killed at intervals from twelve to one hundred and seventeen hours and the distal segments examined by physiological and histological methods. For the functional study, the cathode-ray oscillograph was employed to record the conducted action potentials of the severed nerves and their controls. For microscopic examination, chief reliance was placed on the appearance of fragmentation in the medullary sheaths, as evidenced in osmic acid-stained preparations. Ranson’s pyridine-silver modification of the Cajal method was used to check the relative rates of degeneration in myelinated and nonmyelinated fibres.

Results of the physiological study showed that increase in the absolutely refractory period may be detected as early as twelve hours after section. Significant reduction in potential area was usually not apparent before thirty-six hours. The fibre groups of somatic and visceral nerves characterized by different physiological properties lose their action potentials in sequence. The potential from visceral afferents disappears first at from fifty to seventy hours; the potential from somatic motor and sensory fibres, five to ten hours later; then the potential from myelinated autonomies, and finally, the potential from unmyelinated fibres. A potential from unmyelinated fibres has not been elicited later than ninety hours postoperatively.

In differential counts of three groups of myelinated fibres (large fibres, selected range, diameter from 6.7 to 18.1 microns, sheath thickness, from 1.4 to 2.4 microns; medium-sized, selected range, diameter from 4.8 to 6.5 microns, sheath thickness, from 0.43 to 0.85 micron; fine, selected range, diameter from 1.4 to 3.0 microns, sheath thickness, from 0.4 to 0.65 micron), fragmentation was set as an arbitrary criterion of degeneration. Those fibres not exhibiting fragmentation in the portion available for study in a 5-micron longitudinal section, stained with osmic acid, were counted in one group; those exhibiting fragmentation in at least one place, in another. The comparative values indicate that fragmentation occurs earlier on the average in the medium-sized, relatively thin-sheathed group, next in the large, myelinated fibres, and finally in the smallest, myelinated ones. Numerous nonfragmented unmyelinated axons persist at a stage when in osmic acid stained preparations the sheaths of almost all myelinated fibres appear to be fragmented.

R. M. S.

NEUROPATHOLOGY.


In contrast with what obtains in other cases of meningoencephalitis, the lesions of poliomyelitis in the brain first cause infiltration of the
central substance and move outwards; the meninges are involved from the cerebral side. Focal manifestations may be altogether lacking although vascular infiltrates are present. The lesions of the vascular system in the neuraxis do not help to solve the problem of local affinity. They are the expression of a resorptive 'inflammation,' a distant reaction to primary inflammation of grey matter. The focal lesions are not clustered round vessels invariably or often. Since the brain lesions are those of an encephalomeningitis, it is clear the fluid does not convey infection.

In the motor zone of the cortex, no systematization of the lesions can be traced; the virus has no selective action on motor ganglion-cells, nor is neuronophagia or cell-outfall so marked as in the case of the cord. In the latter, again, any part whatsoever of the grey matter may be affected, with the exception, perhaps, of the substantia gelatinosa and the tip of the dorsal horns. Speaking broadly, the anterior two-thirds of the grey matter contains the bulk of the lesions. Within that area, wide variations occur. Dorsal horns and Clarke's columns may be more diseased than any other section. Spinal root-ganglia, gasserian ganglion, and the nuclei of the cerebellum may be involved. Many motor nuclei, on the contrary, are often spared. In other words, poliomyelitis is no disease of the motor system, and attacks mechanisms whose functions are quite diverse. The virus also damages substantia nigra, globus pallidus, thalamus, etc.

The only permissible conclusion is, that neither the vasal factor nor the system factor accounts for the histopathological phenomena. Local vulnerability, that is to say, has not yet received a convincing explanation. This affinity of the virus for certain masses of grey substance is independent of the spot at which the virus enters, or is made to enter, the body—an old observation, though some writers are under the impression it is novel. No theory has as yet accounted for the observed fact that the anterior two-thirds of the spinal grey matter harbour the maximum number of foci.

As is known, evidence is accumulating which goes to show that the virus travels to the spots it favours by means of axonal spread.

The paper finishes with a discussion of the reasons for the differences of the histological reactions in different places and alludes to the occasional occurrence of lesions in white matter.

