[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.

[192] **Occlusion (embolic) of the posterior inferior cerebellar artery confirmed at autopsy, with pains and hyperalgesia to cold** (Un cas d'occlusion (par embolie) de l'artère cérébelleuse postérieure inférieure verifée à l'autopsie, avec des douleurs et de l'hyperalgésie au froid).—KNUD WINTHER. *Acta Psychiat. et Neurol.*, 1927, ii, 294.

A CLINICOPATHOLOGICAL account of a fatal case of Wallenburg's syndrome in a spinster of 64. The chief points of interest lay in the appearance of spontaneous pains and dysæsthesiæ in the affected side of the face, some three months after the ictus. She described a sensation of wetness or of "rain" behind the left eye. Objective examination showed an analgesia over the left face; there was an impaired appreciation of warmth over that area and a complete failure to recognise cold objects. Two months later burning pains appeared in the right arm, and an altered response to thermalgesia tests became apparent. She over-reacted intensely to contact with cold objects applied to the left face or right side of the body; the resulting sensation was one of pain; the actual *coldness* of the object could not be recognised at all. Ability to detect pain and warm stimuli was impaired but not lost. Winther remarks that the over-reaction to cold and the presence of spontaneous pains recall vividly the features of the thalamic syndrome, but in this case the pathological evidence places the lesion well below that level. This particular manifestation is on a par with the central pains due to focal lesions within the pons as described in syringobulbia. The author remarks on the similarity to the manifestations of causalgia and queries whether the sympathetic system may not share in the causation of the pain. He remarks on the presence of a Claude Bernard-Horner syndrome in his case, although this latter may exist with the presence of pain and dysæsthesiæ.

Another interesting clinical feature of this particular case lies in the symptom of obstinate hiccough.

M. C.

[193] **Thrombosis of the spinal arteries** (La thrombose des artères de la moelle épinière).—HELGE VEDSMAND. *Acta Psychiat. et Neurol.*, 1927, ii, 177.

THE role of primary vascular changes in the production of myelomalacia is imperfectly understood. In numerous cases of syphilitic myelitis it is claimed that the vascular changes are primary and not the result of an inflammatory process. The clinical manifestations of spinal arteriosclerosis, however, are almost unknown, except for a proportion of those cases spoken of as senile paraplegia. Vedsmand gives details of eight cases which he has diagnosed as instances of thrombosis of the spinal arteries: in only two, however, was autopsy verification possible. These two cases were characterized pathologically by complete necrosis of the dorsal cord with ascending and descending degenerations. Vascular occlusions were demonstrable and there was no

evidence of an inflammatory process. All of the eight cases were characterized clinically by a rapid onset of flaccid paraplegia, preceded for a varying period by pains and dysæsthesiæ in the legs. Sphincter control became lost, and sensation grossly impaired over the paralysed members. Superficial and deep reflexes were in abeyance. Except for the two cases which ended fatally, more or less complete recovery was the rule. Syphilis was admitted by six of the patients, although the blood and spinal fluid reactions were not strongly positive.

M. C.

[194] **The evolution of an encephalitic dystonia into a hypertonic-akinetic syndrome resembling Wilson's progressive lenticular degeneration.**—S. BROCK and S. KATZ. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 460.

A CASE of encephalitis is described in which observations were carried out at intervals of three years. The chief feature of the disease on the first examination was dystonia with marked involuntary movements. Three years later there was extreme rigidity and abnormal posture with no involuntary movements. The authors attribute this alteration to progressive changes in the lenticular nuclei and compare it to the clinical features found in Wilson's disease.

R. G. G.

[195] **Encephalitis after vaccination.**—D. WIERSMA. *Acta Psychiat. et Neurol.*, 1927, ii, 167.

AN interesting and valuable contribution to our knowledge of the neurological complications of vaccinia.. The author has had under observation eleven cases of encephalitis occurring at intervals from nine to 19 days after vaccination. Convulsions occured in five of the cases; in three there were definite palsies; meingeal symptoms were seen in four; trismus in two; in seven cases a Babinski response appeared. Muscle tonus was diminished in all the cases and frequently the tendon jerks were unobtainable. Death occurred in five of his cases, but in two only was an autopsy allowed. Contrary to the opinion of Lucksch, Wiersma agrees with most other writers in regarding vaccinial encephalitis as distinct from encephalitis lethargica; the anatomical picture, too, differs in several important particulars. Thus in vaccinial encephalitis the perivascular infiltration often contains polymorphs, and the white matter is affected more than the grey. Wiersma discusses at length Bastiaanse's theory that a latent virus in lethargic encephalitis is activated by the vaccination.

