

author also found that in this disease the red cells were diminished, the specific gravity increased and the spleen became smaller. He also observed that closely following the attacks the leucocytes were not as numerous as one hour later. A few hours after the attack there was an absolute and relative increase in the eosinophiles and disintegrated cells. The author holds that there are alterations taking place in the spleen during the epileptic attack which account for these changes.

BENOIT.

179. BEITRÄGE ZUR LEHRE VON DEN AUS NERVENGeweBE BESTEHENDEN GESCHWÜLSTEN (Contributions to the Study of Nervous Tissue Tumors). H. Haenel (Arch. f. Psychiatrie, 31, 1898, p. 491).

Haenel describes some anomalous tumors of the dura in a patient dying of a sarcoma of the white matter of the right hemisphere, that had undergone cystic degeneration. These two tumors were located on the dura near the superior orbital fissure.

One was a true myelo-neuroglioma, about 2 cm. in diameter, and consisted of strands of nerve fibers, running parallel, having large cells at their terminal filaments. These cells, from their general shape and size and appearance, strongly resembled the ganglion cells of a spinal ganglion, especially the Gasserian ganglion.

The other tumor resembled a fusiform cell sarcoma, soft in consistency, which was partly made up of fibers from the other tumor. Hemorrhagic foci about the lymph spaces and proliferation of the endothelium gave it the stamp of Ziegler's lymphendothelioma. There was also pachymeningitis interna, obliterating endarteritis, and fatty degeneration of the dura.

JELLIFFE.

180. ZUR ANATOMIE DER SEHNERVENATROPHIE BEI ERKRANKUNGEN DES CENTRALNERNVENSYSTEMS (On the Anatomy of Optic Nerve Atrophy in Diseases of the Central Nervous System). A. Elschnig. (Wiener klin. Wochenschrift, March, 1899, No. 11, p. 275).

The author presents the results of a study of the anatomic conditions underlying optic nerve atrophy in two cases, one of disseminated cerebro-spinal sclerosis and one of tabes. In the first-named condition he believes the atrophy to be the result of a peculiar interstitial inflammation which occurs absolutely at random in localized portions of the nerve and causes first a rapid destruction of the nerve-sheaths and fibers, and then an increased formation of connective tissue. This process he considers identical—so far as its inflammatory nature is concerned—with that which gives rise to the central sclerotic changes. In the tabetic case, Elschnig found complete atrophy of the nerve fibers and ganglion cell layer of the retina, diminution in volume of the entire nerve, increase of glial tissue in its intraocular portion, and in the remaining portion a partial disappearance of the nerve fibers, this more marked in the distal than in the proximal portion; further, a disappearance of the finer branches of the connective tissue septa which were themselves, both absolutely and relatively, somewhat thickened; thickening and sclerosis of the walls of the vessels.

From the fact that the anatomical changes were most marked in the retinal end of the nerve and diminished in intensity centrifugally, the conclusion is drawn that the process which leads to optic nerve atrophy in tabes is to be considered analogous in its primary topographical origin, and in its subsequent developmental direction with that which involves the other sensory neurons in this disease; in other words, that it is a peripherally originating centripetal process which is in nowise to be considered a pressure neuritis in the ordinary sense.

COURTNEY.