and lateral portion of the nucleus triangularis, the dorso-medial portion of the substantia reticularis and the fibres of the acoustic nucleus descending in the caudal portion of the posterior longitudinal bundle could all be destroyed without remission of the hypertonus.

In another experiment in which, as was afterwards found, the nucleus of Deiters and the grey matter of the formatio fasciculata (Roller's nucleus) were destroyed, there was a distinct diminution of extensor rigidity in the homolateral limbs, but a considerable excess of tone still remained. The conclusion is that, in addition to the vestibular nuclei, other centres play an important part in causing decerebrate rigidity.

Sherrington showed that decerebrate rigidity could be relieved by section of the anterolateral columns of the cord (the efferent path thereby being interrupted), and Spiegel has confirmed this. Portions of the anterolateral column go to the cerebellum and to the thalamus, but neither of these portions can be concerned with the excitation of decerebrate rigidity, for the spasm occurs in animals from which the cerebellum has previously been removed, and, as for the thalamus, it lies above the spasm-producing plane of section. There remain fibres (of the anterolateral column) which give off branches in the medulla and pons to end around large cells in the substantia reticularis. The conclusion is that the additional centres of static innervation consist of these large cells in the substantia reticularis: their efferent impulses travel in the reticulo-spinal tract. With this view two of the experimental findings harmonize, for, when the dorsomedial portion only of the formatio reticularis was destroyed the rigidity was not influenced, but when the lateral portion and the vestibular nuclei were destroyed the loss of tone was almost complete.

In studying the influence of the cerebellum on the centres of static innervation, Bernis and Spiegel confirmed a finding of Dusser de Barenne that stimulation of the anterior lobe (Bolk) caused in the decerebrate animal flexion movements of the fore limb of the side stimulated. The impulses which thus inhibit the extensor spasm travel, they find, by certain fibres which, after winding round the brachium conjunctivum, leave the cerebellum in the median part of the corpus restiforme. Whether their action is on the cells of the substantia reticularis or on the vestibular nuclei cannot yet be decided.

This tone-inhibiting action of the cerebellum the authors considered as probably associated with the fronto-ponto-cerebellar system of fibres and from their third series of experiments, which are, however, far from convincing, they drew the conclusion that both the frontal and temporal lobes of the cerebrum had a slight effect on tone-regulation.

J. P. MARTIN.

NEUROPATHOLOGY.

[221] The chemical composition of the spinal fluid: diagnostic value.—


Three conditions seem to be responsible for the chemical composition of the spinal fluid, whether the disease is of a metabolic or infective nature, viz.:—
1. The chemical content of the blood, producing an effect on that of the spinal fluid through the permeability of the cells of the choroidal plexus; the so-called haemato-encephalic barrier.

2. Mechanical agents such as tumours or haemorrhage into the spinal cord, producing congestion of the covering membranes.

3. Acute inflammatory bacterial or chemical conditions of the meninges, brain or cord.

In the hope of gaining some information concerning the variations in the general or selective permeability of the choroidal cells under the influence of disease, the authors examined the spinal fluids of 105 children. They were suffering from a variety of diseases, and clinically had one point in common—sufficient evidence of meningeal irritation to warrant lumbar puncture.

The following conclusions of diagnostic value were obtained:

1. The relation of spinal fluid chlorides to blood chlorides is not constant, and a determination of blood chlorides is not necessary or useful in diagnosis.

2. The protein content of the spinal fluid is increased in acute and chronic inflammations of the meninges and brain substance. In certain altered metabolic states accompanying infections or nephritis, some increase in protein can be expected.

3. Spinal fluid sugar should always be determined in relation to blood sugar. The normal relative value is from 40 to 60 per cent. of the blood sugar. High relative values are found consistently in epidemic encephalitis, meningismus, convulsions, acidosis and nephritis, in poliomyelitis of the bulbar type and in a certain number of so-called normal cases. Low relative values are found in acute and tuberculous meningitis with great regularity, and this is of considerable diagnostic importance.

4. The character of the toxin at work apparently determines to some extent the degree of general or selective change in choroidal permeability.

5. Chlorides, protein and sugar are likely to vary directly with the acuteness of the infective process. They may bear some constant relation to the disease itself.

6. A knowledge of the spinal fluid chemistry is helpful diagnostically in many conditions of the central nervous system; according to the experience obtained in this series, most strikingly so in tuberculous meningitis, less so in purulent meningitis, encephalitis and poliomyelitis.

7. Calcium and phosphorus in the spinal fluid bear no relation to their content in the blood or to the clinical condition of the patient.