S. A. K. W.


In this contribution the authors have been concerned with the changes in classic neuroglia as the result of injury to the human brain. In the first few days following trauma, the astrocytes in the immediately adjacent area
undergo regressive changes with the formation of amœboid glia. In the zone nearest the point of injury the cells undergo complete destruction. Beyond this zone of destruction active proliferation takes place. Here, within a few hours after injury, double nucleated forms indicate active direct cell-division. In a rather narrow intervening zone, apolar, bipolar and unipolar forms have been found, which, because of their ready impregnation with gold sublimate, are probably viable cells. This is furthermore suggested by the presence in these cells of double nuclei having normal morphological characteristics. These elements are interpreted as regressive forms capable of reversible action.

Gliosis occurs only as a result of tissue destruction. The degenerating tissue probably serves as a chemical stimulant to the regional astrocytes. In the formation of the glial scar, glia-cells assume their active or kinetic function of space compensation in the process of healing. Extensively damaged brain tissue, which requires a rather prolonged activity on the part of the phagocytic mesoglia, delays for a considerable period the formation of the ultimate glial scar. The astrocytes taking part in this delayed reaction are themselves often victims of regressive change, particularly of a hyaline nature.

Judging from studies on comparatively recent cases, it seems likely that the neuroglial reaction is a purely local affair and consequently that generalized gliosis following head injury does not occur. The remote clinical sequelæ of such injuries are largely to be interpreted on the basis of focal and distant cortical injuries. In addition, there are numerous interruptions in the connecting pathways incident to focal and subcortical injury, together with the changes following widespread petechial haemorrhages. In this sense localized gliosis is only a histological manifestation of the damage which produces the symptoms.

R. M. S.


In 1928 the author found spirochetes for the first time in the brain of a case of disseminated sclerosis. By September, 1930, the number of 'positive' cases has risen to 7 out of 24. A description of his findings was given before the Neurological Congress of Berne last year, and slides of the 'spirochaeta myelophthora' shown. In addition, Dr. Steiner drew attention to the occurrence of 'silver cells' in the periadventitial lymph-spaces in the same affection, cells which appeared to be laden with what might be considered spirochetal debris. Similar argyrophil inclusions were to be seen in round cells in the parenchyma, in the neighbourhood of infiltrated vessels.
The present communication deals with the same questions and is illustrated with a number of microphotographs. A new case has proved to be richer in spirochaetes than its predecessors; and by means of a Zeiss multiplicator it has been possible to photograph in series, at different optical levels, the objects under discussion. From the pictures there cannot be any doubt of their spirochaetal nature. The fresher and more acute the lesion, the more readily may spirochaetes be expected therein; chronic and organized lesions seem to be free from them.

This communication deserves close attention on the part of the neurologist and is conspicuous for the temperate and judicial character of the author’s comments on his results.

S. A. K. W.

[75] Research on the microglia and oligodendroglia (Ricerche sulla microglia e la oligodendroglia).—D. BOLSI. Riv. di pat. nerv. e ment., 1931, xxxvii, 1.

The author shows that the microglia under the influence of certain toxic factors may show acute swelling which Penfield and Cone thought was a specific reaction of the oligodendroglia; there is however no evidence of any true similarity in structure or function of the two types of cells, which much research has proved to be fundamentally different. He further describes his own method of vital colourization, which confirms him in his opinion that the microglia should be classed in the reticulo-endothelial system.

R. G. G.


The interest of this case consists in the association of multiple tumours with a unilateral cerebral sclerosis secondary to a postnatal meningoencephalitis. The assertion is commonly made that tuberous sclerosis alone is apt to show this connexion, but according to the history and description of this case the statement must be reconsidered. The tumour lesions consisted of a hypernephroma of the kidney, lipoma of the diaphragm, and fibromyoma of the stomach. The brain lesions were clearly those of a postnatal inflammatory process involving both cortex and meninges on one side, with resulting atrophy of the hemisphere.

S. A. K. W.

The occurrence of paralysis of the cervical sympathetic in cases of unilateral lesion of the medulla is familiar to the neurologist, but the exact position of the 'centre' has not been located satisfactorily. Cases of thrombosis of the posterior inferior cerebellar artery can be utilized for the purpose, though the area involved is apt to be rather too large and the syndrome too complicated. Dr. Winther has had the opportunity of studying pathologically four cases in point, and comes to the conclusion that the enophthalmus of the syndrome is not caused by paralysis of the orbital muscle of Müller but is due to atrophy of orbital fat. The sympathetic paths concerned are situated in the lateral part of the formatio reticularis, between the nucleus ambiguous and the descending root of the fifth. The ocular fibres are close to the former; those for the face are rather in front of the others. Those for the body are still farther forward, in relation with the spinothalamic tract.

