cytomas. The tumour belonging to the former group is extremely rapidly growing and gives rise to acute mental changes. The average duration of life from the onset of symptoms was found to be only 9.1 months, whereas in the more benign astrocytomas it was three and a half years. Histological study reveals no pure type of glioma. The term spongioblastoma multiforme fails to cover all the elements contributing to its structure, and the name "glioblastoma multiforme" seems more appropriate. The occurrence of combined forms of glioma in the same tumour was as high as 14 per cent. Besides the combined forms, many tumours show transitions between related types of cells as, for example, between astrocytoma protoplasmicum and astrocytoma fibrille, ependymoblastoma and ependymoma, neuroepithelioma and ependymoblastoma, etc. One tumour was presumably formed almost entirely by oligodendrogli.a.

C. S. R.

SENSORIMOTOR NEUROLOGY.


In the experience of most observers epilepsy must be an excessively rare accompaniment of epidemic encephalitis. Professor Wimmer, however, has been able to collect no fewer than 32 of such cases. In 12 instances there were other neurological signs present, but in ten the epilepsy constituted the sole symptom. It must be stated that the accounts of the cases are not convincing as regards the diagnosis of encephalitis, and in only two or three were signs of Parkinsonism present; in the majority, other signs coexisted, such as areas of altered sensation, pyramidal types of hemiplegia, disc changes which are themselves outside the usual encephalitic picture. In one so-called monosymptomatic case in which a bloodstained spinal fluid was withdrawn, the diagnosis of spontaneous subarachnoid haemorrhage seems much the more obvious solution of the symptoms. Some diagnosis other than encephalitis which would harmonize better with the rest of the symptoms can indeed be suggested in almost every instance. Although the manifestations of epidemic encephalitis are admittedly protean, we feel that to broaden our clinical conception too far is to threaten the stability of encephalitis as a nosological entity.

M. C.


Brief outlines of the histories of 34 cases are given.

The point which the author makes is that a very slight recurrent attack beginning in childhood may be the precursor of major epilepsy in later life. Some of the early attacks he describes are uncommon and interesting, e.g., laughter and crying, excessive flow of saliva, belching, feelings of loneliness, everything "looking blue."
He urges the importance of attention to diet, bowels, exercise, sleep and emotional life. Bromide and luminal he finds are the most useful drugs, and he also gives thyroid in some cases. He has no sympathy with the view that emotional conflict of some sort can cause epilepsy.

P. W.

[221] Considerations on pyknolepsy (Considérations relatives à la pycnolépsie).

Under the composite title of "para-epilepsy" Vergara has included such other paroxysmal affections as narcolepsy, migraine, vaso-vagal attacks, dipsomania, periodic crepuscular states, and pyknolepsy. The last-named disorder resembles petit mal but differs in the large number of attacks, the absence of occasional major seizures, the lack of intellectual deterioration and the tendency to spontaneous cure. In an excellent review of the condition, Moreau deals with each of these criteria in turn, and gives eight personal examples. He emphasizes the inefficacy of bromide in treatment; he concludes that the two chief points of differentiation from minor epilepsy are the multiplicity of the attacks and their tendency to disappearance at puberty.

M. C.


Cerebral aneurysms may be caused by arteriosclerosis, septic emboli or congenital weakness of the vessel walls. The signs of rupture are those due to diseases causing the aneurysm, those due to pressure or irritation of the surrounding brain structure, and those due to subarachnoid haemorrhage. In a person suffering from hypertension or arteriosclerosis, or from a general or local infection, especially infective endocarditis, the sudden onset of headache, nausea, vomiting, unconsciousness and convulsions, and the presence of cervical rigidity and Kernig sign, disturbances in pupillary reflexes, blurring of the discs, papilloedema, diplopia, paralysis of the cranial nerves, disturbances of deep reflexes and the presence of bloody spinal fluid, point to a ruptured intracranial aneurysm.

Absolute rest in bed is the most important therapeutic measure; after the first confirmatory lumbar puncture, further punctures are contra-indicated.

R. M. S.


An insane epileptic patient was admitted to hospital in 1901. At that time neurological examination revealed no abnormality. In December, 1925, he was transferred to another institution under the author's observation. Epileptic fits still occurred at intervals, but owing to the patient's irritability and maniacal assaults, full neurological examination was never attempted. The
attendants noted, however, that movements of the limbs were slow and awkward; speech was monotonous but correctly articulated. Fourteen months after admission the patient died of influenzal pneumonia, at the age of 57.

Post-mortem investigation of the brain revealed a very thick blood vessel on the surface of the right hemisphere, extending upwards and backwards from the frontal operculum. Here there was a knot of large vessels which actually formed part of a vascular tumour (haemangioma). The cortex of the foot of the precentral gyrus and Broca's area was destroyed by this mass. Coronal section through the brain revealed the deep extent of the haemangioma. The white matter of the lower aspect of the frontal lobe was involved as far inwards as the outer wall of the lateral ventricle.

