bundle. The author thinks that this constitutes a direct connection between pallidum and tegmen. He also claims to have established the existence of a subthalamico-peduncular system of fibres.

R. G. G.


Continuing his researches on the substantia gelatinosa Rolandi through the animal series, the author summarises the results of his investigations in favour of the view expressed by Ranson, according to which protopathic sensation is associated with the substantia gelatinosa, while epicritic sensation chiefly passes upwards in the cord, by way of the posterior funiculi. In lower animals where protopathic sensation is practically the only kind present the posterior funiculi are small or absent; in mammals, these form no less than 39 per cent. of the total white substance of the cord. Ranson found unmyelinated fibres in the lateral part of the posterior root at the point where the latter enters the cord; they proceed into Lissauer's zone and end in the substantia gelatinosa. Similar unmyelinated fibres have been seen in the descending root of the fifth as well as in its peripheral branches, and they are considered by some to be conductors of protopathic sensibility.

S. A. K. W.

[81] The diencephalic vegetative mechanisms.—S. E. JELLIFFE. Arch. of Neurol. and Psychiat., 1929, xxi, 838.

The author presents a masterly review of the anatomy and physiology of the central vegetative mechanisms. The illustrations have been chosen with care and make it easy for the reader to follow the intricate fibre connections of the diencephalon.

R. M. S.

NEUROPATHOLOGY.


A review of the literature is given and the author's experiments are described, bearing on the healing of non-septic wounds of the brain and the influence thereon of certain internal secretions. The neuroglia of the grey matter forms a barrier separating wounded from healthy tissue. That of the white matter shows amœboid transformation and hypertrophy typical of protoplasmic behaviour. The neuroglia does not show proliferation or phagocytosis and
granules may be seen to traverse but not to be formed in its substance. If a lacuna is formed by the wound the glial cells do not fill it up. In animals whose thyroid is removed there is rather more tendency to protoplasmic hypertrophy and ameboid degeneration of the glial cells. Cicatriziation and the filling of the cavity form the role of connective tissue derived from the pia if the wound is near the surface; if deeper, a current of fibroblasts may be seen spreading from the margin of the lesion. At the same time a proliferation of vessels is observed which gradually invades the blood clot. This network encloses the necrosed tissue and phagocytosis and absorption take place. The main point of the author's technique is that he is able to distinguish between the neuroglia which proliferates and the microglia which does not. He thinks that reticulate elements of microglial origin have no phagocytic function, but probably represent a regressive form of the hypertrophied microglial elements in which the eventual inclusion of fat particles may lead to fatty infiltration. He concludes that phagocytosis and absorption are the function of mesodermic lymphatic cells of the reticulo-endothelial system.

R. G. G.

[83] Contribution to the study of heterotopia of the cerebral grey matter
(Contributo casistico allo studio della eterotopia della sostanza cerebrale).—L. Ajello. Riv. di pat. nerv. e ment., 1928, xxxiii, 1.

Full pathological details are given of one case but the clinical history is not available. The author reviews the literature, and points out that while idiocy and epilepsy commonly accompany the condition it is compatible with perfect mental normality.

R. G. G.

[84] The question of cerebral air-embolism in cases of attempted abortion

Among the methods of criminal abortion, the injection of soap and water into the uterus under pressure is one of the more common; the injection of air at the same time is practically unavoidable. In consequence of this manœuvre serious symptoms and even death not infrequently occur. In some cases the symptoms are chiefly circulatory, in others they are cerebral and take the form of prolonged unconsciousness and epileptiform convulsions.

Air-embolism has been regarded as the cause of the cerebral symptoms in such cases and in the only case fully reported with pathological findings (Neubürger) the histological changes were analogous to those in a case of fat-embolism. The possibility of cerebral arterial air-embolism has clinical and experimental confirmation. Symptoms will only arise when the capillary circulation offers a more or less definite obstruction. In one of the recorded post-abortive cases both pulmonary and cerebral symptoms obtained.
When it is a matter of venous air-embolism the explanation is more difficult; with injection of air into the great veins of the body death results from partial filling of the right side of the heart with air, and the severity of the circulatory symptoms is dependent upon the amount of air injected. If cerebral air-embolism results from entrance of air into the venous system the right heart must be able to overcome the amount of air and the air must pass either through the capillaries of the lungs or through a patent foramen ovale. In experiments on animals it has been found that if air had passed through the pulmonary circulation some was always present in the coronary arteries, and moreover, the lungs and the central nervous system (especially the medulla), suffered damage from the presence of air-containing blood in their capillaries.

