that there is a specific nervous strain of spirochætes for paresis. They feel with Kraepelin that alcohol and other toxic and infectious agents do have a contributing predisposing influence in the production of paresis, and they think it possible that trauma favours a decadence of the nervous system in syphilitics. Orton found two types of lesions in the blood-vessels. One was stationary and the other showed a chronic progressive inflammation. The stationary lesions were those of healed syphilitic endarteritis with thickening of connective tissue between the intact endothelium and the elastica. He believes that these lesions are due to the varying degrees of syphilitic arteritis during the early stages of the infection, and are not characteristic of the paretic process in the vessels. This latter process consists in an active inflammatory change characterised by a lymphocytic and plasma-cell exudate in the adventitia. These processes were found also in many extra-cerebral vessels. Orton considers that these findings are sufficient to prove the hypothesis of the invasion of the brain by way of the peri-arterial lymph-spaces. The writer concludes from the pathological findings noted above and those of Adolph Meyer, Frazier and others, along with his own clinical observations, that any injury to the small blood-vessels which will allow the spirochæte to enter the brain-tissue will immediately favour the institution of the paretic process. This injury may be an actual trauma with hæmorrhage, or it may be an infective or toxic process which injures the intact endothelium and favours the entrance of the spirochæte into the adventitia and the perivascular lymph-spaces and thus into the brain tissue. Trauma of the brain may, by causing vascular injury or brain destruction, be followed later by gliosis and nerve-cell sclerosis, allowing first the spirochætal invasion and later adding to the gliosis and sclerosis of nerve-cells which are also an integral part of the paretic brain pathology. The writer thinks it possible that an excess of the adrenal content of the blood and a hypersecretion of the thyroid gland caused by emotion may produce an increased permeability of the blood-vessels or an actual damage to their structure, and so favour the passage of the spirochæte. Account should also be taken of the changes caused in the nerve-cells by fatigue and emotion (Crile).

C. W. FORSYTH.

*Encephalitis Lethargica.* (*State Hosp. Quart., November*, 1920.) *Montgomery, W. H., and Waldo, L. T.*

The apparent chronological relationship to influenza led to the conclusion that encephalitis lethargica was probably a sequel to influenza. Wittich and Sennert noted a comatose variety of influenza as early as 1580. In 1712 there appeared in Tübingen the first outbreak of sleeping-sickness definitely associated with influenza historically. Following the epidemic of 1889–90 in Northern Italy a condition called "nona" appeared, accompanied by fever, delirium, and coma. This did not extend to other European countries or America, in contradistinction to the wide-spread distribution of the influenza epidemic it followed. The present epidemic of encephalitis lethargica had its beginning near Vienna in the winter of 1916. Early in 1918 it was recognised in Western Europe, and about a year later had arrived in America. This history shows why encephalitis lethargica was at first

considered probably a sequel to influenza. On the contrary, Flexner pointed out in 1916 that, when the first cases of encephalitis were recognised in Austria, the influenza epidemic had not made its appearance, also that the location of the epidemic of nona coincides approximately with that of the first cases of the present epidemic, and suggests that encephalitis may be endemic in that section of south-eastern Europe contiguous to the boundaries of Austria, Italy and Switzerland, awaiting only favouring conditions to become epidemic. Such conditions were produced by the war. The pandemic of influenza prepared the soil by lowering resistance. Influenza is thus a separate infection and predisposing only in its relation to encephalitis lethargica.

The pathological changes are found mostly in the basal ganglia, midbrain, and bulb, but cerebrum and cerebellum may show changes. Congestion, œdema, and round-celled infiltration of pia-arachnoid are found. Those cases with delirium are most likely to show cortical changes. There are also deep perivascular infiltrations with small hæmorrhages; these account for the focal signs.

The ætiology is still undetermined. Attempts have been made to infect animals by inoculating with the affected nerve-tissues and with a minute filtrable organism from the naso-pharynx. Some are stated to have been successful (Strauss, Loewe, Hirsfield); others have been failures. Some writers have attempted to identify it with poliomyelitis ætiologically. The disease is infectious and mildly communicable, most probably through the mucous discharges of nose, mouth, and throat. At first in America cases were missed and diagnosed as tubercular meningitis, Bell's palsy, or other disease.

