extensor reflex. Evidently posterior root section removes proprioceptive inhibitors which normally are activated by sudden contraction.

Rotation of the head (chin towards the recording muscle) has the same effect on the crossed extensor reflex, with the exception that it diminishes markedly the rate of relaxation of that reflex.

This same head rotation reverses the reflex effect produced by a single break-shock applied to the ipsilateral sciatic nerve. Similar reversals occur in the response of the knee flexors.

From these experiments it seems clear that neck and labyrinthine proprioceptors influence spinal centres. Some impediment which hinders the passage of impulses across the cord is removed by rotation of the head. Presumably this impediment is due to some form of continuous inhibitory stimulation. The same release which facilitates the passage of impulses from one side of the cord to the other also converts the ipsilateral extensor response from one predominantly inhibitory to one predominantly excitatory. This reflex reversal seems to be brought about through some activation of some higher centre of the nervous system than the spinal.

J. V.

NEUROPATHOLOGY.


In this excellent review of a complicated subject the author points out the confusion which has arisen from cross classifications in the past. He proposes that we should divide the groups into (1) antenatal, (2) natal and (3) postnatal.

Leaving out of account familial conditions such as amaurotic family idiocy and hereditary family ataxy, many cases of paraplegia seem to show a family incidence. It is necessary, however, to be certain that such cases are true abiotrophies and not coincidental lesions at or after birth. At the same time there is evidence that hereditary factors play a part in determining other lesions later in the development of the infant.

Specific intoxications (lead, etc.), chronic alcoholism, and syphilis in the parents are important factors especially if they occur in both. The author, unlike some other observers, finds definite signs of syphilitic heredity in about 15 per cent. of cases only.

The post-natal infections are confused and comprise many conditions whose etiological nature is obscure. The author thinks that it is only rarely that it is possible to distinguish between primary or secondary lesions either clinically or by pathological examination.

Among the more clearly defined clinical syndromes the author includes (1) infantile cerebral hemiplegia. He points out that if this comes on after the age of eight it is in all respects similar to the hemiplegia of adults. In younger
ABSTRACTS

children it is often accompanied by Jacksonian fits and contractures may be associated with flaccidity. The tendon reflexes may be less altered than might be expected, but synkinesias are frequent. The limb may be small not so much from atrophy as from want of growth. Varieties of this condition may be met with, ranging from a condition presenting athetosis without contracture or atrophy, without modification of reflexes and without grave intellectual defect, produced by a lesion chiefly subcortical, to a condition with contractures, exaggeration of reflexes, epilepsy, and serious intellectual disturbances but without athetosis, representing a lesion chiefly cortical in extent.

2. Cerebral diplegia (Little’s disease). This is a somewhat confused group of all degrees of severity both intellectual and motor, with or without fits or involuntary movements. The lesions may be defective development, sclerosis, or more or less serious haemorrhage. The author considers that cases of lower limb paralysis with completely normal intelligence and upper limbs should be included in this group. He does not favour the separation of a group of double hemiplegias (in which the upper limbs are more seriously affected) from the general category of cerebral diplegias.

3. Pseudobulbar cases affecting the bulbar nuclei and often accompanied by extrapyramidal motor lesions.

Among extrapyramidal conditions the author distinguishes clinically between those reputedly due to lesions in the pallidum (archistriatum) producing rigidity, and those due to lesions of the neostriatum producing involuntary movements, athetosis and chorea. However he points out that pathologically such a distinction cannot be upheld. The clinical types discussed are:

1. The ‘double athetosis’ of Vogt, with generalized athetosis and rigidity of the limbs but without constant changes in the reflexes. This condition which is congenital tends to improve, especially after the first year. The lesion is in the putamen and caudate nucleus.

2. ‘Double athetosis’ with spastic diplegia. This may arise after birth and the pyramidal system is obviously involved. The lower limbs are contracted and walking is impossible. The site of the lesion is inconstant but involves the pallidum.

3. Congenital chorea. In these cases, which are rare, choreic movements occur from birth but the nature of the lesion is very uncertain.