Although there is a certain amount of suggestive clinical and pathological evidence, the possibility of an association between vaccinial encephalitis and polioencephalitis is not discussed.

M. C.

[196] **Encephalitis periaxialis concentrica.**—JOSEPH BALO. *Arch. of Neurol. and Psychiat.*, 1928, xix, 242.

A LAW student, age 23, became ill with aphasia and agraphia; then followed weakness of the inferior branch of the facial nerve and disappearance of the cremasteric reflex on both sides and of the abdominal reflexes on the right side. Later, a hemiplegia of the right side, loss of sphincter control and a total aphasia developed. Before death, tonic spasms occurred in the right arm and leg; trismus developed, and the patient lost consciousness. Simultaneously, the right knee reflex and the achillis reflex became exaggerated, and the Babinski phenomenon appeared on the right side. At the beginning, the fundi were normal; later, optic neuritis developed. There was moderate elevation of temperature at this stage. The Wassermann reaction was negative, and a slight leucocytosis was present. Examination of the cerebrospinal fluid did not reveal any pathological condition. Four months after the onset of the disease an operation was performed, but no cerebral tumour found. The patient died shortly afterwards from intracranial hæmorrhage.

Postmortem examination did not reveal any pathological conditions in the internal organs. In the white matter of the brain foci were found varying in size from that of a lentil to that of a pigeon's egg. Some resembled gray softening; others were characterised by concentric layers composed of normal white matter alternating with layers of gray softening. In the latter fatty degeneration of the medullary sheaths was found. The most characteristic alteration of the neuroglia was the formation of giant glia cells. These neuroglia cells showed degeneration and were the subject of gliaphagia. Near the foci the periadventitial spaces were filled with products of degeneration, such as granule cells, lymphocytes and plasma cells. In areas of the brain widely distant from the pathological changes described above hyaline thickenings of the arterial coats were found. The cortex, basal ganglia and spinal cord were normal.

The changes about the vessels indicate the inflammatory character of the process. Syphilis, apparently, does not play a part in the etiology. Bacteriological examination did not reveal any micro-organisms. Intracerebral inoculation with material from the brain into rabbits did not transmit the disease. One would infer from this that the causative agent is a lecithinolytic ferment that exercises its action from different foci at different intervals. Among the diseases known to date, the condition in the case reported resembles acute multiple sclerosis and the encephalitis periaxialis diffusa of Schilder. The disease differs from Schilder's encephalitis because of its focal character. It differs from multiple sclerosis because in that condition both the brain and the spinal cord and both the gray and the white matter are affected. It resembles most the case of Barré, Morin, Draganesco and Reys and the third case of Marburg described in his work on acute multiple sclerosis. Balo suggests the term Leuko-encephalitis periaxialis concentrica for the pathological condition described in his paper. R. M. S.

[197] **Encephalitis periaxialis diffusa (Schilder).**—GEORG SCHALTENBRAND. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 944.

A SCHOOLGIRL, age 14, was admitted because of a suspicion of appendicitis. When first examined she was apathetic and had a right-sided facial weakness and optic neuritis. During her sojourn in hospital she gradually developed choked discs and signs of extensive bilateral lesions of the brain, so that the probable diagnosis of encephalitis periaxialis diffusa was made. She died after an illness of three months' duration, in a state indicating practically complete disappearance of all cortical functions.

The postmortem examination of the body revealed nothing of importance pathologically. The brain, however, showed widespread softening and disintegration of the white matter of both hemispheres, the gray matter in general being spared. The process involved the corpus callosum and extended downward as far as the midbrain and the chiasma.

The outstanding feature of the less extensively involved areas of the spreading process was an abnormal filling of the blood vessels and a mucoid degeneration of the oligodendroglia, which proved to be identical with Penfield's acute swelling of the oligodendroglia. The areas of severe degeneration showed a complete destruction of the myelin, which was phagocytosed by scavenger cells of microglia origin. The axis cylinders were partially preserved and the neuroglia had hypertrophied in number and in size of the cells and formed a thick fibrous network. The perivascular spaces were enlarged and filled with scavenger cells and a few lymphocytes. In the regions of severest destruction there was degeneration also of the astrocytes into polynuclear forms without prolongations, dissolution of the rest of the nervous tissue, and formation of a scar of connective tissue. The engorgement of the vessels and the occasional perivascular hæmorrhages in this case suggest that the disease may be caused by an ultramicroscopic virus. R. M. S.

