of the fissure of Sylvius. They are described with the greatest minuteness and form an indispensable preliminary to study of the same region in pathological cases. The paper is fully illustrated with drawings and photographs.

S. A. K. W.

[123] Observations on unilateral 'thalamus cats' and 'striatum cats' (Beobachtungen an halbseitigen Thalamuskätzchen und Striatumkätzchen sowie nach halbseitiger Exstirpation des Frontal oder Occipitalpoles).

A 'striatum cat' is one from which the cortex has been removed with preservation of the corpus striatum; a 'thalamus cat' has its thalamus as well.

Immediately after unilateral removal of the cortex the animal moves in a circle towards the opposite side, but, within two or three days, to the same side. Thereafter it moves as at the beginning only when its eyes are covered. When only the frontal pole is cut out this movement in a circle to the opposite side is very pronounced if the eyes are closed.

If the occipital pole is extirpated progression is mainly to the same side as the lesion. Unilateral removal of the cortex is followed by a spastic paralysis with great extensor rigidity, but after a time only a paresis remains. On removal of the frontal pole alone the same results follow as those after complete cortical resection—so far as concerns tonus and movement.

Some of the unilateral thalamus and striatum cats exhibited for a few days after operation a tremor of the opposite limbs at the commencement of movement. Hyperkinesis succeeds operations removing the frontal cortex, also those producing the thalamus and striatum cat respectively. It is not seen after operations confined to the occipital cortex. The extensor tonus of unilateral thalamus cats appears to be greater than that of unilateral striatum cats.

S. A. K. W.

NEUROPATHOLOGY.


In cerebral apoplexy it is necessary to distinguish between the hæmorrhage from a large vessel and hæmorrhages from the small vessels in the adjacent area. (1) The large hæmorrhage is caused by the rupture of a vessel and is dependent on an atheromatous ulcer of local nature which bursts, according to the conception of Rühl, with outflowing blood forcing its way into the tissue and causing mechanical injury. (2) Small vessels in the adjacent areas with altered walls and also small vessels without alteration may thus be caused to rupture. (3) In addition there is ischæmia in some parts of the environment with consequent necrosis of the vessel walls and of the brain tissue; moreover,
in consequence of the chemical influences of the extravasated blood in the tissues vessels that are secondarily altered also rupture.

The study of gunshot wounds discloses similar alterations analogous to those in apoplexia cerebri, dependent on the same principles. The tissue bordering on the gunshot wounds shows necrobiotic processes of the walls of the small vessels and blood in the perivascular spaces. Thus acute lesions are shown to have vascular wall degenerations.

It is not uncommon to make the clinical diagnosis of a cerebral hemorrhage and then to have the pathologist find no gross lesion at autopsy. Such cases used to be spoken of as 'hemiplegia without anatomical confirmation,' but Spielmeyer's observations have demonstrated that although in such cases there is no obvious lesion, careful histological examination will show widespread and characteristic ischaemic lesions. By impeding the cerebral circulation for only six to eight minutes Gildea and Cobb have been able to produce similar lesions in the brains of cats, and it is thus possible to explain on a basis of transitory anæmia the various evanescent hemiplegias, monoparesis and aphasias that are so commonly met with in clinical practice; total recovery may take place in one case, and in another that appears no worse at the beginning the neurological signs may become permanent disabilities. In the first case there is evidently only a passing cerebral anæmia, not prolonged enough to cause tissue destruction, and in the second there is actual tissue destruction in the cerebrum. How long certain brain cells can stand a given grade of anæmia is still unknown; much more research must be made and new methods must be devised before these problems are solved.

Spielmeyer's pathological studies, however, indicate that many vascular disturbances, in young patients especially, may be due to vasomotor spasm in the brain, and the physiological experiments of Forbes and his co-workers prove that the cerebral blood supply may be influenced by vasomotor control of the intracranial arteries and veins.

So the problem of cerebral hemorrhage is still new and challenging to investigators.

