Abstracts.

Neurology.

NEUROPATHOLOGY.

[151] Natural history of cerebral tumours (Histoire naturelle des tumeurs cérébrales).—C. De Monakow. L’Encéphale, 1921, xvi, 177.

The chief part of this interesting paper is an attempt to solve the difficult problem of the origin of gliomata. While many forms of tumour arise in organs which have been subjected either to gross trauma or to long-continued inflammation or irritation, this does not hold true for gliomata, a very small proportion of which follow injuries to the head. What, then, is their etiology, and what measures are we to take to prevent their occurrence? These are the questions which de Monakow attempts to answer.

His theory of their origin is a modification of the ‘cell-rest’ theory of Cohnheim. In the early development of the glial scaffolding of the nervous system, “in the development, grouping, and differentiation of the cells, the rhythm and harmony of the architectual ‘melody’ must be exactly followed”. This harmony may be marred in many ways. Colonies of glial cells after leaving the ependymal mother cells may develop too rapidly or too slowly, or may become displaced, and may thus become danger centres in later life. Such colonies of cells are frequently seen in the spinal cord, and may be the starting-point of gliomata. It is also known that when at an early stage of development the oral end of the neural tube is injured, the sacral end may show a tendency to overgrowth, resulting in reduplication of the lumbar region of the cord, in gliomatous formations due to over-activity of all the developing cells, or in cysts either in the substance of a tumour or near the central canal.

De Monakow considers that the colonies or groups of cells which leave the ependymal mother cells at an early stage are at feud with those which leave them at a later stage of development, and between these various groups there ensues a struggle for existence. Therefore, when the older cells tend to succumb to harmful influences, a group of younger cells which has been imprisoned, functionless, in the neighbourhood, may spring into activity, and by excessive proliferative activity give rise to a tumour. The older cells put up what fight they can against this intrusion, and sometimes or in some places are successful in destroying the invader. Thus arise gliomatous cysts, and encapsulated gliomata. De Monakow thinks that decompressive operation at this stage may aid the normal glial cells in their fight against the tumour-cells, as the excessive intracranial pressure and consequent diminution of blood-supply act in favour of the new-comers.
In his view the neuroglia adds to its functions of scaffolding and scavenging the nervous system the power of protecting the nerve-cells. Thus in neuronophagy newly-formed glial cells cluster round the nerve-cells to protect them from toxins or other harmful influences. A similar protective action is exerted in all forms of tumour formation. This theory seems to be getting a long way away from chemiotaxis, to which most authors attribute the migration of neuroglial and other cells.

Another view which may not receive general acceptance is that in brain tumours, as well as in other lesions of the brain tissue, an additional supply of cerebrospinal fluid is called for by a sympathetic mechanism involving the non-myelinated nerve-fibres in the choroid plexus. This seems to leave out of account such factors as the rise in the general intracranial venous pressure, and the obstruction to the circulation of the cerebrospinal fluid which results from the increasing bulk of the brain. These factors rest on solid scientific foundations, and are quite sufficient to explain the hydrocephalus which invariably accompanies brain tumours.

On the whole this article can scarcely be considered as a serious contribution to scientific knowledge. Many of the newer theories propounded seem to be based on very flimsy foundations of observation or correlated facts, and the expressions used in stating them are often so metaphorical that it is difficult to form a clear idea of the writer’s meaning. In the end we do not seem to get beyond the three theories of Cohnheim, Ribbert, and Adami, which, in combination, suffice to explain most forms of tumour in the nervous system as elsewhere in the body.

J. G. GREENFIELD.


This record of a series of experiments on kittens is of considerable interest to the neurologist. Internal hydrocephalus was produced, after withdrawal of the cerebrospinal fluid, by intraventricular injection of a suspension of lamp black in physiological saline. Intravenous injection of a strongly hypertonic solution of sodium chloride in hydrophalic animals produced a brief initial rise followed immediately by a marked depression, a phenomenon to be explained probably by rapid absorption of the fluid from the dilated cerebral ventricles. By means of the replacement of the ventricular fluid by a solution of potassium ferrocyanide and iron ammonium citrate, and by injection via the aorta, after the animals were killed, of a fixing solution of formalin with 1 per cent hydrochloric acid, a precipitate of Prussian-blue granules was formed where the ventricular fluid had escaped during the two hours that elapsed between the injection and the sacrifice of the animals, and macroscopic and microroscopic examination showed these granules to be massed immediately under the ventricular ependyma and to have penetrated the brain substance in zones of decreasing intensity. Anatomical and physiological proof is furnished by these experiments of the absorption of intraventricular cerebrospinal fluid by the ependyma, a fact which has a direct bearing on the problem, inter alia, of disseminated sclerosis.