S. A. K. W.

The Kahn test applied to spinal fluids.—M. B. KURTZ and N. W. LARKUM. Amer. Jour. Syphilis, 1932, xvi, 377.

The results obtained with a modified Kahn test on the cerebrospinal fluid are detailed. In order to achieve greater sensitivity, concentrations of 20 in 1, as well as the usual 10 in 1, were used and the fluids were also tested with 'presumptive' Kahn antigen (see this JOURNAL, vol. x, p. 386). Special precautions were taken to avoid an excess of ammonium sulphate in the concentrated globulin solutions. By these improvements the authors claim to have increased the number of syphilitic fluids which give a positive Kahn test. They place no reliance however on the 'presumptive' antigen technique, which is only used as a 'check on the general technique of the test.' But they consider that unless concentrations of at least 15 in 1 are tested with standard antigen the result should be returned as an incomplete test.

J. G. G.


This anatomical study seems to establish the following facts.

1. The presence of anomalous sinuses and asymmetrical development of the transverse and sigmoid sinuses are common in epilepsy.
2. The occipital bone in epileptics is frequently smaller than normal, resulting in a small posterior fossa.

3. The accumulation of cerebrospinal fluid occurs first over the cerebral hemispheres, beginning in the frontal and extending backward, and later resulting in dilatation of the ventricles.

4. Encephalograms should be taken routinely in epilepsies to reveal changes in the venous sinuses, the size of the occipital fossa and the distribution of cerebrospinal fluid.

With these facts established, an effort is being made to ascertain whether by operative procedure it is possible to relieve the pressure against the sigmoid sinus and thus stimulate the return venous blood from the brain. In a series of 104 operative cases, most of which have been done at the Children's Orthopaedic Hospital in Seattle, it would appear that the operation has offered a cure in 13 cases, approximately 10 per cent. (duration of observation four years) and been of marked benefit in 50 per cent. The technique for the operation has not been perfected, nor is it yet known what cases will be benefited. Apparently cases in which there is marked cerebral atrophy are not particularly improved, at least no more so than by withdrawing the fluid intake. The most favourable cases are those in which there are demonstrable accumulations of cerebrospinal fluid but not particular enlargement of the ventricles.

R. G. G.


This is a long and well-documented study of the epilepsy that appears late in life, with a full bibliography. It is based on the analysis of some 35 cases of so-called ‘involution epilepsy.’ In a narrow sense epilepsy as a symptom of senile change in the parenchyma itself is an altogether improbable occurrence, but the arteriosclerotic variety is common. Its relation to cardiac and vascular disorders, to vascular cerebral lesions, and to derangements of nutrition of the same derivation is discussed. Arterial hypertension is claimed to be a potent cause of the condition, and a direct one. Yet the author is unable to get away from the conception of an ‘epileptic disposition’ (whatever that may be) acting as a supplement to the vascular factor.

S. A. K. W.


This somewhat inconclusive study from the Phipps Institute showed the following results:
The alkali reserve, determined in 81 dispensary epileptic patients, ranged between 54-74, extreme normal limits.

Blood calcium was measured in 86 ward patients and in 26 out-patients. It was found to be within normal limits (9-11 mgm. per cent.) in the out-patients, and 8·1-11 mgm. per cent. in the ward patients. In this last-named series of 86 cases, eight showed subnormal values, ranging between 8·1-8·9 mgm. per cent.

Calcium in the cerebrospinal fluid was found to be within 4·3-5·8 mgm. per cent. It was below normal in one case and above the normal in two out of 86 cases.

The alkali reserve figures and the blood calcium content of the author's epileptics, apparently, do not contribute to the concept of a basic relationship between epilepsy and tetany.

The fluid Ca/blood Ca ratios were within the limits 46-65. Ratios which may be considered as high normals were found in seven cases, and ratios distinctly above the normal in four cases.

No relation could be found between the calcium findings (in blood, fluid and ratios) and the mental state of the epileptic patients; nor have these findings, apparently, been influenced by the occurrence and frequency of convulsions, in so far as the latter took place within the two days before blood and spinal fluid were withdrawn.

The high fluid Ca/blood Ca ratios in 11 out of 86 cases and none with low ratios, suggest that there is in epilepsy a tendency to an increased permeability to calcium of the barrier between blood and fluid.

