as an argument in favour of the theory of 'pathoclisis' or 'tendency to become diseased' which was enunciated by C. and O. Vogt in 1922. In fact the paper is chiefly valuable in giving a brief summary of this theory, which in its fully developed form is so complex as to be almost incomprehensible.

The paper is illustrated by large-scale photographs of the cortex which are rarely met with except in papers from the Vogts' laboratory in Berlin. In the present instance the degree of enlargement is not sufficient to demonstrate the types of cell present, and the photographs do little more than indicate the zones of greatest cell destruction.

J. G. G.


The case on which this paper is based was one of syphilitic Parkinsonism, beginning at the age of 61, and associated with slight dementia. The cerebrospinal fluid was positive to the four classical tests (globulin, cell-count, colloidal and Wassermann reactions). Antisyphilitic treatment however produced no effect. Post-mortem examination of the brain showed numerous senile plaques in the cortex, and a generalised arteriosclerotic condition, probably syphilitic in nature. Perivascular infiltration was very slight in the cortex and rather better marked in the basal ganglia where there was also loss of many of the larger cells and particularly intense changes in the vessel walls. An unusual feature of the case was the presence of nodules of polymorphonuclear cells in the cortex, in the subjacent white matter, and in the red nucleus of the right side. In spite of the absence of spirochaetes in these nodules the authors consider them to have been syphilitic.

This combination of chronic arteriosclerotic with acute inflammatory lesions led the authors to discuss the question of the relationship of cerebral, especially mental, symptoms to the syphilitic lesions found in the cerebrospinal fluid or in the brain. This is a big question and the authors do little more than open it by referring to a series of cases of mental disease in syphilitic subjects with 'positive' cerebrospinal fluids. Obviously a large quantity of carefully examined post-mortem material would be needed if any certain light were to be thrown on this subject.

J. G. G.

SENSORIMOTOR NEUROLOGY.


From a clinical standpoint cases of myoclonus may be divided in two groups: (1) Myoclono-epileptic cases and (2) myoclonic cases without epilepsy. Those of the first group showed pronounced deterioration and symptoms of extra-
pyramidal type, muscular rigidity, mask-like face, hypersalivation; while those of the second group did not show deterioration, remaining in relatively good mental condition until the end, and did not develop symptoms of extrapyramidal type. In the majority of the reported cases with or without epilepsy, symptoms of cerebellar type were present, being mostly of dyssynergic character, and often showing characteristic skeletal deformities (kyphoscoliosis, club foot). From the standpoint of localization of the pathological lesion, it was found that in cases of myoclonus without epilepsy the lesion affected the dentate nucleus invariably, and in some cases involved the inferior olives as well, but not constantly. Other parts of the central nervous system, and in particular the cortex and basal ganglia, were not involved. In cases of myoclonus with epilepsy, the lesion showed a predilection for the dentate system, being usually more pronounced in the nucleus dentatus but, at the same time, it was more diffuse and showed a severe involvement of basal ganglia and midbrain. The authors are inclined to infer that the myoclonic component in the myoclono-epileptic cases is due to a cerebellar- or dentato-tropic lesion, while the epileptic component (deterioration) has for its pathological basis a lesion affecting subcortical grey matter.

C. S. R.

[12] The interparoxysmal respiratory rate of epileptics.—S. M. Weingrow


The observations of the author which are none too numerous persuade him that the respiratory rate of epileptics is lower than that of normals. This rate is not affected by drugs which are being taken by the patient.