S. A. K. W.
[153] **Contributions to the doctrine of cerebellar heredo-degeneration**

(Beiträge zur Lehre der zerebellaren Heredodegeneration).—**KARL SCHAFFER.** Jour. f. Psychol. u. Neurol., 1921, xxvii, 12.

Schaffer's long paper is based on the examination of two cases, one of cerebellar ataxia with idioey, and one of Marie's cerebellar heredo-ataxia, both of which are described in the minutest histological detail.

In the first (which Schaffer notes presented some resemblances to the picture of aplasia axialis extracorticalis congenita of Merzbacher, though he is not convinced of the specificity of the latter) the chief lesions were (1) 'Anlage'-defects, viz., abnormalities of convolutional pattern, hypoplasia of corpus callosum, cerebellum, and pons, doubly nucleated cortical nerve-cells, cytotectonic defects in the association areas of Flechsig; (2) Progressive degeneration of a systemic kind involving cerebro-cerebellopontine tracts (fronto-pontine, temporo-pontine, cerebellar peduncles). In the affected areas the degeneration involved ectodermal elements only, and not mesodermal, and Schaffer suggests this selectivity as regards both certain cortical layers and fibre-systems is a sign of a primary, i.e., of a heredo-degenerative process. The conservation of the cortical motor and sensory projection systems and the deterioration of the association systems was one of the peculiar features of the case, as was the degeneration of the commissural systems, and the suggestion is that motor and sensory systems are ontogenetically older and more resistive. The degeneration in the cerebellum was essentially neocerebellar, but both paleocerebellar and neocerebellar portions of the cortex were hypoplasic. In a word, the disease implicated definite organic anatomico-physiological complexes, apparently ab ovo.

As for the second case, diagnosed as cerebellar ataxia, the patient was a woman who had shown symptoms for some ten years and who died at the age of 42. The main lesions were: (1) In the cerebrum: diffuse and chronic cell changes all over and in all layers, but chiefly in the gyri angularis and fusiformis, in the cornu Ammonis, and in the deeper strata (layers 3 to 6); (2) In the cerebellum: a systematized degeneration of the neocerebellum only, implicating Purkinje cells, and the cerebellar nuclei; (3) In the hindbrain: a remarkable involvement of sensory protoneurons only of the seventh, eighth, and tenth cranial nerves. No vascular alterations were noted, so that the case appears again to be one of pure ectodermal degeneration in certain selected systems in cerebellum and hindbrain, while the additional cerebral changes perhaps represent a chronic spread of the same process, being equally of a neuronal nature.

S. A. K. W.


The authors have devised a method of estimating accurately the protein in the cerebrospinal fluid. This they do by precipitating the protein by sulphosalicylic acid, and reading by means of a colorimeter against a standard prepared at the same time from a blood-serum solution of known
protein content. The percentage error was found to be less than 5. The normal protein content was found to be between 18 and 38 mgm. per 100 cc. They regard any figure above 40 as pathological. The protein may be derived from exudation from meningeal vessels under pathological conditions. All sorts of pathological conditions showed an increase, and often this was the only sign of any abnormality in the cerebrospinal fluid. After repeated lumbar punctures the protein content diminished, suggesting a possible compensatory hydorrheea which may have some relationship to so-called lumbar-puncture headaches.

R. G. Gordon.


The author has carried out an elaborate investigation as to the causes and significance of changes in the viscosity of the cerebrospinal fluid. This is found to be increased by the amount of protein, by the alkalinity, and by the number of cells, but to little or no extent by variations in other constituents. Its diagnostic value is slight, and can only be regarded as confirmatory to other tests which would seem more certain and more easily carried out. The chief value of the paper would seem to be to dissuade ardent pathologists from expending their time and energy on the investigation of this property of the cerebrospinal fluid, since in the hands of several investigators it has proved to be practically without value.

R. G. Gordon.

SENSORIMOTOR NEUROLOGY.


The writers draw attention to the occurrence of symptoms of vertigo, loss of equilibration, etc., in an epidemic of encephalitis, and they describe the following features of a special clinical type of the affection:—

1. A pure type, which was found 12 times in 110 cases. In this type the onset is sudden, with vertigo; the patient often falls down, or staggers like a drunken man. There are general symptoms of malaise and feebleness, with vomiting in some cases. There are no eye symptoms, and no somnolence. Rest abates the symptoms, while walking exaggerates them, especially walking in street traffic, when a veritable crisis may occur, with transient diplopia and mental excitement.

The objective signs are usually only elicited when the eyes are moved to their extreme lateral range, and are worse on looking to one particular side. Rombergism is present, and the Babinski-Weil test with outstretched hands is positive (slow displacement of an arm to the right or left will occur after a few moments). The last described sign has proved more sensitive in the writer’s experience than rotation tests, thermic or galvanic tests, etc. Another sign will be found in some cases, consisting in a derangement of convergence either in the upward or horizontal direction. A minor sign