[180] **A comparison of the tension of the retinal arteries and of the cerebrospinal fluid** (Tension rétinienne et tension du liquide céphalorachidien).—H. CLAUDE, A. LAMACHE and J. DUBAR. *L'Encéphale*, 1927, xxii, 1.

THE systolic and diastolic pressure of the retinal arteries can now be studied by the tonometer of Bailliart, in the use of which ophthalmoscopic examination of the retinal artery on the disc during compression of the eyeball shows pulsation beginning at diastolic pressure and ending at systolic pressure. The authors compared the results obtained by this instrument with the blood pressure and cerebrospinal fluid pressure. The normal pressures in the retinal artery vary from 600 to 700 mm. of water for the systolic and from 300 to 350 mm. for the diastolic. As a general rule it was found that the diastolic retinal pressure bore a close relationship to that of the cerebrospinal fluid and varied with it when it was artificially raised or lowered. When the cerebrospinal fluid pressure was normal the retinal diastolic pressure varied with that of the general arterial system. But sometimes the retinal pressure was found to be unexpectedly high by comparison both with the arterial and the cerebrospinal fluid pressure. In some cases this appeared to be due to emotional stress at the time the retinal pressure was taken. In other cases, as in general paralysis, it was probably due to some local blockage to the outflow from the retinal veins or lymphatics. The authors recommend the examination of retinal pressure both in cases of suspected brain tumour where lumbar puncture is contraindicated, and in cases in which lumbar puncture has proved the existence of raised intracranial pressure when it is considered advisable to watch the effects of hypertonic solutions in reducing this.

J. G. GREENFIELD.

NEUROPATHOLOGY.

[181] **Pathologic changes in Huntington's chorea.**—C. R. DUNLAP. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 867.

THE late Dr. Charles Dunlap has in this paper contributed a minute and painstaking account of the pathological changes in seventeen cases of Huntington's chorea. It would be hazardous to attempt a summary of the many points discussed, but Dr. Dunlap's views seem to amount to this, that in pure and well developed Huntington's chorea there are constant lesions of definite type in the corpus striatum and in the cerebral cortex and marrow which are characteristic enough to make a postmortem diagnosis of Huntington's chorea at least probable if not certain, for the peculiar combination of cortical changes and basal nuclear lesions is not reproduced in any other disease.

R. M. S.

[182] **The neuropathological findings in a case of acute Sydenham's chorea.**—L. H. ZIEGLER. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 273.

THE study of a case under the care of the author and of the literature shows that this syndrome is associated with chromatolysis of practically all cells of the central nervous system, with swelling of nuclei and excentric displacement; destruction of some neurones especially those of the sixth nerve and calcarine cortex, where glia cells were much proliferated; neuronophagia, fatty deposits in the large cells of the motor cortex and globus pallidus; fat in the perivascular spaces and petechial hæmorrhages in a small area near the dorso-medial aspect of the restiform body of the medulla. Recent reports show changes in the corpus striatum similar to those found in the lesions of epidemic encephalitis. Some of the elements of the brain may be less resistent to toxins or infections, or the latter may have a selective action. Though recovery takes place awkwardness of certain movements which persist suggests an inherently inferior motor system, or maybe a residuum of the disease.

R. G. G.

[183] **The histology of post-vaccinal encephalitis.**—J. R. PERDRAU. *Jour. Pathol. and Bacteriol.*, 1928, xxxi, 17.

PERDRAU has examined the lesions in the brain and spinal cord of three fatal cases of post-vaccinal encephalomyelitis. The onset of the disease was seven, nine and twelve days respectively, after a normal vaccination. He lays particular emphasis on the nature of the lesion and confirms McIntosh and Turnbull in finding perivascular zones of softening or, more correctly, of demyelination with relative preservation of the axis-cylinders. The cellular reaction in these areas is characterised by proliferation, mitosis and karyorrhexis in wandering cells, apparently of endothelial origin, and by early changes in the microglia leading to the formation of granulo-adipose cells. Demyelination was complete even in a case which ended three days after the onset of symptoms.

Comparing this type of lesion with that of poliomyelitis and encephalitis lethargica, he finds that although they resemble one another in the perivascular cellular exudate (lymphocytes and plasma-cells), yet post-vaccinal encephalitis is sharply distinguished by the tendency to perivascular demyelination. This lesion is in fact histologically identical with that found in early cases of disseminated sclerosis, and similar to that in some early cases of Schilder's encephalitis. It is also similar to that described in the published accounts of encephalitis or myelitis following smallpox, measles, and antirabic inoculations. The possibility that paralysis occurring after varicella and typhoid fever has a similar pathological basis is also suggested. He is inclined to the view put forward in 1925 by Bastiaanse, that the essential virus causing this form of encephalomyelitis is not identical with that causing the eruptive fever, but rather is an independent virus stimulated to activity by the febrile disease. In this connection he refers to the view put forward thirty years ago by Sottas

and Pierre Marie that disseminated sclerosis owes its origin to an attack by one of the eruptive fevers. While this view is not generally held at the present time, the similarity of the early stages of the lesion in disseminated sclerosis, Schilder's encephalitis and post-vaccinal encephalitis suggests a resemblance in the etiological agent, and leads to the hope that the discovery of the virus of one may throw light on that of the others.

