From these experiments on animals the authors believe that, normally, the pyramidal is the main motor system which is responsible for the clonic convulsions that occur when the pyramidal system is entirely or almost entirely intact. Tonic convulsions arise from the lower motor mechanisms in the period immediately succeeding an injury to the cortical motor mechanisms. After the lapse of sufficient time, clonic responses may be elicited from them. This is, however, not evidence that clonic convulsions may and do arise from the lower motor mechanisms when the cortical mechanisms are intact.

Tonic convulsions are absent in the early postoperative stages in animals in which the midbrain has been split longitudinally in the median line, while clonic convulsions persist if the pyramidal system is intact. All parts of the motor mechanism act together as one system when the brain is intact. It does not seem probable that when the whole motor system is intact, one part of the mechanism gives rise to movements of one type and some other part of the mechanism independently gives rise to movements of another type.

R. M. S.


The author discusses the evidence both pathological and physiological in favour of a centre for the regulation of sleep. On the whole he thinks himself warranted in postulating a point at the junction of the thalamus and interbrain which though not in itself a localized centre regulating sleep is the nodal point of an area of grey matter concerned with this function. If such be the case he considers it of importance not only as an aid in the localization of disease more especially of tumours, but also because he thinks it may be possible to influence such a centre by radiation or diathermy, and so treat insomnia by methods which he regards as superior to drugs, physiotherapy or psychotherapy.

R. G. G.

NEUROPATHOLOGY.


This article gives a fairly full résumé of the work on the microglia which has been done in Spain, America and Germany, and adds several facts which have been brought to light by Roumanian studies under the author's direction. The normal distribution and arrangement of the microglia in various parts of the central nervous system of man and the common experimental animals is described more completely than is usual except in the original Spanish articles of Hortega. In addition a short note is given on the microglia of some cold-blooded animals.
In this connection it is worthy of note that the developing microglial cell passes through a stage in which it contains fatty granules similar to that found by Wells and Carmichael in tissue cultures of analogous cells from the fowl. A full description is also given of the changes in the microglia in a great variety of diseases, among which we note ischaemia, rabies, herpetic and lethargic encephalitis, borna, vaccinia, septic meningitis, disseminated sclerosis, and neuroptico-myelitis. In general paralysis, in addition to the well-known formation of rod-cells, various other changes are described including the formation of giant-cells, and of rosettes which sometimes encircle a giant-cell. Changes are also seen in the microglia in poisoning by arsenic, lead, morphine, veronal and coal gas. These are rather indicated than described, as are also the alterations in such chronic degenerative diseases as senile dementia, dementia praecox, Huntington's chorea, and epilepsy. The author has also studied the microglia in a juvenile case of amaurotic family idiocy, in which he found many stages towards the formation of fat granule cells. Mention is also made of the author's recent studies of the microglia in senile plaques and in experimental replacement of the cerebrospinal fluid by Ringer's solution of varying hydrogen-ion concentration.

The article is thus more a review of recent literature than a fresh contribution to the subject, but the fact that much of the author's own previous work and that of his collaborators has been published in Roumanian, as well as the wide scope of the present article and the numerous references given, make it a useful contribution to our knowledge of the microglia.

J. G. G.


The histological changes recorded exhibited great similarity to those seen in the various types of amaurotic family idiocy. There were differences, of which the most striking were the changes in the cerebellum. Marked as the latter are in some late infantile types of amaurotic family idiocy, they were much more marked in the case recorded. The prevalence of glia tissue and blood-vessels in the various cortical layers, the extreme scarcity of Purkinje cells, the density of the fibrous glia and the abundance of neutral fat substances in the perivascular spaces of the blood vessels suggest a far advanced parenchymatous lesion with the transformation of the cerebellar cortex and optic thalamus into the so-called glial scar. One gains the impression that in this disease the pathological phenomena are much farther advanced than in ordinary amaurotic family idiocy, though the fundamental type of changes is generally the same. Here one has the same expansion of the ganglion-cell bodies with practical obliteration of the dendrites, the identical accumulation in them of prelipoids, and the same pronounced cerebellar and thalamic changes. The microchemical changes also are similar and denote that the conditions under discussion are
members of one pathological group. As Bloom well puts it, Niemann-Pick's disease appears to be "a profound disturbance of metabolism in which the infants do not thrive: there is a piling up of lipoid material, especially phosphatids, in phagocytic cells throughout the body while neutral fats are more or less gone or destroyed. In this they differ from Gaucher disease where no deep metabolic disorder is present, for such patients may live up to 50 years."