4. Wilson’s disease. Hepatico-striate degeneration characterized by rhythmic tremors, interference with swallowing and speech, rigidity of muscles and later contraction and mental deterioration. It is a progressive and fatal disease. The lesion consists of a bilateral degeneration of the lenticular nuclei and cirrhosis of the liver. The pseudosclerosis of Westphal-Strümpell is so similar that it may be included with Wilson’s disease though some claim that a pigmentation in the former condition distinguishes the two. The condition is familial and probably an abiotrophy.
5. Torsion spasm. This may occur alone or as part of another syndrome and consists of a dystonia of the muscles of posture.

6. An atonic form in which atonia of certain muscles accompanies a cerebral lesion.

Various formes frustes are found which may be due to arrest of development, especially when we consider the hypertonus and inco-ordinated movements of the newborn infant.

R. G. G.

[156] Contribution to the pathological anatomy of chronic chorea (Contributo all’anatomia patologica delle coree croniche).—C. Bellavitis. Riv. di pat. nerv e ment., 1928, xxxiii, 407.

After reviewing the literature the author describes three cases of chronic chorea—one of Huntington’s chorea, one of doubtful origin, and one following an acute attack. While he found the main lesions in thalamus and corpus striatum, others also occurred in the cortex, especially in the fronto-rolandic area. In all the cases the liver was the seat of pathological change of one kind or another. He concludes that Huntington’s chorea and chronic chorea following an acute attack seem to be different diseases both anatomically and clinically; the first is a disease of the nervous system with few signs of inflammatory reaction and having affinities to systemic nervous disease; the second shows lesions in all parts of the system and is more easily explained as resulting from an inflammatory process.

Efforts are still made to maintain the hypothesis of a purely striate lesion to account for all forms of chorea, including the hereditary variety; but it seems necessary to suggest a less restricted theory, one which includes cortex, cerebellum and thalamus among the sites of lesions. In Huntington’s chorea there is a complex of lesions in the striate and fronto-rolandic areas.

R. G. G.


Seven cases are described of changes in the brain in severe infections and toxæmias. These conditions include typhoid fever, acute rheumatic fever, toxæmia of pregnancy, erysipelas, Hodgkin’s disease, chronic tuberculosis and an undetermined toxic condition. The clinical picture consisted of delirium, at times of a most severe degree, with increase of psychomotor activities such as restlessness, muscle twitching and even convulsions; hyperæsthesia, visual and auditory hallucinations and meningeal irritative signs were at times present.
Pathologically, the predominant change occurred in the small cortical vessels with resultant secondary manifestations in the brain substance. The lining cells of the small vessels showed swelling and proliferation, with formation of new vessels. Scattered throughout the cortex were small microscopic areas of partial or complete softening resulting from cutting off of the blood supply by the swollen endothelial cells. Cloudy swelling in the ganglion cells was a universal observation. Reactive glial formation occurred. The process was not an encephalitis.

In the chronic stages, regression occurs. The endothelial cells of the vessels become shrunken and atrophic; the media becomes swollen and hyalinised and the adventitia fibrous. Organisation takes place in the areas deprived of their blood supply.

Recent work from the chemical standpoint of altered pH of the blood in toxæmias and infections may well account for the pathological manifestations. Therapy directed toward this altered chemical state is indicated.

R. M. S.


The case is described of a woman of 51, whose condition was one of progressive motor weakness of the extremities, especially the lower, with increasing atrophy but without any objective and only trifling subjective sensory change. The deep reflexes were abolished. Apparently the symptoms came on after a febrile illness diagnosed as typhoid. After a steadily progressive course of about two years death ensued from myocarditis.

Pathological examination showed an advanced state of parenchymatous peripheral neuritis, coupled with pronounced interstitial change. Similar alterations considerably less in extent and degree were found in the spinal roots, while the cells of the ventral horns were practically normal. Investigation of the mixed peripheral nerves showed a distinct division in the bundles between normal and degenerated fibres. The healthy (presumably sensory) fibres were situated as a rule to one side in the fasciculi, and did not surround the degenerated motor elements.