J. G. GREENFIELD.

[184] **Zoster myelitis. The acute tephromyelitis of herpes zoster** (La myélite zostérienne. La tephromyélite de l'herpes zoster).—J. LHERMITTE and M. NICHOLAS. *L'Encéphale*, 1927, xxii, 245 and 313.

THE authors report a case of herpes zoster affecting the skin area supplied by the first to the fourth left cervical segments and followed by severe neuralgia. The patient died of bronchopneumonia eight weeks after the onset of the herpes. In addition to the typical lesions in the dorsal root ganglia they found inflammation and necrosis of the posterior horn on the left side of the second and third cervical segments of the spinal cord. In a wide survey of the literature they found that this histological finding was by no means uncommon. Not only so, but in many cases clinical evidence of myelitis was afforded by increased tendon jerks or absent abdominal reflexes on the side of the eruption, or by wasting of muscles supplied by nerves other than those implicated in the cutaneous eruption. For example, oculomotor palsy might be associated with facial herpes, or facial palsy might accompany an eruption on the neck or chest. It is not, therefore, possible in all cases to attribute the accompanying paralysis to pressure on the motor root by the swollen dorsal root ganglion.

With regard to the vexed question of the pathogenesis of the skin lesion, the authors refer to the work of several authors, in particular Lehner, who found that the skin lesions even from the earliest stages showed the characteristics of an inflammatory dermatitis. They consider it as proved that the virus of zoster attacks directly both skin and dorsal root ganglia, that it frequently reaches the spinal cord, and that it may even spread within the central nervous system.

J. G. GREENFIELD.

[185] **Metastatic carcinoma of the central nervous system.**—N. W. WINKELMAN and J. L. ECKEL. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 1.

TWENTY-THREE cases are presented and the literature is reviewed. The authors point out that the breast and prostate seem to predominate as the seats of primary tumour sending metastases to the central nervous system. These metastases may be in relation to the dura, to the nervous tissue itself, or within the perivascular spaces and blood-stream. Metastatic tumours do not differ from primary tumours of the central nervous system in their clinical manifestations. A history of operations for tumour especially on the breast or prostate is of the greatest importance in diagnosis, especially in view of the fact that metastases may recur years after such operations.

R. G. G.

[186] **Colloidal gold reactions with spinal fluids contaminated with blood.**—H. G. MEHRTENS, H. A. WYCKOFF, and R. M. DAVIS. *Arch. of Neurol. and Psychiat.*, 1928, xix, 296.

SPINAL fluids which are contaminated with blood, but from which the cells are removed by centrifugalisation before hæmolysis occurs, produce a colloidal gold curve altered in intensity only ; the type of the curve remains the same.

Spinal fluid from patients with general paralysis made normal by treatment, on contamination with blood plasma, shows a tendency to revert to the former general paralytic type of curve. The original colloidal gold curve of spinal fluids experimentally contaminated with the patient's blood can be approximately reconstructed, provided hæmolysis has not occurred. There appears to be reason to believe that similar reconstruction can be made of curves of spinal fluids accidentally contaminated.

R. M. S.

[187] **The permeability of the haemato-encephalic barrier in nervous and mental diseases** (Sur la perméabilité de la barrière nerveuse centrale dans les maladies mentales et nerveuses).—S. BAU-PRUSSAK and L. PRUSSAK. *L'Encéphale*, 1927, xxii, 176.

OF the many tests for increased permeability of the barrier between the blood and the central nervous system, the bromide test of Walter and Hauptmann appears to be the most delicate, as by it we can estimate not only slight degrees of increase but also diminution of the permeability of the barrier. This test has been applied recently to the diagnosis of mental diseases, since the work of Stern and Gautier showed that any substance which can pass from the blood into the cerebrospinal fluid will also, under the same conditions, pass into the tissues of the brain, and since Monakow demonstrated certain changes in the choroid plexus in cases of mental diseases such as schizophrenia. The test is performed as follows : The patient is given for three to five days a daily dose of sodium bromide corresponding to 30 gr. for every five stones of body weight. On the day following the last dose specimens of blood and cerebrospinal fluid are collected at the same time and the percentage of sodium bromide in them estimated by a colorimetric method. (Precipitate the protein from an aliquot quantity of cerebrospinal fluid or blood plasma by trichloracetic acid. To 1 c.c. of the filtrate add 0·2 c.c. of ·5 per cent. gold chloride, and compare in the colorimeter with a standard sodium bromide solution similarly treated.) The percentage of bromide in the blood plasma, divided by the percentage in the cerebrospinal fluid, gives a quotient of permeability which is normally between 2·9 and 3·3. (The lower the quotient the greater the permeability : v. this JOURNAL, viii, 270.)