R. G. G.


In animals after various surgical procedures upon the nervous system, after intravenous injections of glucose, tapwater, adrenalin, ammonium carbonate, luminal and potassium borotartrate, as well as after adrenalectomy and nephrectomy, there is an approximation of the minimal convulsive to the lethal doses of absinthe in experimentally produced convulsions as compared with control animals. When tapwater or ammonium carbonate is injected intravenously the ' convulsive factor ' is almost zero per cat and per pound as compared with 0·21 and 0·03 gm. respectively in the control animals. There seems a reduction in the number of the fits after adrenalectomy. Dilatation of the stomach is the most prominent symptom observed, that of the colon next, and contraction of the colon next. Some central and peripheral lesions of the nervous system show constant effects in dilatation or contraction of the parts of the digestive tube. These are not necessarily in
accordance with accepted neurophysiological principles. Emptying of the
gall-bladder does not seem to be affected by the violent respiratory reactions.
The occurrence of widespread and extensive petechial haemorrhages in the
lungs and congestion of other organs is a constant finding in all groups of
experimentally produced convulsions.

C. S. R.

[83] Experimental researches on subcortical epilepsy (Recherches expéri-
mentales sur l'épilepsie sous-corticale).—G. Marinesco, O. Sager,
and A. Kreindler. Bull. soc. romaine neurol. et psychiat., 1932,
xiii, 17.

In these experiments an attempt was made to reproduce the clinical features
of tonic epilepsy. Cats were chosen for the purpose, and choline was
administered by intraventricular injection (1 mg. in 0·1 c.c. of Ringer's
solution). Tonic attacks occurred, with trembling, and were accompanied by
salivation, mydriasis, and incontinence. After the attacks the animals fell
into a deep sleep.

In another cat, deprived previously of its cortex by two operations, and
still living after eight months, analogous attacks occurred seemingly in a
spontaneous fashion. It also, however, manifested symptoms like those of
fugue, without fits, and affective attacks of rage.

The authors believe that their work tends to show that periventricular
vegetative centres play an important part in the genesis of the epileptic fit,
and that the tonic phase is of subcortical origin.

S. A. K. W.

[84] The permeability of the haemato-encephalic barrier in epilepsy, as
determined by Walter's bromide test.—S. Katzenelbogen. Jour.

The distribution ratios of bromide between blood and cerebrospinal fluid in
epilepsy was found to be within normal limits in 42 out of 87 cases (48·3
per cent.).

A distinct increased barrier permeability was observed in 12 out of 87
epileptic patients (13·8 per cent.).

A moderate rise above the normal permeability was found in 24 out of
87 cases (27·6 per cent.).

A decreased permeability was noted in nine out of 87 cases (10·3 per
cent.). There was a marked decrease in three, and a moderate decrease in
six cases.

As tested by Walter's method, the barrier permeability in epilepsy is
likely to be found either normal or more or less increased in most cases.
There are relatively few chances to find a decreased barrier permeability to bromide in epilepsy.

The tendency toward a high permeability to bromide in epilepsy and the particular efficacy of the bromide treatment in this disease concur in contributing, apparently, to the concept that substances introduced into the general circulation affect the cerebrospinal system through the fluid.

The susceptibility of the barrier to a high permeability in epilepsy is also significant when compared with the frequent observation of an increased permeability in certain organic diseases of the cerebrospinal system and in conditions associated with meningeal congestion.

R. G. G.


The chlorine balance in adult epileptics shows a marked disturbance. Experimental evidence points to the existence of an abnormal tendency to retain sodium chloride. The capacity to store sodium chloride varies within wide limits and there seems to be no relationship between the number of convulsions or equivalents in the mental condition and the amount of sodium chloride stored. In some cases moderate or large doses of sodium chloride have the tendency to favour convulsive attacks or equivalents. According to experimental and pathological investigations the excess sodium chloride is probably stored up in the integument without consecutive water retention. The controls under the same experimental conditions showed no retention. From the data available at present the authors do not feel justified in giving an explanatory theory as to why the sodium chloride is stored up. The possibility of a central or endocrine disturbance is most likely because disturbance of the renal excretion cannot be demonstrated. If further investigations confirm the salt retention in all forms of the convulsive state, the rest may be of diagnostic value, especially in cases where no convulsions or equivalents are noted but where mental upsets of a doubtful nature exist.

The mechanism of the water balance in epilepsy is still obscure. From the data reported there seems to be no disturbance of the renal water output, while the extrarenal output varies in rather wide limits. A water retention would not be demonstrated definitely.

