NEUROLOGY

NEUROPATHOLOGY.

[41] Affections of the ependyma and their pathogenesis (Ependimopatie e loro patogenesi).—P. REDAELLI. Riv. di pat. nerv. e ment., 1931, xxxvii, 309.

The author has examined 27 cases of atrophic alteration of the walls of the cerebral ventricles in elderly people. These included 15 cases of granular ependymopathy, three of the reticulate variety, and three of the varioliform ependymopathy of Pierre Marie. After having traced in each of them the histopathological picture, he discusses the etiological factors, and lays stress on the extreme complexity and diversity of this in each form. He concerns himself particularly with the granular variety and describes a series of physico-chemical disturbances which specially affect the marginal glia beneath the ependyma. The same marginal glia is concerned in a variety of ways with the genesis of ependymal gliosis and gliomatosis. With regard to the varioliform ependymopathy of Pierre Marie, he considers that it is primarily concerned with the glial elements according to the hypothesis of Nageotte, and does not arise from the degeneration of perivascular adventitia.

[42] The origin and formation of senile plaques.—A. FERRARO. Arch. of Neurol. and Psychiat., 1931, xxvi, 1042.

Senile plaques were first described in 1892 by Blocq and Marinesco, who called them sclerotic plaques of neuroglia, a name which was later changed by Redlich to miliary sclerosis. They may originate from both neuroglial elements and from nerve-cells.

Of the neuroglial elements the oligodendroglia cells give origin to some of the plaques, while the astrocytes do not seem to participate in their histogenesis. Conversely, the microglia cells represent an important element from which senile plaques primarily develop. Senile plaques may also originate from isolated disintegrated nerve-cells or from a collection of such elements. The histochemical process that leads to the transformation of a cellular element into a senile plaque is as yet unknown. All that Ferraro can say is that it leads to the formation of a granular argyrophile substance which gives at times, in the central portion of the plaque, some of the reactions of amyloid substance, and at other times the reaction of fat substance. Once the plaque is formed, its development in size and in volume depends on the participation of nerve-cells, microglia, oligodendroglia and, less frequently, astrocytes. Of the cells that do contribute largely to the development of the plaques, the microglia constitutes the largest number. Nerve-cells also participate in the development of the senile plaque through a process of disintegration.

In a lavishly illustrated paper the authors give a complete presentation of the histological features of the oligodendroglioma, based on a study of four cases. They confirm the observation that these tumours are composed of oligogliocytes.

R. M. S.


The author has followed the technique of Chevassut in four cases of disseminated sclerosis, and is unable to confirm the view assigning pathogenic value to the 'spherula insularis.'

J. V.


In addition to the well-known reflexes of the carotid sinus (cardio-inhibitory, respiratory, and that of arterial tension) the authors have been able to demonstrate another reflex characterised by descent in the plethysmographic curve during compression of the sinuses.

The excitability of the sinuses is diminished in epilepsy and is revealed by reduction of all the reflexes depending on that vascular segment. This hyposensibility explains the instability of vasomotor mechanisms and of arterial tension in the affection. In certain organic nervous diseases of the brain, and in some cases of epilepsy, compression of the sinuses initiates convulsive attacks.

The favourable effect of phenylethylmalonylurea in epilepsy is explained in part by its sensitising action on the carotid sinuses.

In encephalitic Parkinsonism the respiratory reflex of the sinus undergoes modification, and its vasomotor reflex is diminished in myasthenia and uprarenal insufficiency.

J. V.


This is a very long and elaborate study of the clinico-physiological and clinico-pathological aspects of sweat secretion in their relation to the functions and lesions of the nervous system at all levels, from the peripheral to the cortical.
The method used for demonstration is that of Victor Minor, and a full description of the technique is supplied. The details of the various investigations are of considerable clinical interest, and for these, with the tables indicating the spinal segmental supply of the dermatomes from the sudatory standpoint, the original should be consulted.

J. S. P.


It will not surprise some neurologists that the conclusions of the writers, here quoted in extenso, are critical of the value of lipiodol injection.

1. In twenty-nine of thirty-one cases a definite clinical localisation was possible without the use of iodized oil as a diagnostic aid. We believe, therefore, that in the great majority of cases a definite spinal cord localisation can be made by careful clinical examination.

2. In ten animals injected with iodized oil intracisternally, definite evidences of leptomeningeal reaction, fat encystment and degenerative changes in the gray matter were found. We conclude, therefore, that the injection of iodized oil into the subarachnoid space is to be regarded as a dangerous procedure.'

J. V.


The Boltz test is rarely positive in non-paretic cases except in certain conditions of meningeal involvement, such as are but infrequently found in mental hospital work. It is not invariably positive in general paralysis, and a negative Boltz is of little diagnostic value. It agrees with no other usual test, though it probably disagrees less with the Lange than with, say, the globulin reactions. It readily becomes negative after malarial treatment, but mostly so after some years have elapsed since the first attack of malaria. The test does not depend only on the globulin content of the cerebrospinal fluid, or on the total protein increase, or the protein partition, though it probably does bear a relationship to the total tryptophane value of the fluid proteins, and perhaps also to cholesterol. If the test is to be further investigated, it would be preferable to use it in its Hopkins-Cole form, because (a) the Hopkins-Cole reagent can be standardized, (b) the colour reactions can be graded so as to render it quantitative, and (c) there is not the brown charring found when using acetic anhydride.

C. S. R.
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