It is possible that in cases of abortion the soap may give rise to a corrosive encephalitis.

V. Hoesslin gives a clinical account of a case in which this method of abortion gave rise to severe nervous symptoms—unconsciousness for four days, epileptiform convulsions affecting the left side, and, later, paresis of the right side. The patient made a complete recovery in four weeks. He regards air-embolism—possibly by way of a patent foramen ovale—as the most likely explanation of the symptoms.

J. P. M.

[85] On the tumours of the ependyma and of the choroid plexus (Sui tumori dell’ependima e dei plessi coroidei).—F. Battaglia. Riv. di pat. nerv. e ment., 1928, xxxiii, 434.

A discussion of the nature of certain tumours. The author concludes that tumours which arise from the choroid plexus retain the appearance of ventricular papillomata. Such tumours have hitherto been confused owing to an imperfect knowledge of the morphological differences normally existing between the two epithelia of the plexus and the ependyma. Nowadays it is possible to distinguish them on cytological criteria. Such criteria recently used by some authors to differentiate fibro-epithelial tumours of a malignant nature arising in the central nervous system seem of doubtful value. It is possible that those tumours which have no connexion with the ventricular cavity have their origin from a relic of ependyma. Other tumours are ascribed to the embryonic tissue of the ependyma and may be justly referred to as spongioblastomata. In a case of the sort here described there may be noticed a double evolutionary orientation, (1) the production of glia cells and (2) of cells having the appearance of epithelium without prolongations. This may represent the attempt of the spongioblasts to develop towards the form of ependymal cells, and may throw light on the neuroepithelial formations met with in certain gliomata. Other epithelial developments which have been described in gliomata may be regarded as true detached ependymal tissue.

R. G. G.
[86] **Dural herniations of the pia-arachnoid.**—**GERALD H. J. PEARSON.**


At the autopsy of a man dying at the age of 55 from progressive bulbar palsy numerous small dural herniations were noted over the right frontal pole and both parietal eminences. The author puts forward the following suggestion as to their mode of origin: there was first a proliferation and fibrosis of the arachnoid over the whole cortex, which interfered with the absorption of cerebrospinal fluid. The fluid then collected in the pial meshes and by pressure-irritation caused further fibrosis and thickening of the membranes. Weak points in the upper layer of the pia and arachnoid gave way and the underlying layers herniated through them to form papillomatous projections. Where these projections were subjacent to a weak spot in the dura or where their size produced a dural atrophy, the herniation extended through this membrane.

M. C.

[87] **The pathogenesis of osteoarthropathy in paralysed limbs consecutive to trauma of the spinal cord** (La patogenesi delle osteo-artropatie e delle para-osteo-artropatie negli arti paralizzati in seguito a traumi del midollo spinale).—**E. REPETTO.** *Il Cervello*, 1929, viii, 53.

A number of different theories have been advanced to account for the development of arthropathic change in limbs paralysed after spinal trauma.

The author has conducted a series of experiments in rabbits, sectioning the spinal cord and endeavouring to produce arthritic lesions by various procedures. He examines in turn the theories of abnormal trophicity, pathological innervation, infection, toxicity, arteritis of joint vessels, pathological calcification, and vasomotor or sympathtic disorder, and dismisses them all as a result of his experiments. His conclusion is that intra-articular trauma of periosteum leads to post-paralytic osteo-arthropathy, and trauma alone. In his experimental animals it was produced by the galvano-cautery.

S. A. K. W.

[88] 1. **Nitrogenous metabolism in postencephalitic rigidity.**

2. **Creatine and rigidity.**—**MARTIN HIRST and A. G. IMRIE.** *Quart. Jour. of Med.*, 1927, xxi, 139; 1928, xxi, 401.