R. M. S.


Examination of the cerebrospinal fluid in doubtful but suspected cases of encephalitis lethargica is of the greatest importance. The constant negative findings of the Wassermann reaction, Pandy's test, Lange's test, and an absence of increase in the cell-content, will exclude many conditions which may give rise to confusion. When a negative Wassermann reaction and a negative Lange reaction have been found, the sugar and the chloride content
of the fluid may be of value in differentiating epidemic encephalitis from other diseases, especially tuberculous meningitis. It is essential that the methods employed should be the same in all cases.

R. S. C.

[223] Biological reactions in cerebrospinal fluid and blood of untreated general paralytics (Graphiques représentant l'évolution des réactions biologiques du liquide céphalorachidien et du sang chez les paralytiques généraux non traités).—A. BailLl and A. SÉZARY. Revue neurol., 1924, xxxi, 460.

From a study of twenty-one cases of general paralysis the authors conclude that marked variations of a spontaneous and temporary character may be encountered in the leucocyte count and albumen content of the cerebrospinal fluid. The Wassermann reaction is more stable but is nevertheless subject to temporary fluctuations and consequently caution must be exercised in estimating the value of therapeutic intervention. It is not legitimate to conclude that alterations in the serological findings are due to the effects of treatment unless the improvement is maintained for a long period.

R. M. S.


The author is convinced that postpuncture headache is caused by trauma to the spinal dura sufficient to result in excessive leakage of cerebrospinal fluid to a point at which the brain is left without a water cushion. If, during puncture, a large rigid needle is used, the see-sawing of the dural sac over this needle by movements of the spinal column will result in excessive trauma to this membrane. A small needle with a point that is round, tapering and sharp should be used. Gauge 22 is the most suitable.

R. M. S.


Attention is drawn to the fact that, while some authors have found a positive Wassermann reaction in both the blood and the spinal fluid in all their cases of general paralysis, others have obtained a positive reaction in only blood or spinal fluid in some cases. Emphasis is laid upon the necessity of having an adequate amount of spinal fluid for testing. In this series Wassermann tests were done on blood and spinal fluid in 143 cases. In three cases both blood and spinal fluid were negative, and in five the spinal fluid was negative and the blood positive.

W. G. W.


The inorganic constituents of the blood and cerebrospinal fluid were deter-
mired in seventeen cases of spasmodia. Except in the case of the calcium content of the blood no significant changes from the normal were found. The usual decrease of the calcium content of the blood was found in the acute phase of the illness, but the calcium figures for the cerebrospinal fluid were almost the same before and after recovery.

W. G. W.

[227] The changes in the corpus striatum resulting from senility (Über die Veränderungen des Striatum im normalen Senium).—OSEKI. 

It is well known that considerable abnormality may be found in the corpus striatum of patients who have shown during life no disturbances of tonus or movement, and the object of this paper is to discover the nature and degree of the changes in the striatum which may occur in old age without causing any recognized clinical signs or symptoms; or in other words what changes and what degree of change may be regarded as normal. Oseki has examined the brains of ten patients of ages from seventy to eighty-five, none of whom had during life presented any abnormal neurological signs. He found pronounced conditions of désintégration lacunaire, with changes in all the structures of the part, nerve cells, nerve fibres, glia and vessels. The nerve cells showed extensive fatty degeneration which affected the large and even the small cells, though the latter are usually 'lipophobic.' There was an increase of the lipofuchsins and a disappearance of the normal cell plasma; only some of the lipoids took the colour with Scharlach R. The nerve fibres showed no definite diminution in number but a marked diminution in size; the fibres were very thin in consequence of the reduction of their medullary sheaths; the interfibrillary glia cells were increased in number, particularly around the damaged ganglion cells, where they were evidently concerned in neuronophagy. There was slight but definite increase of glia fibres. The vessels in all cases showed the most severe changes. The vessel walls were heavily calcified, even the smallest arteries and in some cases the capillaries containing deposits. This calcification was most advanced in the globus pallidus. In spite of the great degree of change in the vessel walls there was no definite softening in any part of the region examined. In nearly every case there were small spongiform masses round the vessels which could only be explained on the supposition that the more delicate structure, the parenchyma, had disintegrated, while the more resistant glia had remained, and even increased (état lacunaire). Extensive though this process might be, however, the greater part of the cells was in every case intact. In other parts, probably as a result of contraction elsewhere, the feet of the glia cells had withdrawn from the vessels, whorls of glia were formed and sometimes corpora amylacea were present in abundance around the vessels; these latter lay in the outer wall of a free space, by which each vessel was surrounded. This condition is regarded as a peculiar form of the état gruyère.

