In 1927 the authors observed a patient with sensory aphasia who would have hurt herself severely if left alone. She pushed everything that came into her hand against her eyes, heedless of the pain she thus inflicted on herself. Further examination showed that she did not react to pain or only in an incomplete and local way. There was no real defence action. Sometimes there was a local withdrawal; the facial reaction of the patient was that of slight pain. When exposed to a strong faradic current she showed pain reactions, but not a real defence. The patient perseverated in actions once undertaken. The perseveration and the sensory aphasia almost disappeared, whereas the changed attitude toward pain persisted. The patient also did not appreciate threatening gestures and was insensitive to loud noises and sudden flashes of light.

At autopsy a gross lesion was found in the gyrus supramarginalis, but there were also softenings in other situations.

Two other cases seemed to confirm the localisation of the symptoms in the anterior part of the lower portion of the left parietal lobe, and up to the present time the authors have observed ten cases. In their experience all types of lesions may be the cause of the symptom—softening, haemorrhages, tumours, syphilis and fractures of the skull.

The conclusion is reached that a lesion in a particular region of the left parietal lobe makes it impossible to build up a full perception of pain. Asymbolia for pain is not alone an expression of a deficiency; there is also a particular attitude toward pain which lacks the integration into a higher propositional act. It is from this interesting point of view that the disturbances resemble closely the reactions in a particular group of catatonic cases; but it seems that in the catatonic cases the lack of the reaction to pain has a closer connexion with the individual problems of the patient.

R. M. S.
(commissural and intra-hemispheral), and loss of left prefrontal, frontal, and parietal substance. The patient was right-handed, a polyglot, and recovered gradually the idiom in which he had been systematically reeducated. In his mother tongue, in which he was not reeducated, there was practically no recovery. After examination of the interior of the brain with serial sections for myelinization, the author undertakes a minute review of the causes of recovery and discusses some fundamental problems of the physiopathology of speech.

R. G. G.

[99] The relation of time of day, sleep, and other factors to the incidence of epileptic seizures.—Frederick L. Patry. Amer. Jour. of Psychiat., 1981, x, 789.

Over a period of a year 31 epileptics were studied. It was found that they fell into three groups—those in whom the attacks were predominantly diurnal, mainly nocturnal, or occurring indifferently by day or night. Attacks were more frequent in the nocturnal and diurnal groups than in the diffused group. Approximately two-thirds manifested a marked difference in the diurnal and the nocturnal incidence of their convulsions and two-thirds exhibited a concentration of attacks at or around certain hours. There appears to be a basic distinction between the patients in the respective groups depending on endogenous and exogenous factors, important among them being the type of sleep, transition from waking to sleep and vice versa and consequent postural and circulatory changes, alveolar CO₂ tension and temperature variations. Many showed a regular periodicity of fits, not only with respect to day, but also the day of the month, and the month of the year and season. All three groups showed a greater seasonal incidence during the spring. A larger percentage of the diffused group craved salt, milk or meat than of the other two groups. Various forms of excitement precipitated more fits in the diurnal and nocturnal types. In the latter group there was a higher percentage of mental instability than in the others. Individuals in the diffused group are more likely to have a longer psychotic period. Patients showing isolated single fits are more numerous in the nocturnal and diffused. The nocturnal type seems more prone to status epilepticus. The diurnal group shows the longest interval between attacks, and the diffused group, the shortest. There is a tendency for the number of fits as well as the time duration of the auras to diminish with the increased duration of the disease. The regular occurrence of nocturnal fits and to a somewhat less degree of diurnal fits is usually a poor prognostic sign. The generally held view that the younger the individual at the time of onset of the convulsive state, the longer the duration of the period of fits, seems to be confirmed by these findings.

C. S. R.

Two patients with defect of cranial bones, one of whom suffered from epilepsy, were studied. Pressure on the sinus caroticus caused evident changes in brain volume (by the plethysmograph) not found in the epileptic. Epileptic attacks were accompanied by an increase in brain volume. The increase in volume produced by intravenous injection of adrenalin was less in the epileptic patient than in the other.

The conclusion is that in epilepsy there is a reduction in the carotid sinus reflexes as a result of which the conditions of cerebral circulation in that affection are altered.

S. A. K. W.


Encephalographic studies were made on a group of 17 patients with recurrent convulsions of unknown origin (‘idiopathic epilepsy’). None of the patients showed clinical evidence of a focal lesion. This selection was made purposely, with the aim of ascertaining possible pathological changes in the idiopathic group.

The ages of the patients ranged between 18 and 49 years. Three showed no evidence of mental deterioration, one was mildly deteriorated, while the rest were in a more or less advanced state of deterioration. In eight patients, or 47 per cent., the observations were normal; in four, or 24 per cent., dilatation of the ventricles and of the subarachnoid space and some evidence of leptomeningeal adhesions were found. In five other cases, or 29 per cent., the encephalographic observations seemed to point to a definite pathological process, inflammatory or perhaps neoplastic, but apparently not active. No correlation could be made between the degree of the deterioration and the extent of the pathological changes.