In other words, the two morbid conditions are due to a metabolic disturbance. However, one is so far hardly justified in considering Niemann-Pick's disease and amaurotic family idiocy one disease process. As Sachs justly pointed out, it is rather singular that no striking visceral changes such as are seen in Niemann-Pick's disease have been described in amaurotic family idiocy. This would be the case if they were one and the same condition. Nor do all cases of Niemann-Pick's disease exhibit cerebral changes typical of amaurotic family idiocy. However, the pathological material has not been sufficiently abundant, nor were the visceral organs in amaurotic family idiocy thoroughly studied. Until such studies are forthcoming it may be considered that the two conditions are caused by a metabolic disorder which in some instances affects preferably the central nervous system (amaurotic idiocy), in others preferably the visceral organs (Niemann-Pick's disease) and in the minority of cases, the two.

R. M. S.

[51] **Intracerebral calculi.**—R. C. Buckley. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 1203.

While intracerebral areas of calcification are known to occur in many tumours, infections and abnormalities of the brain, there are few verified examples of the formation of actual calculi or 'brain stones.' In the author's case the patient had experienced for 23 years infrequent epileptic attacks, each being preceded by muscular twitchings in the lower part of the left leg.

Roentgenograms showed two dense calcified areas 1.8 and 1 cm. respectively in diameter lying deep in the right cerebral hemisphere in the vicinity of the ventricles. They remained unchanged in size, position and appearance during the twelve-year period of observation.

Death occurred suddenly from a large haemorrhage in the left cerebellar hemisphere. Both calculi were found in the white matter of the right cerebral hemisphere near the cingulum. The larger stone was embedded in a smooth shining cavity the wall of which was made up of a dense mass of collagen fibres with an outer zone of fibrous astrocytes; the small stone was sharply marked off from the brain tissue by a dense connective-tissue wall.

As the patient showed postmortem evidence of other healed vascular accidents as well as an acute, fatal cerebellar haemorrhage, it is likely that the calculi represented the end-result of an area of degeneration or haemorrhage with consequent absorption of the necrotic material and calcium deposition.

R. M. S.
[52] The anatomical substratum of the convulsive state.—W. Spielmeyer. 
Arch. of Neurol. and Psychiat., 1930, xxiii, 869.

Pathological anatomy cannot give support to-day to the view that there is a 'genuine' epilepsy, but it does permit of conclusions being drawn as to certain functional phenomena which immediately precede the epileptic seizures, and from his study of the pathological changes which lead to the well-known sclerosis of Ammon's horn Spielmeyer concludes that vasomotor disturbances are effective in the mechanism of the epileptic attack.

In the earliest stages the nerve-cells of this region are ischaemic and surrounded by Hertega cells, while in the cerebellum very similar changes are present. It is important to note that the histological quality of the changes is not dependent on the etiological process itself, for that process does not affect the characteristic location of the lesion in Ammon's horn. On the contrary, it is the same histological picture that one finds in all the different symptomatic epilepsies with their different anatomical substrata.

Since exactly the same changes may be found in cases of cerebral arteriosclerosis, thrombosis and embolism it is obvious that organic occlusion of the circulation produces changes like those seen in epilepsy, and from this the author concludes that in epilepsy, also, an impediment to the circulation must have been present; this hindrance is first felt by those parts poorly supplied with blood, viz., Ammon's horn and the cerebellum, whereas in other parts of the brain with a better blood supply, a compensatory reaction to the disturbance is possible.

R. M. S.


There is a noteworthy tendency in present-day medicine to explain certain organic lesions on the basis of functional circulatory disturbances. In neuropathology, especially, the pathogenesis of many lesions that could not be ascribed to organic causes is now regarded as the result of functional disorders of the blood supply to the central nervous system. G. Richer has shown that slight stimulation of the vasomotor nerves results in a dilatation of the bloodvessels with acceleration of the blood stream. Stronger stimuli lead to vasoconstriction followed by dilatation of the capillaries, and when accompanied by a slowing of the circulation, prestasis with petechiae usually occurs. This state frequently develops into a stasis. If such a stasis develops rapidly, agglutination of the blood-cells may occur. A stasis is reversible and is not accompanied by transudation of serum, whereas prestasis with retardation of the blood flow, corresponding to what happens in inflammation, is associated with transudation. With exudation of serum, diapadisis of erythrocytes may also take place, and this often characterises prestasis in the environment of a necrosis,
itself caused by complete stasis. Such a combination of the necrotic effect of stasis, as in a softening, with the resulting prestasis of the surrounding tissue, leads to a red infarction. When prestasis is not followed by stasis there results an incomplete necrosis with hyperplasia of the mesodermal and glial elements in the nervous tissue.