R. G. G.


The case described corresponds more or less exactly to the syndrome described by Foerster in 1909, of hypotonia in a child when lying down and hypertonia when supported in a vertical position, associated with defects of speech and mentality. Choreiform movements of the face and tongue and restlessness of the limbs are common accompaniments of the syndrome which may be congenital. Foerster found the anatomical basis for this syndrome to be a cerebellar sclerosis, whereas in Vogt’s case the lesions were in the frontal lobes. In the present case the symptoms began at the age of four years with a febrile illness thought to be malaria. This was followed by a series of convulsions, after which a condition of diffuse hypertonia with trismus set in. This gave place after ten days to the condition in which the patient was found, which persisted till within a few days of death two years later. He could not speak, but uttered a monotonous cry when hurt. He could not feed himself, but masticated food
put in his mouth. Continuous lateral movements of the head were present on lying down and slow forward and backward swaying of the body on sitting up. When lying down the limbs could be hyperextended and hyperflexed, and appeared completely toneless. He could however stand with very slight steadying support, and in this position the limbs were extremely rigid and remained so if he were lifted by the armpits. Along with this hypertonus were athetotic movements especially of the left arm and slow spastic grimacing movements of the mouth and face. The tendon reflexes were active and the plantars flexor in type.

A post-mortem examination revealed an extreme fatty degeneration of the liver, sclerosis of the putamen and caudate nuclei, areas of degeneration in the cortex with thickening of the overlying meninges and some loss of Purkinje cells in the cerebellum. In addition a demyelination of the pyramidal tracts was found which was much greater in the lumbar than in the cervical region of the cord and was not seen in the brainstem or internal capsule. The authors comment on the points of similarity between this case and some cases of hepato-lenticular degeneration, but confess that the syndrome of Foerster appears in other cases to have a totally different anatomical basis.

J. G. G.


The earliest symptom in cases of tumour of the fourth ventricle is headache, which is often intense and paroxysmal. The pain radiates to the neck and frequently to the shoulders and arms. A stiffness of the trapezius and other muscles of the neck is characteristic, and the patient often holds the head hyperflexed and frequently tilted. At a later stage there appear symptoms of general hypertension and also focal manifestations. The former differ from the hydrocephalic symptoms with other types of cerebral tumour, only in their intensity, their abrupt onset, and their rapid evolution. Focal symptoms include disorders of equilibrium, reeling gait, retropulsion and an attitude of hyperextension. Cerebellar asynergy may be present, and, less often, intention tremor, hypermetria, and adiadochokinesia. Disorders of speech and of writing are usually absent. Tonic attacks may occur, in which the body assumes an opisthotonotic attitude, the upper limbs being hyperextended, pronated and adducted. Trismus, irregularity of the heart-beat and Cheyne-Stokes respiration may be noted. These tonic attacks may be precipitated by emotion, sudden noise, or by alterations in posture. In some instances the tonic crises may be more or less unilateral in appearance, thus recalling a torsion dystonia. As the signs progress the tendon jerks gradually disappear. Rare later symp-
terms include spontaneous tremors and various visceral pains. During the terminal phase, bulbar attacks may occur, characterized by tachycardia with irregularity, alternate flushing and pallor of the face, dyspnœa, polyuria and sweating. Sudden death may result.

M. C.


This is a comprehensive and long review of all the nervous and mental complications of pregnancy described in the literature. These include almost all the conditions familiar to neurologists and can hardly be abstracted. Several illustrations are given and the article must prove a useful reference for those interested in the subject.

R. G. G.

**PROGNOSIS AND TREATMENT.**


From a series of clinical investigations and physiological experiments the author reaches the following, among other, conclusions.

1. The neck reflexes of Magnus and de Kleijn have no place in the clinical picture of Parkinsonism.

2. Righting reflexes in choreic patients are of normal type.

3. In choreo-athetotic states with the rigidity of Little’s disease righting reflexes are wanting or assume a primitive character. In them, further, neck reflexes are seen, but not in cases of chorea.

4. Posture and movement reactions in extrapyramidal conditions show the same imperfections as voluntary movements in respect of inhibition or de-inhibition, slowing, spread of associated movements, and tonization of certain attitudes.

5. Parkinsonism can be produced in animals by acute bulbocapnine toxicosis, but only in restricted dosage. With larger amounts hyperkinesis and epilepsy appear.

6. The corpus striatum does not itself innervate movements, but as a motor centre works in close relation with the centres for voluntary movement, for posture and movement reactions, and with the motor ‘Haushaltszentrum’ (hypothalamus).

S. A. K. W.