The importance of bearing in mind when examining the basal ganglia that these changes are not abnormal in elderly people hardly requires to be emphasized.

J. P. M.

Warkany briefly reviews the various hypotheses that have been put forward to explain the failure of the light reflex of the pupil in syphilitic nervous conditions, and concludes that the essential lesion is likely to lie between the corpora quadrigemina and the oculomotor nucleus. Since investigation of that region by ordinary methods—i.e., by studying nerve cells and fibres or axis cylinders—has given no positive result, he determined to study the state of the glia. In six cases he found a definite thickening of the glia in the subependymal zone around the aqueduct of Sylvius. No indication could be found of any change close round the oculomotor nucleus.

The author points out that loss of the light reflex is usually accompanied by loss of the vestibulo-pupillary reflex and certain other pupillary movements, and that an interruption of fibres at the site of the glial overgrowth found by him, though it might interrupt the light-reflex arc, would not cause any break in the vestibulo-pupillary arc; and hence a lesion in this position is unlikely to be the whole explanation of the Argyll Robertson phenomenon.

J. P. M.


From a review of cases the author concludes that the symptoms of these diseases are due to a chronic inflammation of mesodermal tissue (vessels and connective tissue) which gravely interferes with the health of nervous tissue. Tabes is not a systematic sclerosis of the posterior columns but rather a radiculitis secondary to a chronic meningitis.

In tabes the possibility of vascular lesions must be considered in accounting for isolated symptoms, and of sympathetic lesions in accounting for trophic disturbances and the dissociation between muscular tone and tendon reflexes.

R. G. Gordon.


The histological changes of trichinosis encephalitis may be defined as diffuse, degenerative and inflammatory, with marked hyperplastic phenomena in the pia-arachnoid, choroid plexus and glia. Typical of acute encephalitis, they are associated with the presence of trichina embryos in the cerebral parenchyma, nodules, infiltrated adventitial and subarachnoid spaces, and in the cerebral ventricles. The changes strikingly resemble those of other types of acute nonsuppurative encephalitis, such as those seen in Heine-Medin's disease, and especially in typhus fever. But while in the latter variety the nodules are said to be especially abundant in the medulla, in the authors' case
of trichinosis they were rather evenly distributed throughout the brain, affecting the medulla probably less than other portions. In typhus fever the perivascular infiltration, mainly in the form of plasma cells, is preferably round the small blood vessels and capillaries, while in this case they were mostly of large lymphocytes round the larger blood vessels. The outstanding features—the nodules and perivascular infiltrations—are to be considered as manifestations of a reaction against the infection. This was in the form of trichina embryos, which were thus responsible for the excessive cellular proliferations in the nodules and round the blood vessels. The inference is that in other types of encephalitis, the exact etiology of which has not yet been established, the presence of nodules and perivascular infiltrations denotes an infection. Besides the infiltrations, nodules and syncytia—purely local reactive phenomena—a diffuse degeneration with accumulation of lipoids constituted an important finding in this case and was interpreted as a manifestation of intoxication caused by trichina in the brain tissues. The presence of these worms in the brain seems to settle the much discussed question as to how they travel from the intestines to various tissues, especially the muscles. The embryos certainly could not have covered the long distance from the alimentary canal to the brain by active, voluntary movements through the tissue spaces, and it is reasonable to assume that they were carried there by the blood stream, just as any other substance (e.g., carcinoma cells) is carried to the brain from remote organs (e.g., lymph and blood channels). Reaching the cerebral capillaries, trichinae provoke in them reactive phenomena—swelling of the endothelial and adventitial cells. They then pierce the walls and land in the brain tissues, where they produce the nodules, syncytia and perivascular infiltrations. By the nodules and syncytia some of the embryos are kept from further propagation, being, as it were, destroyed on the spot, while others, like any other waste, are carried to the perivascular channels, and from these to the subarachnoid space and the ventricles.

R. M. S.

best advantage in other chronic heredofamilial nervous diseases, such as heredo-spinal and cerebellar forms, in which an underdevelopment—hypoplasia—is always noticeable in one segment: that is, in the spinal cord or cerebellum.

Tay-Sachs disease is a pan-segmental process in contrast with the familial spinal amyotrophy of Friedreich or Marie, which are afflictions of a single segment.

**R. M. S.**

[232] **Sarcomatosis of the brain.**—B. M. FRIED. Arch. of Neurol. and Psychiat., 1926, xv, 205.