Applying these conceptions to the pathology of the nervous system Hiller, Grinker and Meyer have shown that the characteristic areas of softening in the brain caused by carbon monoxide poisoning occur on the basis of prestasis and stasis. In other words, functional disturbances of the blood-supply lead to lesions of the nervous tissue of a severity as great as one has been accustomed to attribute only to organic circulatory disorders, as in thrombosis and embolism. This conception has been of considerable value for the explanation of lesions of the brain in various forms of eclampsia, such as occur in pregnancy and certain cases of pertussis, but whether the circulatory disturbance in pertussis eclampsia is organic, i.e., due to air emboli, or functional can be decided only by studying a case in which the convulsions have been separated a sufficient time from the period of spasmodic coughing to preclude the possibility of air emboli. The authors have been fortunate to obtain such a case and for comparison describe another case which demonstrates the effect on the brain tissue of an embolic occlusion of a main branch of the middle cerebral artery.

In the case of pertussis eclampsia no evidence of meningitis or hæmorrhage was found and the authors believe that clinically and anatomically these two possible causes of convulsions probably seldom, if ever, occur. The convulsions and the underlying cortical damage took place long enough after the cessation of coughing to preclude the possibility of an air embolism, and although no thrombosis or other organic obstruction could be found the essential pathology corresponded to the softenings typically found associated with vascular occlusions. The type of lesion varied from ganglion-cell 'Erbleichung' to incomplete and complete softening, which points to a slowly increasing diminution of the blood supply. The lesions were confined to the cortical layers and involved them in foci. The cornu Ammonis was similarly involved, but the white matter was entirely spared. This electivity is explainable by the fact that these areas have a functionally different blood-supply from the white matter and deeper structures, and are notably more vulnerable to disturbances of their nutrition. Within the softened areas the cortical blood-vessels showed tremendous dilatation, while in unaffected cortical areas their calibre was normal. This was perhaps the most striking feature and being identical with what is found in carbon monoxide poisoning, speaks strongly for stasis as the basis of the tissue damage.

In the second case an apparently total occlusion of a large branch of the middle cerebral artery had caused a fairly sharply demarcated cœdema with central necrosis of the cortical grey substance in the regions supplied by branches of the occluded vessels. Microscopically, the general appearance was that of a
partial oedema of the grey cortex on the basis of a severe ischaemia of the areas involved. By imbibition of fluid into the tissue, the ischaemia had led to a severe damage of the ectodermal elements, but with a slight proliferative reaction. A comparison of these two cases shows how distinct areas of gray cortex can be destroyed by both organic and functional disturbances of the blood-vessels. In the one a demonstrable organic ischaemia of a large pial vessel took place, with rapid and severe necrosis of the supplied tissue. In the other case, a more gradual and less complete disturbance of the nutrition due to stasis resulted.

The authors conclude with the suggestion that the etiology of many obscure transient nervous syndromes and of severe anatomical lesions of the brain without apparent organic cause may be solved by further study of the possibility of functional circulatory disturbances.

R. M. S.


Of 23 cases which were Boltz-positive, all were cases of general paralysis and all showed other syphilitic reactions. Of 25 cases which were Boltz-negative, 11 showed positive syphilitic reactions and 14 were completely negative in that respect. Taking the cases of general paralysis for which the Boltz test is supposedly specific, we find that in 34 cases 23 were positive and 11 negative. One is therefore forced to the conclusion that whereas a positive result is an additional confirmation of neurosyphilitic disease, a negative result has no particular significance. Apart from the clinical study of a case with, say, indefinite protein reactions, weak Wassermann or only slightly raised cell-count, the author would be more influenced by the result of the gold-sol test than by either a positive or negative Boltz reaction. C. S. R.

SENSORIMOTOR NEUROLOGY.


A man of 34 fell ill with a meningitis of uncertain nature. The lumbar puncture fluid was characterized by a marked pleocytosis, in which there was at first a polymorphonuclear predominance, but later a lymphocytosis. The patient’s temperature fell at the end of a week and the symptoms abated. Weakness of the arms and legs gradually developed, however, about a week later. The tendon jerks became sluggish and the plantar responses were extensor in type. There was retention of urine. Lipiodol was held up between the bodies of the fifth and sixth cervical vertebrae. The cerebrospinal fluid, withdrawn by the