The disease varies in intensity. Some cases are very mild and clear up in a few days. It has the appearance of a general infection. Nausea, headache, high temperature (102° or 103° F.), unaltered temperature, raw tongue, oral inflammation, diarrhœa, constipation, retention of urine are mentioned as symptoms. Rapid loss of flesh and weakness and prostration follow. Pain in the arms and legs often occurs early. There is a general hypersensitiveness of the whole body, and patients may cry out with pain when moved. Pain at the back of the neck increased by pressure is common. Kernig's sign is generally absent, as is Babinski's. Deep reflexes are likely to be hyperactive.

The somnolence appears early, and usually is not deep enough to prevent the patient being roused. Patients may "come to" and easily reach a high level of consciousness. Mild cases get up, walk about, and try to shake off the lethargy, while some cases lapse into a stupor from which they cannot be roused. The stupor may closely resemble a deep sleep. There are present quite constantly jerky movements involving the arms and legs. Focal signs are constant in greater or less degree. The most striking feature may be oculomotor palsy. Strabismus is generally internal. Bilateral incomplete ptosis, facial palsy (which may be bilateral, giving a mask-like countenance), unequal pupils, oscillation of pupils and nystagmus may be present. There are also paræsthesias, hyperæsthesias, neuralgias, and other sensory disturbances. Involvement of the vagus may cause heart and respiratory disturbances, and these are of grave import.

*Mental symptoms.*—Delirious features predominate. There is a

lowering of mental tension. Either a quiet or restless talkative somnolence is usual. The mental content has rather a superficial character, one patient showing an occupation delirium, another a fear reaction.

Prognosis is always grave, if mild abortive cases be excepted. Of three cases reported only one recovered. The duration may extend over weeks or months.

Treatment is symptomatic, having in mind the disease is a general infection. Nourishment must be given freely. This and careful nursing are important. Encourage elimination. Sodium bicarbonate with plenty of water is advised. Urotropin is recommended, but is contra-indicated by albuminuria and any kind of kidney irritation. For the relief of restlessness and delirium, veronal in small doses proved surprisingly efficient. W. J. A. ERSKINE.

*Mental Forms of Epidemic Encephalitis* [*Formes mentales de l'encephalite épidemique*]. (*L'Encéphale, August*, 1920.) *Bremer, M.*

The author narrates 4 cases presenting syndromes of acute delirium (2 cases), confusion (1 case), and mania in a child, æt. 9.

(i) In the first case, a man, æt. 34, the clinical signs were the following: hallucinatory delirium with extreme motor and verbal agitation, insomnia, and fever. The bacillary antecedents of the patient, his rapid wasting, the marked lymphocytosis of the cerebro-spinal fluid made one think, in spite of the absence of clear meningeal symptoms, of a prolonged bacillæmia, while it appeared on questioning the family that he had been sharply attacked by a diplopia, of which no traces remained. The fever and delirium diminished very slowly. Coldness of the legs persisted. A month and a half later myoclonic twitchings of great amplitude developed, affecting the abdomen and the right leg, and at the same time an extreme hyperæsthesia, superficial and deep, of the outside of the left leg and the dorsal aspect of the foot. These persisted. There still exist an accommodative asthenopia and a slow optic neuritis. On the contrary there is no psychical sequel.

(ii) A woman, æt. 47, attacked suddenly by terrifying hallucinations, of the absurdity of which it was possible to convince her and of which she preserves a curiously precise memory. She showed delirium, fever, acetonæmia, diplopia, and myoclonic twitchings. She became normal in mind, but had a myoclonic condition three months after the malady.

(iii) A man, æt. 26, in a state of stupor. There was a past history of insomnia in 1912, shell-shock in 1915, and confusion after an air raid in 1916. After several weeks he complained of insomnia and diplopia. On March 14th he got lost in Paris, was taken home by the police and did not recognise his mother; aspect dull, head hung; when questioned answers only after a long interval; gives his age and knows where he is, but nothing more can be got out of him. There exist diplopia and myoclonic twitchings in the neck and left half of the body. Two months later there was only a certain slowness in response and a want of equilibrium in the external and internal ocular muscles.

(iv) A boy, æt. 9, entered hospital in February last presenting then