbl., April 1, 1918, No. 7, Vol. 37.]

The patellar reflexes were measured according to Hoffmann's method with Edelmann's galvanometer. In measuring the Babinski a special technic was used. Nine cases showing exaggerated tension reflexes as consequence of paralysis of peripheral nerves were carefully examined and are here described. The author says that in all three groups of observations he had the same object, i.e., to determine how the stimulus is communicated from one neuron to another where the mode of communication may have been altered by disease. In the patellar reflexes the problem presents itself in the simplest form, because the paths of the muscle tonus and of the tendon reflex are for the most part identical, consisting of only a few (at least three) neurons. In the author's cases the muscle tonus, as result of various diseases of the central nervous system (diseases which were accompanied by lesions of the pyramidal tracts—tabes dorsalis, etc.) was greatly decreased or diminished. The time of the patellar reflexes, however, was found to

be nearly normal, showing that it is not perceptibly changed by increase or decrease of the muscle tonus. In these instances the stimulus passes only twice from one neuron to another. If the path had consisted of a larger number of neurons the change of their function would perhaps become apparent in a change of the reflex time, as result of the transition of stimulus from neuron to neuron. The reflex of the Babinski sign was always longer than that of the normal tendon reflex, being in some cases several times as long as that of the plantar reflex. The reflex path of the Babinski, a skin reflex, is composed of many more neurons than that of the tendon reflex. The long and changeable reflex time of this skin reflex is obviously not due to the length of the nerve fibers, but seems to be due either to the fact that its path is composed of a larger number of neurons, or because the stimulus travels more slowly than in the path of the normal sole reflex, the great difference in time being confirmatory of the view that the Babinski is not a modified plantar reflex, but a pathological reflex wholly independent of it. In the third group there was traumatic paralysis of the peripheral nerves of the upper and lower extremities accompanied by exaggerated reflexes of the same extremities with injury of the paths of the latter. This was observed both in cases where the nucleus of the paralyzed nerve was above the reflex center and where it was below. No increase of muscle tonus was discoverable, so the exaggerated reflexes must have been due to some other cause. The author thinks it probable that, in time, the paralysis of the peripheral nerve destroys the functions of the original cells of the paralyzed motor fibers so that they do not receive the stimuli from the centripetal fibers. It may then be assumed, in the author's cases, that these stimuli react on the cells of the anterior cornua, but owing to changes in the function of these cells the stimuli is not received. Further, it must be remembered that the centripetal fibers of the path of the tendon reflex is not only connected with the nuclei of the muscles controlling the reflex contractions, but also with the lower roots of the motor nuclei of neighboring segments, a fact proved by the circumstance that where there is exaggerated excitability, contractions occur in numerous muscles, which, under normal conditions, do not react. Thus the stimuli from the affected peripheral nerves spreads to other nuclei and some of the stimuli react on the nuclei of the muscles which give rise to the reflex contractions. The author's cases were not, therefore, cases of exaggerated reflex excitability, but due to an increase of stimulus to the muscles. This view is sustained by the observations of others, Babinski, Déjerine, Egger, etc. Exaggeration of muscle reflexes in polyneuritis may perhaps also be explained by the author's assumption that the disease of the neighboring parts causes the stimulus to spread to the intact paths and that the reflex paths receive part of it.