R. M. S.


The author describes some acute cases of cerebral disease in which multiple lesions of vascular origin, electively localised in the cortical gray matter, formed the most interesting part of, or even the entire, pathological process. In two cases toxæmia of pregnancy was the probable cause; in one case encephalitis, and, in another, severe shock with fall of blood pressure. In the first case, which showed small foci of necrosis or necrobiosis distributed apparently without rule over the various layers of the cortex, spasm of the cortical
arteries was the probable cause. In the other cases the lesions took the shape known as laminated cortical softening; the latter may destroy the cortex in its entire depth or (often in other parts of the same hemisphere) may be restricted to one or two cell-layers. The third cortical lamina is most frequently the seat of this lesion, but it can also be found in the fifth and sixth layers together. In addition to these peculiar lesions others of the common vascular or thrombotic type were present, mostly in connection with widespread diffuse degeneration of ganglion cells.

From a study of the literature it is evident that the direct cause of spasm of vessels which gives rise to cortical ischaemic necrosis of the laminated type is not yet known. Probably functional disturbances of the cerebral circulation play a much greater role in the pathogenesis of foci of softening in general arteriosclerosis than has hitherto been believed.

R. M. S.


The cells of Schwann, whether in normal nerve or in the area of proliferative activity in the scar of a regenerating nerve, never assume vital coloration however long it is applied. They proliferate much more intensively in the peripheral than in the central end of a divided nerve, but, in order of time, secondarily to connective-tissue elements. The cells of the epi-, peri-, and endoneural tissue and of the interfascial connective-tissue proliferate early, forming a fibrous scar which in its disposition effects an arrangement of nerve-bundles differently from the normal nerve. These connective-tissue elements can at once be distinguished by the fact that they take on vital coloration intensively.

R. G. G.


This reaction in 150 cases has shown in addition to the constancy of the results a sensibility not so far reached by other reactions utilised to demonstrate an increase or alteration in the protein content of the spinal fluid. It will reveal an increase in the protein in diseases in which this is so slight as usually to escape notice, but notably in dementia præcox, disseminated sclerosis, epilepsy, etc. A fluid should not be regarded as completely normal unless the potassium bichromate reaction is negative as well as the others.

R. G. G.
ABSTRACTS

[128] One the value of a precipitation reaction to formaline in the cerebrospinal fluid in syphilitic nervous disease (Sul valore di una reazione di flocculazione alla formalina nei liquidi cerebrospinali di luetici nervosi).—L. GIOVANNI. Riv. di pat. nerv. e ment., 1930, xxxv, 231.

The author reports 72 cases in which this reaction gave positive results. The nature of the precipitated particles is not determined. R. G. G.

[129] On inorganic phosphorus in the blood in epileptic subjects (Sul fosforo inorganico del sangue nei soggetti epilettici).—E. MONDIO. Riv. di pat. nerv. e ment., 1929, xxxiv, 524.

The normal phosphorus content in the blood is 2.3 mg. per cent. for men and 3.5 per cent. for women. The excess in women may be due to ovarian function which seems to diminish the elimination of phosphorus by the urine. Epileptics of either sex as a whole show no noteworthy disturbance of phosphorus content of the blood, but those with few fits have a low content while those with frequent fits have a high content. This may have some relation to muscular metabolism.

R. G. G.


The difficult question of regeneration in the neuraxis (as opposed to the peripheral system) is here examined in an interesting way, with numerous evidential data supporting the general argument, viz. that such processes do in point of fact exist. Of no less importance is the author's contention, which the philosophically-minded neurologist must be gratified to find so definitely expressed, that with the technical means at present disposal what we know of fine neural structure provides an altogether unsatisfying basis for elucidation of the course of nervous function. Over-valuation of the possibilities of morphological knowledge has led to disappointment and disillusion, with the result that the method is falling into disrepute. The author thinks unnecessarily so. Morphology is not dead if it seeks in form an expression of function. All living processes must be accompanied by changes in their anatomical substratum, and it is these the morphology of the future has to discover.

S. A. K. W.


The brain was that of a woman of 27, completely deaf from the age of four. Macroscopically, the left Sylvian fissure was longer than the right; the planum temporale of the left was very small, and almost completely absent.
on the right; the upper temporal surface was longer and narrower than the other; on both sides, Heschl’s gyrus was remarkably short and small, and the sulcus intermedius awanting.