[198] **Centrolobar sclerosis of the brain** (La sclérose cérébrale centro-lobaire à tendence symétrique. Ses rapports avec l'encéphalite périaxiale diffuse).—C. FOIX and J. MARIE. *L'Encéphale*, 1927, xxii, 81.

THE authors describe in detail three cases in which monoplegia, diplegia or quadriplegia came on in an acute or subacute manner, remained at a maximum for one or two months and then receded somewhat, leaving the patient severely crippled. The ages of the patients at the onset of symptoms were 18 years, 4 years, and a few months respectively. Death occurred at the ages of 28, 14 and 65. In none of the three was there any tendency to increasing paralysis or to mental deterioration after the initial attack. In all, however, the lesion in the brain was identical with that in Schilder's encephalitis in macroscopic appearance and differed only in the absence of any evidence of inflammation or of recent demyelination. There was in fact a neuroglial scar which replaced a large area of the white matter of one or both cerebral hemispheres, but spared the cortex, the subcortical zone of fibres and the basal ganglia.

The authors review the literature of Schilder's encephalitis, and show that the case of Pierre Marie and Foix (which appears to be the same as the first of the cases described in this paper) is the only one in which the disease was not progressive, although in the case of Claude and Lhermitte there were phases of slight regression of symptoms in the intervals between the phases of progression. There appears therefore to be a form of encephalitis, similar in its effects to that described by Schilder, but differing from it in clinical course. The onset is rather more acute (although it could scarcely be more acute than the loss of vision in two of the published cases of Schilder's encephalitis); there is then a period of maximal paralysis lasting for several weeks or months followed by a period of slight regression of symptoms and permanent disablement. The extreme spasticity of the paralysed limbs is emphasized. In the third case here described a spastic club foot was the only sequel, but this remained unchanged for over 60 years. It is obviously impossible to distinguish the anatomical lesion from that of Schilder's encephalitis, since the only difference is one of age. It may be indeed that some of the more acute cases described as Schilder's encephalitis, if life had been prolonged, might have become stationary and would then have conformed to the type of Foix and Marie. The evidence given by this paper suggests in fact, if it does not prove, that Schilder's encephalitis, in some of its more acute forms, may be non-progressive and may in fact constitute the anatomical basis of some cases of spastic diplegia developing in infancy or childhood.

J. G. GREENFIELD.

[199] **Chorea of Huntington with endocarditis and polyarthritis.**—JAMES F. CLANCY. *Acta Psychiat. et Neurol.*, 1927, ii, 87.

A CASE is described of a female, age 69, with chorea of 10 years' standing. There was an indefinite family history of choreiform movements but none of mental disorder. At the age of five the patient developed endocarditis, and rheumatism at 17. One year before coming under observation she began to have fits without complete loss of consciousness. There was no severe dementia and orientation was good. Choreiform movements were severe and universal in distribution: speech was almost unintelligible. Two months before death she developed a subacute polyarthritis with fever. Autopsy revealed recent and old endocardial vegetations. Cross-section of the brain showed a marked shrinkage of the caudate nuclei on both sides, with some ventricular distension. The oral part of the caudate was the more affected; there was a numerical atrophy of cells and a glial increase. Some of the ganglion cells contained a yellowish pigment. Although the smaller cells of the neostriatum were most affected, it was thought that the larger ones were also altered. The globus pallidus was almost normal. In its anterior half the corpus callosum was small though well myelinated. There were very pronounced cortical changes, most marked in the frontal lobes and central gyri.

The polyarthritis and endocarditis are important factors which possibly played an exogenous role in the etiology by precipitating symptoms in an organism already weakened by hereditary influence. Connection between the chorea of Huntington and of Sydenham, a conception discussed in the older literature, is once more suggested.

M. C.

[200] **Cyst of third ventricle; complete destruction of infundibular region without any so-called hypophyseal signs** (Kyste du troisième ventricule, déstruction totale de la région infundibulaire sans signes dites hypophysaires).—L. FREY. *L'Encéphale*, 1927, xxii, 21.