Air was still present after 24 hours in all cases, and sometimes after 216 hours. Its presence seemed to correspond more to the extent of the dilatation of the ventricles and of the subarachnoid spaces than to the amount of air injected. Subarachnoid dilatations may rarely be seen in roentgenograms taken before the injection of air.

R. M. S.

Investigation of the capillaries in cases of idiopathic epilepsy leads the author to conclude that genuine epilepsy is in the vast majority of cases due to exogenic rather than endogenic influences.

The brain in some instances (dystocia) may suffer sufficient trauma during birth to give rise to capillary haemorrhages. The more protracted and pathological the delivery process, the greater seems the tendency to them. They may be regarded as the starting point for the metabolic disturbances.

From the data here presented it is concluded that 'genuine epilepsy' has its probable etiological substrate in capillary haemorrhages occurring at birth, which in turn give rise to the development of the metabolic imbalance and, when severe enough, cause the convulsion.

R. G. G.


The author reviews the literature and points out the confusion which exists in regard to this subject.

He describes thirteen cases of his own—two, alcoholic epilepsy; three, poriomanic affect-epilepsies; three, hystero-epilepsies; and five, cases of psychopathy with so-called affect-epilepsy. There is a strong psychopathic background in practically all cases. The author on this basis suggests the possibility of a hysterical manifestation transforming itself into a more severe organic reaction of an epileptic nature. All idiopathic epileptics do not deteriorate. Petit mal does occur in affect-epilepsy. Hyperpncea during affective states in psychopaths is held to be significant both in relation to the attacks and the aphonie conditions. Vasomotor disturbances and an overflow of senso-affective stimuli from the thalamus are also suggested as possible causal agencies. Alcohol itself may produce convulsions as a toxic agent or by causing biochemical alterations.

The suggestion is made to omit all the various classifications and to consider one cryptogenic group of convulsive states with a multiplicity and variety of etiological factors, in addition to the unknown 'something' which gives rise to the convulsive attacks.

R. G. G.
NEUROLOGY

[104] Narcolepsy.—H. A. CAVE. *Arch. of Neurol. and Psychiat.*, 1931, xxvi, 50.

From a study of forty-five cases of narcolepsy found in the records of the Mayo Clinic during the years 1919 to 1928 Cave concludes that the essential features of this condition are as follows: Narcolepsy is characterised by the occurrence of sudden attacks of sleep and of cataplexy. While the attacks of sleep present many characteristics that are foreign to normal sleep, there is sufficient similarity between them to justify the clinical application of the term sleeping attack and the assumption that they may have a common etiological mechanism. All gradations between sleeping attacks without cataplexy to cataplexy without sleeping attacks may occur. Narcolepsy occurs in both sexes and is essentially a chronic condition; although it may incapacitate the person so afflicted, it apparently does not shorten his life. The sudden onset, brevity and rapidity of recovery of the two types of attack are sufficiently similar to suggest a common etiology. The occurrence of dreams during the diurnal attacks of sleep, together with the marked nocturnal restlessness and vivid dreaming, indicates that one is dealing with a mechanism that is disturbed nocturnally as well as diurnally. Narcolepsy may occur as a sequel of epidemic encephalitis. The association of obesity with narcolepsy points to a disturbance of the vegetative centres of the brain and of the endocrine system. Narcolepsy shows many clinical manifestations similar to other conditions, especially myoplegia and epilepsy.

Pavlov's work on conditional reflexes with the production of sleep as a result of internal inhibition is reviewed and the theoretical relation of this work to narcolepsy is discussed. The relation to narcolepsy of the sleep that may be produced by organic lesions of the floor of the third ventricle and the cerebral aqueduct is discussed. Attention is drawn to the marked loss of muscular tone and abolition of deep reflexes with the occurrence of an extensor plantar response in both cataplexy and normal sleep.

R. M. S.


In this long paper the late Professor Redlich discusses and analyses a series of outstanding questions relating to the problems of narcolepsy. The author inclines to separation of 'true' from 'symptomatic' (metencephalitic) narcolepsy, although it is clear that he sees the disadvantages of such a division. He collects some 28 cases of narcolepsy following encephalitis, and affirms that the main point of distinction between the two groups is the...
relatively good prognosis of the latter. How far this is justified in the first place, or of any value nosologically in the second, are matters on which the author's contentions might readily be challenged. Among the cases cited are several of narcolepsy following other infections than epidemic encephalitis. Apparently the writer is also inclined to exclude those not distinguished by loss of tone (cataplexy). So far as the question of relation to epilepsy is concerned, the conclusion is that typical cases of narcolepsy have nothing to do with the other, but the admission is made that a combination of the two is possible, and also that in some respects attacks in either group may offer features resembling those of the other.