The authors investigated 108 cases by this test. These fell into five main groups. (1) Mental disease including general paralysis, 26 cases. (2) Organic disease of the brain including meningitis, 39 cases. (3) Affections of the spinal cord, 26 cases. (4) Affections of peripheral nerves and inorganic nervous

diseases, 14 cases. (5) General diseases, three cases. Of the mental diseases general paralysis was usually associated with an increased permeability, but after malarial treatment this became normal. Schizophrenia was often associated with a lowered, and never with a raised permeability. In arteriosclerotic and senile dementia a raised permeability was present. In acute meningitis and in syphilitic meningitis the permeability was increased, but not in encephalitis lethargica or in subarachnoid hæmorrhage. In tumours of the cord the permeability was increased in the lumbar fluid. In epilepsy, hysteria, affections of the peripheral nerves and in general diseases not affecting the nervous system the quotient fell within normal limits. In seven cases where the cisternal fluid was compared with the lumbar fluid, it was always found to have an abnormally low percentage of bromide, indicating that the normal values as given by Walter only hold true for the lumbar fluid. This observation indicates that it is necessary to restandardise the test on the cisternal fluid if it is to be applied to the diagnosis of mental diseases.

J. G. GREENFIELD.

[188] **The pathology of amyotonia congenita.**—ROY R. GRINKER. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 982.

AMYOTONIA congenita has been linked by some to the myopathies and by others to the infantile type of progressive muscular atrophy, and the pathological reports show considerable divergence. In the case reported by Grinker the symptoms were clearly those of amyotonia and the essential pathological changes were entirely within the confines of the lower motor neurone. The absence of glial reaction, scar-formation or vascular change suggests that this was not an active process, and certainly not an inflammatory or toxic condition. The simple paucity of cells of the anterior horn and the presence of cells which were either small, rounded and poor in chromatin, or long and fusiform, suggested a developmental defect. The muscle changes were most marked in the skeletal muscles. There were no fatty or degenerative changes, but simple muscular fibres in miniature containing all the markings of the normal muscle. Hypertrophic fibres were not noted, although they have frequently been described by others. The smooth muscle did not share in the process, and correspondingly the cell group of the lateral horn was well developed.

The author concludes that amyotonia congenita and the Werdnig-Hoffmann type of progressive muscular atrophy are identical as far as the pathological changes in the muscle and the site of the changes are concerned, but differ as to the type of lesion in the cord. In one a paucity of cells of the anterior horn with the presence of abnormal cells is found; in the other actual degeneration of the ganglion cell and neuronophagia. The fundamental difference in the two groups is probably only a matter of the time at which the ganglion cells are affected. All the infantile degenerative muscular atrophies may be classed according to this pathological criterion and there is no definite clinical differentiation between them, each type merging into the other. R. M. S.

[189] **Lesions of nerves in experimental lead poisoning** (Lesions des nerfs dans l'intoxication saturnine expérimentale, etc.).—J. M. DE VILLAVERDE. *Travaux du lab. de récherches biol.*, 1926, xxiv, 158, 267.

THE author has set out to examine the results of lead poisoning on the peripheral nervous system by the aid of Cajal's silver impregnation methods. He found that the nerve suffered as a whole, neurofibrils, axoplasm, myelin sheath and sheath of Schwann all being affected. This led to some curious anomalies in the microscopic appearances, for axis-cylinders might remain dead for some time without being broken up and removed by phagocytic activity either in the Schwann or other cells. Similarly, even when an axis-cylinder appeared to be destroyed very little attempt at regeneration took place, and this was always abortive. This might be attributed to some extent to the lesions found in the anterior horn cells, which are described as shrunken and as nowhere presenting the phenomena of axonal reaction. The motor endings on the muscle fibres were also studied and found to present changes similar to those found by Boecke after nerve section, but of less degree. There was an early loss of the network of Boecke in the end-organ and an early degeneration of fine intracapsular branches, with varicosity of the terminal part of the muscular twig. The poison therefore appears to attack all parts of the neurone simultaneously and independently, a fact which explains the slightness of the recovery which may be expected in severe cases. The paper is rich in microscopical detail and illustrations and contains a full bibliography of previous work on the subject.

J. G. G.

SENSORIMOTOR NEUROLOGY.

[190] **Subarachnoid hæmorrhage from a medico-legal point of view.**—WILLY MUNCK. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 484.

THE author points out that an isolated subarachnoid hæmorrhage may be the cause of sudden death and describes nine such cases. He remarks that in the absence of a reliable history it is practically impossible to determine whether the hæmorrhage is spontaneous or due to trauma, or to ascertain its starting-place.

R. G. G.

[191] **Intraventricular hæmorrhage.**—I. J. SANDS and M. LEDERER. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 360.

THREE cases are described and the authors point out that premonitory symptoms of intraventricular hæmorrhage are usually absent though the patient may show signs of cerebral arteriosclerosis or may be suspected of cerebral aneurism. An acute onset of cranial symptoms with the early appearance of coma, persistent blood-stained spinal fluid, the presence of repeated tonic spasms of the entire somatic musculature with the absence of classical signs of paralysis, should lead to the diagnosis of intraventricular hæmorrhage.

R. G. G.