A MAN, age 33, became suddenly ill with symptoms of a brain tumour, which could not be localized. He died two months after the onset of the first symptoms; the necropsy showed to the naked eye merely a small tumour nodule, but the microscope revealed the widespread presence of tumour cells throughout the entire cerebrum, cerebellum, pons, medulla oblongata and meninges. The growth proved to be composed of small round cells which showed numerous mitotic figures, and had a definite perivascular arrangement. The parenchyma and interstitial tissue of the brain showed extensive secondary degenerative changes. Tumour cells undergoing mitoses occurred in the adventitia of vessels possessing an intact tunica intima, intima pie and limitans gliae; they were not of glial, nervous or ependymal origin.

The author concludes that the presence of tumour cells in the vascular adventitia, which has been shown to possess neoplastic potentialities, indicates that the tumour was a true sarcoma of the brain, originating from the adventitial cells of the Virchow-Robin spaces.

**R. M. S.**

[233] **Mucin degeneration of the neuroglia** (La dégénérescence mucocytaire de la névroglie).—PAGES, BENOIT and PÉLISSIER. L'Encéphale, 1925, xx, 587.

This paper is an attempt to assess the diagnostic value of the presence of mucin (1) in the brain and (2) in the cerebrospinal fluid. Described first by Grynfeltt in 1923 in a senile brain, it has been more frequently found since in the sequelae of encephalitis lethargica (v. this Journal, vol. V, p. 364). The author considers that mucin originates in a degeneration of the interfascicular glia, which during its formation frequently disintegrates and disappears. It is always found principally in the white matter of the brain but may invade the cortex as well. When formed it tends to be carried along the perivascular channels to the cortex and to the subependymal layer, whence it may break through into the ventricles and appear in the cerebrospinal fluid. As a test for its presence in this fluid the authors recommend Derrien's method, which consists in underlying the cerebrospinal fluid in a test tube with a syrupy (i.e., saturated) solution of citric acid. If mucin is present, even in traces, a haze forms at the junction of the two fluids. They used this test in a series of 126 cases, and obtained positive results in postencephalitic states (27 out of 31 cases), generalized epilepsy (5 out of 6 cases), in 6 cases
of lacunar cerebral sclerosis, 2 of uræmic delirium and 2 of mental confusion of infective origin. The test was less constantly positive in dementia præcox and cerebral syphilis. It was negative in general paralysis, tabes dorsalis and disseminated sclerosis, as also in all normal fluids. The test may therefore be of value in distinguishing general paralysis from other syphilitic brain diseases, especially those associated with mental confusion, and also as demonstrating an organic basis for mental or hysterical symptoms.

In the second part of their paper the authors discuss the relationship of mucin degeneration to confusional states, a relationship previously indicated by Buseaino, who described a similar degeneration of the brain as "grape-like disintegration." According to Cajal the insulation of axons may vary in completeness according to the extension or retraction of the processes of the interfascicular glia (oligodendroglia). Similarly it is possible that degeneration of these cells may lead to defective insulation and consequent confusion of mental processes.

J. G. Greenfield.

SENSORIMOTOR NEUROLOGY.


The figures herein given are based on the examination of 318 cases of the disease. In some respects, as is to be expected, they differ from those of previous investigators; and while they cannot be conveniently summarized attention may be directed to some of the points which they raise.

The percentage of men is 58, of women 42. This is rather contrary to accepted opinions, but is explained to some extent by the nature of the clinical material, a large part of it being derived from the artisan class. Between the ages of 21 and 30, 38.6 per cent. of the cases commence, and 30.3 per cent. between the ages of 31 and 40. The frequency of neurological symptoms as the first indication of the disease takes place in the following order: difficulty in walking (64 per cent.), paraesthesiae (26 per cent.), sphincter trouble (24 per cent.), giddiness (19 per cent.), diplopia (18 per cent.), pains (18 per cent.), dysarthria (6 per cent.), affection of upper limbs (4 per cent.), facial weakness (1 per cent.). It is unusual, perhaps, to find so high a percentage commencing with pains (chiefly in the back); this may be to some extent the expression of muscular weakness. When the disease is fully established, in the sense that the diagnosis is certain, the following is the list of symptoms in order of frequency: spastic lower extremities, loss of abdominal reflexes, nystagmus, intention-tremor, pallor of the temporal halves of the optic discs, sphincter disorder, scanning articulation, paraesthesiae, 'psychical alterations.'

A feature is made of the curious finding that the symptoms are not uncommonly aggravated after lumbar puncture; sometimes, indeed, within two or three weeks a previously unremarked symptom will make its appear-