resection of the cervical sympathetic. Normal posture, gait, and tonus were then repeatedly examined for a month or more. The animals were subsequently decerebrated. At no time was any difference as between fore and hind legs discoverable. Decerebrate rigidity developed simultaneously in the two fore legs, and was equally maintained.

The conclusion is drawn that tonus, as a component of total skeletal muscular function, is in no direct way dependent on an intact sympathetic innervation of muscle. Thus the literature records are almost overwhelmingly in disagreement with Hunter and Royle.

J. V.

NEUROPATHOLOGY.


1. Rats fed on a diet deficient in both vitamin $B_1$ and $B_2$ exhibit only slight chromatolytic changes in the ganglion-cells of the cord.

2. Rats fed on a diet deficient in vitamin $B_1$ exhibit the same changes but to a greater extent for the same duration of life. In addition, when symptoms of paralysis are of long standing, early degeneration may be found in the myelin of the peripheral nerves.

3. The histological changes found in the nervous system of rats fed on a diet lacking in vitamin $B_2$ consist in swelling and vacuolation of the anterior horn cells of the spinal cord with the deposition in them of lipochrome pigment, a noticeable increase in the surrounding satellite cells, and an increase in the number of $\pi$-granules in the peripheral nerves.

These findings bear some resemblance to the lesions of the nervous system in human pellagra, while skin lesions found in the same series of rats resemble very closely those of the same disease of man.

J. V.


The experiments brought forward fully confirm the findings of Boas that when dried egg-white is fed to young rats as the sole source of protein there results a syndrome characterised by nervous and cutaneous symptoms. The fact that the same disease may be produced by feeding the mothers on dried egg-white as the sole source of protein suggests that in the drying process some essential dietetic factor is destroyed in the egg-white, rather than that a toxic substance is formed. If the existence of a hitherto unknown dietetic factor is postulated, there is a close analogy with the known occurrence of the deficiency diseases beriberi and scurvy in infants fed at the breast.
Investigation of the pathological changes occurring in young rats fed on dried egg-white shows that very characteristic changes occur in the central nervous system, which still further emphasises the similarities of the condition in the rat to Swift’s disease in man. These may be thus briefly summarised:—

1. Both diseases occur in young animals. Swift’s disease has never been recorded in adults, while in the adult rat it is only after from three to five months that cutaneous lesions can be produced.

2. The diseases may occur on a diet of mother’s milk or a ration containing an ample supply of the known vitamins.

3. The clinical symptoms of both are nutritional, nervous, and cutaneous.

4. In rats there is a characteristic ‘kangaroo’ position, in children a knee-elbow position.

5. In rats and children there is a curious mousy odour.

6. Death is often due to an intercurrent bronchopneumonia.

7. There is no evidence that the diseases are due to an infection with bacteria or ultramicroscopic viruses.

8. The pathological changes in the skin and nervous system are similar.

Although the possible formation of a toxic substance during the drying of egg-white cannot be excluded the experimental evidence strongly suggests that for the normal nutrition of both young and adult rats a factor is required in addition to the known vitamins. If Swift’s disease in man is identical with this new syndrome in the rat then this new factor must also play a part in human nutrition.

AUTHORS’ ABSTRACT.


The mechanical theory of epilepsy has had many exponents in one form or another. Among the firm believers that some extracerebral factor was concerned, Kocher can be mentioned. He looked upon increased intracranial pressure as perhaps the cause. Alexander favoured increase in fluid over the cortex. Dandy has insisted on the existence of some organic basis for the attacks. Elsberg and Pike considered the importance of increased intracranial pressure and suggested that decrease in intracranial pressure consequent upon the diminution of fluids taken might explain the relief seen in starvation treatment. Foerster considers pressure and increased fluid as a factor associated with irritation from arterial pulsation. Mixter suggests the probable underlying cause is due to failure of subarachnoid fluid absorption. Lind believes cerebral anaemia from compression to be the cause of seizures in the presence of intracranial pressure. Swift believes that constriction due to distortion of the large venous outlets (sigmoid and lateral sinuses) may produce
in patients diarrhoea the injection of the in related proportion in the Pacchionian bodies so far studied in 150 brains; (2) the fact that prolonged dehydration may be carried out on certain patients for a period of a year without deleterious effects to their general health and that in the presence of this controlled dehydration this small group of patients suffering from generalized convulsive seizures with loss of consciousness have become attack-free and have remained so, as long as the fluid-intake restrictions established for them have been maintained. Those suffering from slight attacks or petit mal have not been benefited.

C. S. R.


Eighty-seven rabbits were used for experiments undertaken with the object of inquiring to what extent factors which are known to modify convulsions in patients have a similar effect on convulsions experimentally induced in animals. Two convulsants were used intravenously: thujone, made from oil of wormwood, and homocamphor, a preparation of camphor.