From the standpoint of cytoarchitectonic structure a number of minute details are furnished for which the original should be consulted; they include reduction in number and size of cells, especially of the large pyramidal cells of the third layer, and disappearance of the vertical striation of the cortex in this region (‘organ-pipe formation’).

S. A. K. W.

[132] Zones of grape-like disintegration in the brains of rabbits (Le zolle di disintegrazione a grappolo nell’ encefe di conigli).—V. M. BUSCAINO. Riv. di pat. nerv. e ment., 1929, xxxiv, 382.

A series of experiments which seem to show that the particular lesions were not accidental, and in a number of cases at any rate were associated with serious intestinal lesions and liver degeneration. This would appear to offer an analogy to the pathology of Wilson’s disease.

R. G. G.

[133] Pathological changes in paralysis caused by drinking Jamaica ginger.—A. R. VONDERAHE. Arch. of Neurol. and Psychiat., 1931, xxv, 29.

Four cases of poisoning with commercial extract of Jamaica ginger are reported. The pathological changes revealed degeneration in the radial and anterior nerves, degenerative changes in the anterior roots, marked pathological changes in the anterior horn cells, characterised by swelling, chromatolysis, eccentric nuclei and death of the cells, and occasionally by their shrinkage. Central chromatolysis was the predominating type of cell change. All sections showed infiltration with amyloid bodies. The sensory nuclei were only slightly affected. Severe cellular changes, similar to those found in the anterior horn cells, were noted in the hypoglossal nucleus, dorsal motor nucleus of the vagus and nucleus ambiguus. There were no signs of inflammation, no phagocytic glia cells, lymphocytes, polymorphonuclear leucocytes or endothelial cells.

In the author’s opinion these observations suggest a toxin which reaches the peripheral nerves in the extremities through the circulation, but which subsequently travels into the central nervous system along the nerve fibres and anterior roots. In severe cases the toxin may then make its way to the medulla oblongata, where involvement of the vagal nuclei produces death by bulbar paralysis. Although the toxin exerts its pathological effects most severely on motor cells and fibres, none of the changes can be regarded as characteristic of any one toxic agent.

R. M. S.
[134] **Cholesteatoma of the spinal cord with some considerations on the cholesteatomata of the cerebrospinal axis** (Su di un caso di colesteatoma del midollo spinale, con alcune considerazioni sui colesteatomi dell'asse cerebrospinale).—P. Pitotti. *Riv. di pat. nerv. e ment.*, 1930, xxxv, 36.

The case observed by the author is the sixteenth in the literature. The tumour is particularly noteworthy as containing besides the epidermic form of epithelium, hairs, sebaceous and sweat glands and the covering of adipose tissue which brings it nearer to the structure of the skin, all of whose parts it reproduces. The author discusses the origin of these tumours and how they come to be included in the cerebrospinal axis.

He thinks that the malformations (spina bifida, etc.) which frequently accompany them are independent, but that both are caused by a common failure of development.

R. G. G.


The study of six cases has shown in addition to the usual findings of the disease special foci of rarefaction in the lateral columns at the level of the cervical segments, analogous to those found in the posterior columns in pernicious anaemia. If the nature of these foci is the same in both cases, it is suggestive of a toxic origin for amyotrophic lateral sclerosis. The different location of the lesions in the two syndromes may be associated with a different toxin which has a biochemical affinity for a different system of fibres.

R. G. G.

**SENSORIMOTOR NEUROLOGY.**


Two cases, clinically, seem quite different; however, both show the same essential features: epilepsy and the symptomatological pattern of neurosomatic deterioration. In the first case—that of a child—deterioration developed more rapidly and was more destructive, in nine years leading to the ultimate stage of neurosomatic deterioration, viz. profound dementia and cerebral flexion paraplegia. In the second case—that of a man 66 years of age, with late epilepsy—the neurological picture characteristic of neurosomatic deterioration developed more slowly, was not so dramatic, and the symptomatological pattern was one that could be identified with arteriosclerotic Parkinsonism.