R. G. G.

Basal metabolic rate in epilepsy.—L. A. Damon. Arch. of Neurol. and Psychiat., 1932, xxviii, 120.

The metabolic rates of 300 epileptic patients were tested and about half of the cases showed abnormal rates, the number of abnormal minus readings being about twice the abnormal plus readings.

R. M. S,

In the convulsive attacks in 69 epileptics the Babinski sign was positive in 65, i.e. 94.2 per cent., after cessation of the clonic movements. In 37 (56.9 per cent.) it became positive immediately after the last convulsive movement. In 28 instances (48.1 per cent.) the sign was positive two or three minutes after cessation of the convulsion. During this lapse there was no response at all, or occasionally even a flexor response. In 17 observations (26.1 per cent.) the sign could be elicited on one side only; in the other 48 cases it was elicited on both sides. Its duration is from half a minute to 25 minutes and appears to increase with the age of the patient. Its mechanism is still in the realm of speculation. The writers suggest a transitory derangement of the pyramidal system with a consequent appearance of pyramidal tract signs of a fleeting character.

Immediately after the convulsion there is a pronounced tachycardia with a gradual decrease in pulse-rate until the recovery of the interparoxysmal rhythm in 5 to 30 minutes. The systolic pressure rises at once after the seizure in about half the cases, and in the other half it is decreased. The diastolic pressure remains normal in the cases where the systolic pressure is increased after the fit, but decreases markedly with a decrease in systolic pressure. Experimental results show a rise of blood-pressure and generally of heart rate in intact, unanaesthetized animals after intravenous injection of absinthe or camphor monobromide in sufficient doses to elicit a well-marked convulsion. Neither the increase of blood-pressure nor of heart rate is invariable. The fact that no increase of blood-pressure occurs in animals experimentally when the skeletal muscles are eliminated by the intravenous injection of curare, or when the spinal cord is transected high in the thoracic region, shows that such rises of blood-pressure as do occur in other animals are due to the action of the striated musculature during a clonic convulsion. A similar rise of blood-pressure does not occur when the convulsant is injected within a few hours after bilateral ablation of the cortical motor areas; the convulsions under these conditions are tonic. Nor does the rise of blood-pressure occur in control animals when the dose of the convulsant agent is such that a tonic and not a clonic convulsion appears. The wide variability of cardiovascular changes in the human subject during epilepsy indicates a lack of any constant or specific primary action upon the cardiovascular mechanism.

Under experimental conditions, there is usually an increase in the respiratory rate at subconvulsive doses of the convulsant. At the convulsive
level the record of respiration is obscured by clonic movements. After sublethal doses the subsidence of the convulsive movements is followed by a period of rapid respiratory movements. When the dosage reaches the value at which tonic phenomena are elicited, the respiration is slowed, and may even be intermitted during the period of greatest tonic activity. The pupils are dilated and rigid to light during both phases of the fit. At the early phase of relaxation they may be unequal and react sluggishly to light. In the second or third minute of relaxation they may become pin-point, giving paradoxical light reactions. The reaction of the pupil to light returns before the Babinski sign disappears, and before the pulse-rate and blood-pressure return to normal. Under experimental conditions, there is also dilatation and rigidity of the pupils to light, but the corneal reflex may be present at times while the pupil is still rigid. When one cervical sympathetic nerve is divided some hours before the experiment, the pupil on the same side contracts under absinthe, while the other dilates. The usual response of the pupil to convulsants is decreased when the cortical motor areas are removed a few hours before.

C. S. R.

SENSORIMOTOR NEUROLOGY.


The fundi of 114 epileptics were studied with the object of ascertaining the presence or absence of increased intracranial pressure. Of this number 58 showed a distinct pathology, while nine could not be examined. The most frequent finding was a venous engorgement of the eye fundus. This group constituted 55.1 per cent. of the total. Of these 82 cases of passive hyperæmia 18 persisted, while 14 occasionally cleared up during the interparoxysmal period. Temple Fay’s mechanical theory of epilepsy is reviewed and discussed. The work of Kalt, Claude, and others on the relation between intracranial pressure and the pressure in the retinal vessels, is also referred to. The conclusion is reached that the relatively frequent incidence of passive hyperæmia in the fundus of epileptics would tend to prove the existence of intracranial hypertension in at least a considerable number of cases.

C. S. R.