

NEUROPATHOLOGY.

- [145] **Experimental studies on the problem of so-called spontaneous encephalitis of guineapigs** (Experimentelle Studien zum Problem der sogenannten Spontanencephalitis der Kaninchen).—H. PETTE. *Zeits. f. Hyg. u. Infektionskrankh.*, 1928, cviii, 700.

MATERIAL from the brains of 12 cases of general paralysis was injected into the testes of guineapigs. In five cases a slow chronic meningoencephalitis developed. The histological characters were of the usual type, and there was a pronounced tendency to granuloma-formation. Similar results were obtained in three cases where spinal fluid from general paralytics was injected cisternally.

In seven animals fresh luetic material was injected by the intratesticular route, with a similar positive result in four.

Eleven times was a guineapig infected cisternally with the spinal fluid from cases of acute epidemic encephalitis; in three animals a meningoencephalitis developed which was identical histologically with that already described.

An entirely similar encephalitis, clinically and pathologically, appeared however in a number of animals spontaneously.

Some twenty-seven guineapigs were used as controls, being injected with material of another kind, and all remained healthy, with the exception of one only, in which encephalitis developed, four months afterwards.

In all these cases of meningoencephalitis the process is the same, and is attributable to a microsporidium; the infection is general, though in the later stages the virus has an affinity for the central nervous system.

The conclusion is to the effect that the encephalitozoon cuniculi exists in the animal harmlessly until it is stimulated into pathogenic activity by the presence of infective material which itself has a special tendency to attack the nervous system. Immunobiological equilibrium is then broken down, and the encephalitis appears.

It is considered that the results of these experiments have an important bearing on problems of the activation of infective diseases.

S. A. K. W.

- [146] **Punctures of the brain.**—W. PENFIELD and R. C. BUCKLEY. *Arch. of Neurol. and Psychiat.*, 1928, xx, 1.

A HISTOLOGICAL study of punctures of the brain. In one series punctures were made by a sterile blunt brain needle, and, in the other, by a sterile open cannula of the same calibre.

When a blunt brain needle was used, all the injured tissue remained behind, and a closed track containing a connective tissue core firmly attached to the

overlying dura resulted. The track was surrounded by a moderate gliosis in the cortical grey matter, but gliosis did not occur, and there was even a decrease in astrocytes, in the white matter.

When stab wounds were made by an open cannula, and a cylinder of cerebral tissue was removed, a gaping track resulted which contained little if any connective tissue and which was slightly attached to the overlying dura. The track was surrounded by a gliosis in the cortical gray matter but there was none in the white matter. There was, in general, greater formation of adhesions, greater distortion and greater destruction of the brain about the tracks made by a closed needle than about the tracks made by an open needle of the same size.

As a general principle, it follows that if destroyed cerebral tissue is left in a wound in the brain, a cicatrix results containing connective tissue which contracts and causes distortion of the brain; a greater resultant gliosis also occurs. Superficial adhesions are reduced to a minimum if the damaged brain tissue is removed, and hence for exploratory brain punctures it is preferable to employ the cannula.

R. M. S.

[147] **Changes in the brain in pyæmia and in septicæmia.**—I. B. DIAMOND
Arch. of Neurol. and Psychiat., 1928, xx, 524.

THE author reports two cases of hæmatogenous infection of the central nervous system. In the first, a case of pyæmia, the changes were mainly inflammatory with marked infiltrative and hyperplastic phenomena in the pia-arachnoid, choroid plexus and glia; degenerative changes, though present, were rather insignificant. The changes were invariably associated with the presence of the staphylococcus pyogenes aureus, which evidently was responsible for the reactive phenomena in the mesodermal and glial tissues. The mesodermal reactions were represented by polymorphonuclear cells, proliferation of the endothelial and adventitial cells and new formation of the capillaries. The glial reaction was represented by granulomas and nodules.

The second case, which was one of septicæmia, differed from the other in the presence of signs of "healing" phenomena, in the form of numerous fibroblasts, masses of glia cells round the foci, mild signs of recent thrombosis in a few of the blood vessels and the better preservation of the parenchyma. The reactive phenomena were in the form of multiple foci of softening, and were the result of invasion of the parenchyma, not by micro-organisms, but by their toxins. For this condition Hassin and Bassoe have proposed the term multiple degenerative softening.

R. M. S.

- [148] **Cerebral gliomata.**—E. ARNOLD CARMICHAEL. *Jour. of Pathol. and Bacteriol.*, 1928, xxxi, 493.

SEVENTY-FIVE tumours were examined histologically; of these, 62 were classified as gliomata. The classification suggested by Bailey and Cushing was found applicable to the present material.

The author offers a simpler classification, into two large groups, viz.:

- (a) Spongioblastic tumours.
- (b) Astroblastic tumours.

These two groups show definite pathological and clinical differences. The former is made up of an embryonic type of cell staining poorly with gold solutions; it causes a reactionary gliosis and is highly vascular at its edge; it presents a microscopical picture of rapid growth and is prone to degeneration; clinically it has a short history; it occurs commonly in persons over 40 years of age; it usually causes focal signs before signs of general intracranial pressure.

The other class contains tumours made up of a more adult type of cell, staining well with gold; it causes little if any reactionary gliosis; it is much less vascular; it is less prone to degeneration and has a longer clinical history; it occurs in persons under 40, and usually causes pressure symptoms before focal signs.

J. V.

- [149] **The occurrence of brain-tissue within the nose; so-called nasal glioma**—D. GUTHRIE and N. DOTT. *Jour. of Laryng. and Otol.*, 1927, 733.