S. A. K. W.


A case of thrombosis of the superior cerebellar artery is described, and a similar case is quoted from the literature; both cases show a definite combination of symptoms, viz., loss of pain and thermal sense on the homolateral side and ataxia, atonia and asthenia on the heterolateral side. Such a combination of symptoms could not be produced by a single lesion in any other place, and might well be named the syndrome of the brachium conjunctivum and the tractus spinothalamicus.

R. M. S.


A paralytic-dystonic syndrome with cerebellar deficiency is described resulting from a traumatic lesion in the region of the right crossed pyramidal tract of the fourth cervical segment.

The analysis of the symptomatology seems to indicate the existence in the area of long tracts from the cerebellum similar to those connected with the red nucleus which exert an influence on the spinal centres. It would also seem that efferent tracts identifiable with the reticulo-spinal fibres accompany the crossed pyramidal tracts coming from the supraspinal centres concerned with the maintenance of tone.

R. G. G.

The author describes the case of a youth of 27 who during life showed a spasm of the neck (which started five years previously with a tonic torticollis) and a rigidity of the antigravity muscles of the decerebrate type, which became constant some months before death. Besides this he had severe spastic dysarthria which was followed eventually by a complete anarthria. The spasm was most obvious in the erect attitude and diminished in the prone position. No pyramidal or cerebellar symptoms were observed.

Cellular and myelin degeneration of the corpus striatum and of the substantia nigra and the sixth layer of all the frontal and insular areas were found. In the degenerated zones there was noticed an enormous invasion of neuroglial nuclei with few processes. The pyramidal tracts, cerebellum and red nuclei were normal. The liver was normal.

The other possible syndromes to which this case might belong are discussed but the author thinks that it is one of primary progressive dystonia of posture. The syndrome is associated with a primary systemic degeneration of the striate and of the neural areas known to be associated with the fifth and sixth layers in the frontal area, and with the locus niger. It is allied to Wilson’s disease and may be due to endogenous intoxication.

R. G. G.


Spinal neurofibromas invade the bone only by direct pressure and consequent erosion. The earliest demonstrable change is thinning of the medial border of the pedicle of the vertebra at the site of the growth. One or both pedicles may be involved according to its size. Changes in the contour of the bodies of the vertebrae are among the last signs to be recognised.

A spinal endothelioma produces similar alterations except that it does not extrude through the intervertebral foramen, that structure never being involved by the lesion. Occasionally calcified bodies accompany the tumour and are easily seen.

Intramedullary tumours rarely show any change unless they have caused such expansion of the cord as will produce pressure effects.

Ependymoma causes erosion; it expands the bone by direct pressure and the margins of the eroded bone are sharp and well-defined. More than one vertebra may be involved.
Chordomas produce massive erosions of the sacrum and complete destruction of laminae and spinous processes. The margins of involved bone are irregular and not clean-cut.

Dermoid tumours and the antesacral group commonly known as Middelrod tumours are capable of producing localised erosions in the body of the sacrum but rarely invade and destroy laminae and spinous processes.

Osteochondroma arises usually from the various bony processes of a vertebra rather than from its body. Contiguous bony structures are distorted and eroded.

Hæmangioma of the vertebrae causes irregular absorption of trabeculae with thickening of vertical trabeculae, the result being parallel vertical striations in the body, which may extend into arches and laminae.

Osteogenic sarcoma results in dissolution and destruction of bone, with secondary involvement of paravertebral soft tissues.

The X-ray characters of metastatic malignant growths are well-known.

J. V.

**PROGNOSIS AND TREATMENT.**


Sedative, narcotic and anesthetic drugs may be divided into two distinct groups. The administration of drugs of the first group results in the phenomena of release and activation and finally of muscular flaccidity. This may be interpreted as being due to the successive involvement of the several neurones that make up the long or cerebral reflex arc. Administration of drugs of the second group results in an absence of phenomena of release and activation. This may be interpreted as being due to a simultaneous involvement of all of the neurones that make up the long or cerebral reflex arc. The clinical picture of the effect produced by the first group of drugs is similar to that of the epileptic seizure and to the onset of normal sleep. In the same manner that the onset of normal sleep is favourable to the occurrence of the epileptic seizure, so the action of these drugs is rather favourable than otherwise to the occurrence of epileptic seizures. The action of the second class of drugs, which tend to counteract the symptoms of the epileptic seizure, is like that of exceptional sleep that is devoid of release phenomena and characterised from the beginning by muscular relaxation. Such sleep can be produced artificially by means of a mechanical appliance. Like the second class of drugs in question, therefore, it has the effect of temporarily diminishing the frequency and severity of epileptic seizures.

R. M. S.