M. C.


This is a valuable communication meriting full study. It deals with those cases of hemichorea which are attributable to lesions in the mesencephalon, more particularly to those assigned by some writers to lesions of the corpus Luysii. A personal case of the kind is included. The author gives a long and useful discussion of the problem of choreic pathogenesis and his conclusion is, that all can be explained as being caused by "irritation" at one or other level on the course of the well-known cerebello-central path, which leads from the cerebellum by red nucleus and regio subthalamica to optic thalamus and cerebral cortex. He adduces evidence in favour of this view and excluding all attempts to explain chorea by lesions of striatal cells; he believes that striatal cases, such as they are, produce effects by "irritation" of the thalamo-cortical part of the above-mentioned system.

S. A. K. W.


In young children postvaccinal encephalitis begins acutely without obvious prodromata, nearly always with convulsions, unilateral or bilateral, which may continue for hours or days. Consciousness is lost at an early stage. The temperature rises, often to 40° C. or higher. Following the stage of excitation comes one of general paralysis with coma, the total duration seldom exceeding four days. In the case of adults the clinical picture is somewhat different. Convulsions are much less frequent, but local manifestations are common, in the shape of monoplegia, hemiplegia, or paraplegia. Meningismus of greater
or less degree usually accompanies these palsies, which are of the flaccid type. Deep and cutaneous reflexes are lost, and a Babinski response is not infrequent. Rectal and bladder impairment is often noted. Cranial nerve involvement has been remarked, sometimes of bulbar type. The spinal fluid is either normal or shows a moderate lymphocytosis. Speaking generally, the acuteness and the fleeting character of the symptoms are of diagnostic significance, for the palsies often disappear quickly and the reflexes return.

This symptomatology corresponds to a diffuse inflammatory state of the central nervous system. Its histological basis, as found in acute fatal cases, is invasion of the white matter of the neuraxis (though the grey matter is never entirely unaffected), with involvement (round the small vessels) of parenchyma, notably the myelin sheaths, and to a less extent the axis-cylinders. The myelin is attacked by gliogenic elements, undergoes fatty changes, and disappears. The glial reaction in the immediate vicinity of the affected vessels is very striking. Swelling of glial cells in relation to limiting structures (e.g., on the periphery of the cord and under the ventricular ependyma) is sometimes pronounced. Mesodermal reactions are highly variable; infiltrates in meninges and vessel walls are sometimes absent, sometimes well-marked. Actual changes in ganglion-cells are of minor degree and out of proportion to the severity of the general process.

It is natural to see in these findings a close resemblance to those of so-called acute disseminated encephalomyelitis of the adult, a condition long familiar to the neurologist. The exact nosological position of the latter, and its relation to disseminated sclerosis, have been a fruitful subject of controversy.

In the opinion of the author, acute disseminated encephalomyelitis has in recent years been definitely on the increase, an opinion held also by others. The clinical picture is one of encephalitis, or myelitis, or of a combination; its onset is acute and the symptoms often exhibit a surprising fleetness of duration. Death in the acute stage is exceptional. The histological appearances of several recent cases of the author are almost identical with those of postvaccinal encephalitis, especially in the involvement of the white matter; any difference consists in the relative diffuseness in the case of the latter, and relative local concentration in the case of the former. But this statement must be qualified as a result of a recent examination of a typical case of postvaccinal encephalitis, fatal on the eighth day, in which a distinct tendency to local lesions was found.

S. A. K. W.

[226] Acute tabetic ataxia, with rapid bulbar termination (Les ataxies aiguës tabétiques à terminaison bulbaire rapide).—L. VAN BOGAERT. Jour. de Neurol. et de Psychiat., 1929, xxix, 81.

In 1921, Guillain drew attention to the occurrence of acute ataxia in the course of tabes dorsalis; in such cases, the ataxia is transitory, responding to antisyphilitic treatment in the course of a few weeks. The two cases here recorded
differ in that fulminating bulbar symptoms appeared and rapidly led to death. In the first patient, a left facial palsy was followed by severe dysphagia, unilateral palatal paralysis, tachycardia and impeded respiration. Death supervened ten days later. The second case comprised somnolence, lingual and palatal hemiparesis, left-sided ptosis, tachycardia, Cheyne-Stokes respiration and syncope; the patient died after four days. Pathological study of these cases demonstrated, in addition to the characteristic changes of tabes, an extensive meningo-vascular inflammatory reaction throughout the central nervous system. Vascular reactions were particularly intense in the bulbar nuclei.

M. C.


A man of 51, suffering from double hernia, received a spinal injection of 'tutocain' for the production of spinal anaesthesia. The paralysis which followed did not disappear, and from the level of L4-5 downwards a complete motor and sensory interruption, with involvement of bladder and rectum, remained. Sixteen months after the operation cystopyelitis and tuberculosis of the bladder resulted in death. Pathologically a severe toxidegeneration of conus and cauda equina was found, with ascending changes in the posterior columns of the cord.