THE occurrence of brain tissue in the nasal cavity as a development from an embryonic rest of the central nervous system is sufficiently common in young children to constitute a definite clinical entity. Apart from this encephalocele, intranasal tumours containing glial tissue are extremely rare. A case is recorded by the authors of true nasal glioma. It illustrates the penetration of the nasal cavity by a cerebral frontal glioma of the spongioblastic type. The mode of penetration is thought to have been a neoplastic invasion of a minute pressure hernia of the cortex.

J. V.

- [150] **The pathogenesis of disseminated sclerosis** (Ueber die Pathogenese der multiplen Sklerose).—H. PETTE. *Deut. Zeits. f. Nervenheilk.*, 1928, cv, 76.

THIS paper is concerned with the connexions between disseminated sclerosis and disseminated encephalomyelitis. Three cases were studied clinically and pathologically; the duration of one case was about two weeks, the second about five weeks, and the third about seven years. The type of the first two was that of acute ascending paralysis with involvement of the lower cranial nerves.

No distinct pathological separation of disseminated sclerosis from the other can be made. Any differences are those of degree and not of kind. Complex factors of an etiological and constitutional kind are responsible for the variations in the course of the two.

The elective affinity of the morbid agent for a particular tissue, viz., one of ectodermal origin, characterises the histological picture. The parenchyma is the first to suffer, especially the myelin sheaths, while vessels and meninges (mesodermal) exhibit secondary reaction. The type of tissue reaction suggests that the agent is a living virus.

Disseminated sclerosis offers close resemblances to certain kinds of encephalomyelitis which in recent years have been seen to follow infective conditions such as mumps, chickenpox and vaccinia. Yet the actual relationship has not been established.

S. A. K. W.

- [151] **Acrodynia : A study of the pathology.**—J. W. KERNOHAM and R. L. J. KENNEDY. *Amer. Jour. Dis. Child.*, 1928, xxxvi, 341.

THE authors record one case of erythrœdema-polyneuritis, that of a boy, age 23 months, on whom an autopsy was obtained. The child, on admission to hospital, showed a generalised papular eruption, stomatitis, gingivitis and loosening of the teeth. Photophobia and restlessness were very marked. The gait was ataxic, and there was lack of reaction to pinprick. The condition of the reflexes is not recorded. Death occurred on the eleventh day.

They quote the various necropsy reports in the literature. Warthin's two cases showed generalised œdema and signs of slight meningeal irritation. He felt that the condition might be due to a food deficiency, and suggested that it might be an infantile form of pellagra.

Byfield considers it to be primarily a disease of the nervous system, and is supported in his contention by Paterson and Greenfield, who called it a polyneuritis affecting particularly the peripheral parts of the nerve trunks. There was marked myelin destruction in some fibres of the peripheral nerves and some of the anterior horn cells showed chromatolytic changes in their two cases. They considered the condition to be similar to but distinct from Bashford's "acute infective polyneuritis."

In the authors' case the findings of importance were also in the nervous system. They found fragmentation of varying degree in the myelin sheaths of the peripheral nerves, more marked in those from the lumbar than the brachial plexuses. There was also chromatolysis in the dorsal ganglia cells of the lumbar and thoracic roots, in some sacral anterior horn cells, and in the cells of the lateral horns and Clarke's column in the thoracic region. Chromatolytic changes were also found in the cells of the cineritia teres, the mesencephalic root of the fifth nerve and the locus cœruleus. Both Gasserian ganglia showed extensive chromatolysis and similar changes were seen in some cells

of the lenticular nucleus and of the anterior nucleus of the thalamus. The changes appeared to be an axonal reaction rather than a primary chromatolysis. There was no evidence of infection in the nervous system.

They found no changes in the cerebral or cerebellar cortex and no increase of glia cells throughout the cord.

In conclusion they state that the association of disease of the upper respiratory tract with the degenerative changes found in the nervous system in this condition, favour the view that it is probably due to an infection, the toxins of which affect the nervous tissues.

P. W.

SENSORIMOTOR NEUROLOGY.

- [152] **New ideas as to the genesis of transient hemiplegia and cerebral softening**
(Les idées nouvelles sur la g n se de l'h mipl gie transitoire et du ramollissement c r bral).—J. LHERMITTE. *L'Enc phale*, 1928, xxiii, 27.

AN interesting exposition of the present status of the problem of evanescent cerebral attacks associated with old age and with arteriosclerosis. The author first discusses the vexed question of a possible nervous control over the cerebral arterioles, and quotes at length Florey's work on the capillary reactions towards various types of stimulus. Passing to the clinical aspect of the subject, Lhermitte throws doubt upon the conception of vascular spasm in the production of transient hemiplegias; he inclines towards a hypothesis which has long been familiar to English medicine and which has lately received a fillip from the work of Naffziger, viz., the theory of local isch mia from an attack of relative hypotension. Thus a state of transient hemiplegia is regarded as the result of two factors; first, the presence of diseased and partially occluded blood-vessels, and secondly, an incidental and temporary lowering of blood-pressure with a local relative retardation of the blood-stream. The importance of this view-point in determining the correct therapy is of course obvious, and stimulating measures (adrenalin, digitalis, saline injections) are advocated. Lhermitte extends this conception of relative hypotension so as to embrace the pathogenesis of encephalomalacia. He quotes the work of Foix and Ley, who demonstrated that—contrary to the previously accepted view—complete vascular occlusion is the exception in cases of cerebral softening.

These views will not prove novel to English neurologists. Although almost certainly the correct explanation of many instances, this hypothesis must not, however, be strained so as to include every case of transient ictus. It seems more probable that evanescent attacks are of varying origins, and that the morbid physiology differs according to the nature of the arterial changes, the presence or absence of hypertension, the age of the patient and other allied factors.

M. C.

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