S. A. K. W.


This fine histopathological study is based on the examination of no fewer than five typical cases of Oppenheim’s myatonia congenita, and merits careful attention. A large number of workers have persisted in arguing for the
anatomical identity of the condition with the infantile spinal muscular atrophy of Werdnig-Hoffman, but Bielschowsky shows that this view is fundamentally incorrect and that Oppenheim’s original contention embodies a true conception of the disease.

The essential pathology is as follows: (1) persistence of various embryonic features (details given) in the nervous system: (2) loss of, and changes in, anterior horn cells; (3) absence of vascular and ‘inflammatory’ reactions; (4) substitutive glial hyperplasia; (5) fibre poverty in anterior roots and peripheral nerves; (6) changes in muscle (fully described).

The problem for discussion may be expressed thus: Are the pathological alterations to be regarded as indicative of a process of degeneration in neural structures that have been normal; or are they significant of an arrest of development, a quantitatively and qualitatively insufficient neuroblastic supply for a developing organism? Bielschowsky examines this crux with great care and minuteness and decides for the latter hypothesis, viz., that the affection belongs to the class of congenital dysplasia or aplasia. Werdnig-Hoffmann’s atrophy, on the other hand, is degenerative. He admits the occasional finding in myatonia cases of some evidence of degenerative change, and believes therefore that under certain circumstances more than one process may be responsible. But the general conclusion he considers thoroughly established on purely pathological grounds, and it finds support in the admitted clinical fact, that cases of amyotonia congenita sometimes exhibit definite and considerable improvement under observation, as has once more been shown in a recent case described by Bernheim-Karrer (Zeits. f. Kinderheilk., xlv, 1928).

S. A. K. W.


The condition described a number of years ago by von Hippel and Czermak has usually been considered an angiomatos formation of retinal vessels, but as long ago as 1913 this view was challenged by Meller, who believed it to consist in reality of a primary hyperplasia of glial elements in the same structure, and suggested the term ‘gliosis retinae diffusa telangiectoides.’ The characteristic case here described in great detail was associated with a cystic glioma of the vermis and left cerebellar lobe, an association noted on numerous previous occasions and recently made the subject of study by Lindau.

A long critique of the views offered by Lindau can be summarised in the statement that pathological evidence proves the retinal condition to be primarily glial and not angiomatous, and the authors now propose it should be known as ‘glioblastoma retinae telangiectoides.’ Numerous minutiae of pathological interest are discussed in connexion with the case: it is pointed out that
although gliomata are often considered to occur singly microscopically study sometimes reveals incipient glial nodules invisible to the naked eye. The multiplicity of the condition in cerebellum (or cord) and retina is not mysterious. Attention is directed to the absence of 'Xanthomzellen' in the retinal tumour in this case, cells imagined by Lindau to be typical of angiomata.

S. A. K. W.

SENSORIMOTOR NEUROLOGY.


The motor discharges of an epileptic fit are shown to possess a cardiac parallel. Examination of the cardiac parallel shows that the abnormal behaviour is due to excitation processes of too great intensity to permit the responding organ to give a normal response. Accordingly it is deduced that the responding organ, mediating mind, must also possess a limited capacity to give a normal response. Fits are thereby indicated to be based on excitation processes of too great intensity to evoke normal activity of the responding organ. The results lead to a consideration concerning which element of an excitation process, colloidal aggregation or electrolytes, holds prior possession of the field of response. Prior possession being conceded to colloidal aggregation, theories are developed of the mechanism of the process hitherto termed 'repression,' but better termed 'expulsion,' shell-shock and emotional states.

Three kinds of unconsciousness are recognized: (1) That due to excitation processes of too small intensity. (2) That due to excitation processes of unfavourable composition. (3) That due to excitation of too great intensity.

C. S. R.


Clonic convulsions arise from the motor cortex when this is anatomically and functionally intact. Tonic convulsions arise from lower motor mechanisms in the period immediately succeeding an injury to the cortical motor mechanisms. After lapse of sufficient time, clonic responses may be elicited from them. But this is not evidence that clonic convulsions may and do arise from lower motor mechanisms when those of the cortex are intact. Clonic convulsions in children may develop from lower motor mechanisms before the fibres of the pyramidal system have become myelinated and functionally active. All parts of the motor mechanism act together as one mechanism when the brain...