In their first paper the authors found that a mild degree of rigidity did not influence the output of uric acid but that in a severe case the output was high. If the rigidity was reduced by hyosine the uric acid was correspondingly diminished. In all cases creatine was excreted. In their later communication creatinuria was found to occur constantly in the marked degrees of Parkinsonian rigidity. They suggest that there is an association between the activity of the muscles in this pathological state and the amount of uric acid and creatine excreted.

E. A. C.

The necessity for an early quantitative examination of the sugar content in the cerebrospinal fluid is stressed; and observations are recorded showing the influence of the length of time between withdrawal and estimation of sugar. A progressive decrease in the sugar content is demonstrated, which diminution is presumed to be due to a glycolytic ferment present in the fluid. It would have been of interest to have had the cellular content of the fluids, as it is possible that the ferment may be the result of the presence of the cells and that it is not an inherent part of the fluid itself. Attention is also drawn to the fact that ventricular fluid contains more sugar than either the cisternal or lumbar fluid. The paper is one of interest and raises many problems regarding the chemistry of the cerebrospinal fluid.

E. A. C.


This reaction is carried out as follows. Three reagents are required: carbonate of soda solution 10 per cent., sublimate solution .5 per cent., and fuchsin (Grubler, non acid) .02 per cent. To one c.c. of spinal fluid is first added a drop of the sodium solution, then with a graduated pipette .3 c.c. of a mixture in equal parts of the sublimate and fuchsin solutions. The reaction is simple and purely chemical, and permits a differentiation with a single dilution between inflammatory conditions of the central nervous system with increase of the total albumen, and degenerative processes with increase of the globulin only.

It cannot be used as a substitute for the Wassermann reaction but it is sensitive to though not specific for neurosyphilitic lesions, especially parasyphilis. In general paralysis treated by malaria and arsenical preparations this reaction, like the Wassermann reaction, may become negative.

R. G. G.

Some observations on the leucocyte count in epilepsy.—H. A. Patterson and S. M. Weingrow. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 412.

The following conclusions were based on a study of 182 epileptics. The total white cell count in epilepsy is widely variable: 52 per cent. of the cases showed leucocytosis: 25.5 per cent. gave normal counts, and 20.3 per cent. showed leukopenia. The inconstancy of the blood picture in epilepsy, together with the relatively few cases on which frequently the conclusions previously reported are based, perhaps accounts for the conflict of opinions in the literature. The leucocytosis observed by the authors was not neutrophilic but lymphocytic.
Definite evidence of epileptic leucocytosis being influenced by digestion of food was lacking. Within the limits of physiological variation, the rise in the leucocyte count is the same in non-idiopathic as in idiopathic epilepsy.

A remote effect is not produced by either grand or petit mal seizures on the white cell count in both idiopathic and non-idiopathic epilepsy. It seems that the leucocytosis frequently encountered in epilepsy is not due to the disease per se, but to some concomitant phenomenon. A connection between the leucocyte count in epilepsy and the presence or absence of an aura apparently does not exist. Observations failed to show any relation between onset and duration and the white cell count in epilepsy. The leucocyte count in epilepsy does not appear to be affected by the secondary anaemia which often accompanies this condition.

R. M. S.

SENSORIMOTOR NEUROLOGY.


This paper is an important contribution to our knowledge of the pathogenesis of transient cerebral attacks and also of cerebral softening in that it illustrates the trend of modern thought as applied to an important problem in cerebrovascular disease. The text of Spielmeyer’s article is that pathologically demonstrable areas of ischaemia can exist in the brain unassociated with adequate anatomical changes in the blood-vessels of that part. It is suggested that there is a functional disturbance of the blood-supply due to a vasoconstriction of the arteries and capillaries lasting a longer or shorter time. The clinical expression takes the form of either transient or permanent focal manifestations. Spielmeyer points out that the ischemic zones may appear in stained preparations as a mere bleaching of the cortex; these may be overlooked on cursory examination and herein possibly lies the explanation of those cases of hemiplegia which are recorded as having no demonstrable pathological cause. It is interesting to note how the clinician—who has been long misled by the negative findings of the physiologist—is gradually returning to the conception of a vasoconstrictor process in the cerebral blood-vessels as a pathogenic agency.

M. C.


A useful review of the whole question of angiospasm in regard to the nervous system leads to the following generalisations.