It was found that the incidence and severity of the reactions did not increase in proportion to increase in the amounts injected. When the convulsant dose was repeated within an hour, the resulting convulsion was less frequent and severe than the first; the larger the dose used, the greater was the difference between the first and second reactions. Preliminary medication with phenolbarbital sodium caused reduction in convulsions only when the dose, based on weight, far exceeded that used clinically. Reduction in severity exceeded reduction in incidence. Rabbits which were made to fast did not show a reduction in the carbon dioxide combining power of the blood. Those which were fed on butter and olive oil showed reduction, presumably because of the diarrhoea induced. In both groups of animals there was some reduction in the frequency and severity if the convulsant drug was injected intravenously, but not if into the heart. Of the rabbits’ reactions, the severity was not related to the concentration of bicarbonate in the blood. Following the injection of amounts of lactic acid sufficient to reduce the bicarbonate greatly, the animals did not have convulsions. Injections of acetone into the heart
did not consistently reduce the bicarbonate in the blood or the severity of the attacks. The injection of sodium bicarbonate did not have a definite effect on the induced convulsion.

The use of a convulsant drug is such a powerful agency that only profound changes in body metabolism will modify the resulting convulsions. Therefore, it is hardly a suitable means for studying factors which might affect the occurrence of seizures in patients. R. M. S.


An account is given of five cases of hydrocephalus in infants with the findings at autopsy. In two instances a complete obliteration of the subarachnoid channels at their point of origin from the cisternæ was found; further, no evidence of an old inflammatory condition was present. The author therefore contends that the hydrocephalus was due to a congenital defect in the opening up of the sub-arachnoid channels. In stressing this argument he takes the presence of other congenital anomalies in the second case as favourable evidence. In a third instance hydrocephalus was of later origin and at post-mortem no inflammatory changes were found; he supposes that only a partial opening up of the subarachnoid spaces had occurred. The other two cases had definite etiological factors such as meningococcal meningitis and trauma. Nothing new has been added to the knowledge of hydrocephalus in this paper and the interest rests in the confirmation of the work of Dandy and Weed.

E. A. C.


Among the more common causal antecedents of cerebral hæmorrhage must be reckoned thrombotic softenings of the brain. In some cases, reported by Globus and Strauss, there was pathological evidence that hemorrhages developed in areas of the brain which had previously been the seat of thrombotic necrobiosis. The present writers record two cases in which clinical evidence of such a sequence was also present and was confirmed by pathological examination of the brain. They do not suggest that this sequence is constant but rather that it is more common than has often been supposed.

The mechanism of the process is scarcely discussed by the authors, but they support the modern view that cerebral hemorrhages are due not to miliary aneurysms but rather to dissecting aneurysms, and it is probable that in the formation of these some part is played by complete or incomplete thrombosis.

J. G. G.

This article is not only a critical survey of recent investigations of senile plaques by means of new silver staining methods, but also gives a detailed study of those found in two brains, one that of a woman of 90 years, the other from a patient aged 45 who had suffered from polyneuritis and mental disturbances. The author insists on the great variety in the appearance of senile plaques. Scarcely two can be found which exactly resemble one another, and the variations in the relative preponderance of cells of neuroglial and microglial origin which take part in their formation are still more striking. In the younger forms the microglia is predominant. In later forms large neuroglial spider cells are seen to surround the plaque. But neither of these cell types plays more than a purely secondary role in its formation. This is directly due to a local metabolic disturbance in the nervous tissue itself, whereby certain lipoid substances which have a special affinity for silver are precipitated out from the lipo-protein complex of the nervous parenchyma. The presence of these substances stimulates the phagocytic activities of the microglial cells which migrate into the centre of the plaque, and thereafter often themselves undergo degeneration with the formation of an amyloid substance. In this way the central nucleus of the plaque is constituted. The microglial cells also proliferate round the plaque; their processes swell and are often found to contain fatty granules. At a later stage the fibrous neuroglial cells hypertrophy, surrounding it with thick firm processes.

The author also discusses the vexed question whether the nerve fibres found within the plaques represent the degenerated remains of pre-existing fibres or are new fibres which have passed into it since its formation. He finds that the evidence strongly supports the view that there is an actual new formation of nerve fibres round it, and that some of the new fibres attempt to penetrate into it, but there, finding conditions unsuited to their further passage, they become arrested.

Several technical methods specially adapted for demonstrating the details of the plaques are described in this article, which should be consulted in the original by all who are interested in the subject.

J. G. G.


The authors report the case of a microcephalic idiot who on X-ray examination showed extraordinary calcification in the choroid plexuses. There were no positive neurological signs and nothing could be found in the general metabolism to indicate the mechanism which favours abnormal deposits of either calcium or bone in the choroid plexuses.

R. M. S.