It appears that another case operated on at the same time, in which an ampoule from the same batch of 'tutocain' was used, was followed by similar lumbosacral paralysis. The authors are unable to explain the result through any error of technique, and are rather at a loss to account for it, since the same agent has often been employed with complete success. They suppose that in their case the patient's nervous system was already predisposed to severe reaction from a relative light 'lesion,' because of preceding tuberculosis and syphilis.

S. A. K. W.


In some cases of slight peripheral facial palsy, patients complain of an asymmetry which appears only when they smile. This apparent dissociation between voluntary and emotional movement may be the only feature remaining after a Bell's palsy. It is explained thus: movements of moderate range are performed equally on the two sides of the face; volitional movements are easily kept within this "subliminal range" and hence no asymmetry appears. When, however, the patient gives a broad smile of genuine emotion, the movement is no longer confined within the subliminal range, and asymmetry at once becomes obvious.

M. C.

The author first outlines a classification of possible causes of nuclear and infranuclear facial paralysis. He then describes a case of polyneuronitis with cranial nerve involvement, and pleocytosis in the cerebrospinal fluid. The second case he outlines is one of myasthenia gravis in which the sudden onset of bulbar and facial pareses caused a misdiagnosis of polyneuronitis to be made.

The differential diagnosis between this condition, acute epidemic encephalitis and polyneuronitis is then discussed.

Parinaud’s syndrome with preservation of automatic reflex movements; vesperal hallucinations in the course of a neurotropic infectious syndrome of unknown cause (Syndrome de Parinaud avec conservation des mouvements automatico-réflexes; hallucinose vespérale, au cours d’un syndrome infectieux neurotrope d’origine indéterminée).—L. van Bogaert and R. Delbeke. *Jour. de Neurol. et de Psychiat.*, 1929, xxix, 91.

The main clinical features of this case were:

1. An acute infection with fever; pleocytosis and hyperalbuminosis of the spinal fluid; sleep lasting seven days; emaciation; general akinesis with rigidity and cerebellar manifestations. The tendon jerks were present but no abdominal responses were obtainable.

2. Abolition of voluntary eye movements although automatic movements were present. Later, the power of voluntarily looking to the side returned, associated with nystagmus.

3. A peculiar episodic hallucinatory state, coming on especially towards nightfall.

The authors discuss the diagnostic possibilities of their case with particular reference to disseminated sclerosis, syphilis, and epidemic encephalitis.


A case is recorded of a man who had marked symptoms of Raynaud’s disease in both hands. The fingers were slatey blue and there was trophic disturbance of the nails: further, he had pains in the hands of paroxysmal type. Owing to the severity of the symptoms removal of the right inferior cervical sympathetic ganglion was undertaken, but the patient died. At post-mortem a large carcinoma of the lesser curvature of the stomach was found, and in the inferior cervical ganglia a metastasis. The authors conclude that the symptoms of Raynaud’s disease resulted through stimulation of the sympathetic ganglia owing to the deposition of metastatic cells in their proximity.
A large number of children suffering from enuresis have been examined critically: 129 were boys and 123 girls. The author concludes that the voluntary nervous system plays an unimportant rôle in enuresis and that the condition is a disturbance of the physiology of micturition, probably due to stimuli arising in the bladder itself, which for the time being place it beyond the control of the will. Consequently disciplinary measures are found ineffectual. Most benefit was derived from the administration of atropine and from massage of the bladder: this form of treatment resulted in a cure in 80 per cent. of the cases.

E. A. C.

The author gives a classification of the types of hypertrophy occurring in infancy and childhood. He divides them into true and false hypertrophies. In the true the skeletal system as well as the muscles and soft tissues is involved. The skin may be rough and thickened and may show pigmented areas, naevi and telangiectases. Characteristically it does not pit on pressure. The true hypertrophies may be partial, that is, affecting one digit or limb only or they may affect one half of the body. They may also be crossed, e.g., the right arm and left leg being affected. Nothing definite is known as to the etiology of either the congenital or acquired variety.

The false hypertrophies are those in which the skeletal system is not involved. Under this heading the author includes Milroy's disease and congenital elephantiasis. In Milroy's disease the skin itself is but little affected though some fibrosis occurs in the subcutaneous tissues. In congenital elephantiasis there is marked fibrosis and an increase of fat in the connective tissue layers of the skin, together with a variable distention of the lymphatics and capillaries.

P. W.

PROGNOSIS AND TREATMENT.

The study is based on the examination and treatment of 61 cases diagnosed as subacute combined degeneration, admitted to hospital during the last four years. The criteria on which the diagnosis has been made do not appear open to criticism, although the authors have excluded cases with loss of deep reflexes because "a certain number of patients with pernicious anaemia exhibit these phenomena" (sic). A study of the geographical distribution of subacute combined would appear called for and likely to prove of value, if an average of 15 cases a year (and of the above-mentioned limited class) is found at the Royal Victoria Infirmary of Newcastle.