THE case reported is an argument against the theory advanced by Camus and Roussy that the signs of so-called dyspituitarism are in reality due to destruction of the infundibulum and not of the pars posterior hypophyseos. A cystic tumour of the third ventricle had destroyed completely the infundibular region but had only compressed the pituitary body slightly. The only symptoms of dyspituitarism were a slight eunuchoid constitution and impotence. The case is interesting also in that the first symptom was trigeminal neuralgia. This persisted and proved to be due to extension of the tumour to the region of the Gasserian ganglion. Another symptom of interest was nystagmus on lateral deviation of the eyes, which was not due to any demonstrable lesion either in the brainstem or cerebellum.

J. G. GREENFIELD.

[201] **Segmental trophic œdema of cerebral origin.**—A. GORDON. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 381.

A CASE is described with a sudden onset of astereognosis of the left hand, probably due to the thrombosis of a cortical vessel in the parietal lobule. Later a localized œdema of the hand developed. Usual tests failed to disclose any signs of sympathetic involvement; medullary disease could also be eliminated, and the author thinks that the injury to the sensory cortex involved in the acute lesion was responsible for the production of the "main succulente."

R. G. G.

[202] **Syphilitic meningitis in infants and young children.**—J. W. AMESSE and W. W. BARBER. *Amer. Jour. of Syphilis*, 1927, xi, 544.

THE authors report four cases of meningitis in young children, in three of which the Wassermann reaction in the blood and spinal fluids was positive, but in the fourth negative. This last case was presumed syphilitic from the course of the illness and the absence of other etiological factors, and also from a suspicious history of parental nervous disease. The authors comment on the rarity of the condition, its clinical similarity to tuberculous meningitis and its prolonged course and unfavourable prognosis even with intensive antisyphilitic treatment.

J. G. GREENFIELD.

[203] **The pupils as an aid to the diagnosis in states of coma.**—W. C. MENNINGER. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 553.

THE state of the pupils examined in 225 cases of complete coma is described. No very definite results are found and the author does not think that the pupillary condition is any help in the diagnosis of coma due to alcohol, diabetes, uræmia or carbon monoxide. In cerebral trauma or tumour the pupil tends to be dilated on the side of the lesion, while in pontine hæmorrhage the pupils are contracted. In fracture of the skull the light reflex tends to be abolished.

R. G. G.

[204] **A case of combined sclerosis in pernicious anaemia with polyneuritis, presenting the symptoms of a senile paraplegia.**—H. DE JONG. *Acta Psychiat. et Neurol.*, 1927, ii, 105.

A WOMAN of 69 gradually became weak in her legs over a course of years. On one occasion she sustained an ictus which was followed by further weakness in the legs and paræsthesiæ in the hands. Neurological examination revealed some weakness of the right face and tongue and arms. Both legs were paretic but especially the right one. The tone was increased; the ankle jerks could not be obtained, although the knee jerks were present. Both plantar responses were of the extensor variety. Some atrophy was present in the leg muscles and the electrical reactions were those of degeneration. Her tongue was red and somewhat painful; blood pressure 216–90; blood count:—reds 2,100,000; hæmoglobin 45 per cent., colour-index 1·38, leucocytes 11,000. There was some sensory loss over the legs. The indirect Van den Bergh reaction was weakly positive.

Autopsy revealed an adeno-carcinoma of the stomach; the cerebral arteries were markedly sclerosed. Microscopic examination of the spinal cord showed a typical combined degeneration in the posterior and lateral column.

The original diagnosis was that of senile paraplegia, but was later changed to subacute combined degeneration. Although no pathological evidence is given, the author suggests that the muscular atrophy was the result of a peripheral neuritis. Perhaps the most interesting feature of this case is the association of a gastric carcinoma with the blood picture of a pernicious type of anæmia, and a combined sclerosis of the cord. Although this association, which has been reported on other occasions, raises etiological points of supreme importance, the author does not discuss this aspect of the question. M. C.

PROGNOSIS AND TREATMENT.

[205] **Intravenous treatment of some epileptics with calcium chloride and gluco-calcium.**—E. KLEIN and E. FORCIONE. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 372.

ENTIRELY negative results are described by the authors who found that neither was the number or severity of the fits reduced nor was there any sustained increase in the calcium concentration of the blood by the use of either substance.

R. G. G.