Wash the specimen for from 12 to 24 hours in running water and place in distilled water for one hour, changing the water three times during the hour.

Then submerge the sections for two minutes, at from 60° to 65°C., in Mulligan’s solution of phenol, covering with at least 2½ inches of solution. The formula for Mulligan’s solution is as follows: 4 per cent. phenol crystals, 0.5 per cent. copper sulphate crystals, and 0.125 per cent. concentrated hydrochloric acid dissolved in distilled water.

Place the sections in a large volume of cold tap water for one minute and then in a 1 per cent. solution of ferric chloride in distilled water for two minutes and wash them in running water for five minutes. Place them in a 1 per cent. solution of potassium ferrocyanide in distilled water until the grey matter is a brilliant blue (this should not take longer than three minutes) and wash them in running water for 24 hours. Finally, preserve them in 70 per cent. alcohol.

R. M. S.

NEUROPATHOLOGY


The results of this investigation dealing with the rhythmic diurnal and nocturnal changes in blood chemistry and water balance indicate that the quality of these changes may be correlated with the clinical observation of increased susceptibility to seizures during sleep, particularly after midnight. There is evidence of a shift of the blood and urinary titre toward the alkaline side during the early morning hours, as well as rising intracranial pressure and brain volume during sleep. In addition, the cholesterol content of the blood has been shown to fall consistently during sleep, reaching minimal values at 3 a.m. Each of these changes has been considered by others to be definitely related to the precipitation of seizures in susceptible individuals. The acid-base, water-shifting, and ionic blood changes in these experiments are qualitatively of the type to encourage the development of seizures during the latter half of the period of sleep. The rhythmic changes are observed to occur in the normal as well as in the epileptic subjects, and may be interpreted as the normal rhythm for blood chemistry.

C. S. R.

[99] Permeability of the meninges in progressive paralysis to different häemolytic amboceptors normal and complementary to blood serum (Permeabilita delle menigi nella paralisi progressiva a vari amboceptors emolitici normali ed al complemento del siero di sangue).—M. PIOLTI. Riv. di pat. nerv. e ment., 1935, 44, 296.

The following results of experiments seem to emerge. The concentrations in human blood of häemolytic amboceptors against sheep, horses, guineapigs,
rabbits and pigs vary in their proportions and quantity considerably from individual to individual. In progressive paralysis the passage of anti-sheep amboceptors from blood to cerebrospinal fluid constantly happens in the proportions—maximum 1-15, and minimum 1-125. The other amboceptors pass through in varying proportions, but the phenomenon is not constant and is regulated by the selective power of meningeal function. Also a concentration of the respective amboceptors in the blood has some importance, but much less than the meningeal selectivity in determining their passage from blood to cerebrospinal fluid. There is no record of the passage of horse, guineapig, rabbit or pig amboceptors in any other affection of the nervous system. It has not been discovered if this is pathognomonic as is the sheep amboceptor in parenchymatous syphilis of the nervous system even in respect of cerebro-meningeal syphilis.

The results confirm the observation that in progressive paralysis a certain amount of complement is always present in the cerebrospinal fluid. It would seem that complement filtered through the meningeal barrier probably undergoes a chemical transformation which changes its value in respect of various hemolytic sera. This phenomenon also can perhaps be explained by the theory of the selectivity of meningeal function on the ground that this does not permit the filtration in equal measure of single components of the complement. Further experiments showed that the introduction of a hypertonic sodium chloride solution in the blood does not increase the meningeal permeability in normal individuals, but mildly hypertonic solutions introduced into the arachnoid cavity produce gross alterations of this permeability. This might be of therapeutic value. In schizophrenia, meningeal permeability is generally lessened, but seems to correspond with a clinical form of the affection. Further investigation of this might prove of importance in prognosis and pathogenesis.

R. G. G.

[100] Possible relation of lead intoxication to multiple sclerosis.—B. Boshes. 
Arch. of Neurol. and Psychiat., 1935, 34, 994.

Specimens of cerebrospinal fluid from 28 patients were analyzed for lead by the Fairhall hexanitrite method.

In one of 16 cases of multiple sclerosis the fluid showed a positive result. The patient had been given sodium iodide, and the urine also showed lead. Of 12 other cases of various conditions, in only one, a case of lead intoxication, were abundant crystals found in the cerebrospinal fluid. In this case there was 0-2 mg. of lead per litre of urine.

Taking all three studies into consideration, there is no adequate proof for, and ample evidence against, the theory that lead is an aetiological agent in cases of multiple sclerosis.

R. M. S.

The histological examination of this case afforded a clear confirmation of the genesis of these conditions from embryological faults. The glioma had originated in a plaque of tuberous sclerosis in which neuroglial alteration was predominant and primary participation of ganglion-cells in the process is perhaps doubtful. The case seems to corroborate the theory of spongiosblastosis. However in typical cases there are malformations of ganglion-cells which cannot be altogether attributed to secondary alteration. Also in the present case in which the principal malformation was neuroglial unusual forms of nerve-cells were found.

R. G. G.

[102] Cerebral complications of putrid pleuro-pulmonary suppuration.—Ira COHEN. Arch. of Neurol. and Psychiat., 1934, 32, 174.

The author reviews 19 cases which presented cerebral symptoms complicating pleuropulmonary suppuration. These are arbitrarily divided into three groups: (1) abscess of the brain; (2) aseptic embolus; and (3) psychosis. It is suggested that a single aetiological factor is present in all groups, namely, an embolus. In the first group the embolus is infected, and in the second group aseptic; in the third group it is postulated as a factor in the psychosis.

R. M. S.


The author has found the syndrome of acromegaly often associated with a tumour in the diencephalon and considers that acromegaly cannot be explained entirely on the basis of a tumour of the hypophysis. He thinks that two factors enter into the production of the syndrome, one hypophyseal and the other diencephalic.

R. G. G.


Enormous enlargement of the bowel was detected at autopsy in four cases of encephalitis lethargica which had been treated by atropine. A similar
enlargement was observed clinically in seven patients suffering from chronic encephalitis treated by atropine for a prolonged period. A disturbance in tone of the musculature of the bowel appeared to be the cause of the enlargement.

M.


Thompson and Cushing repeatedly injected in dogs the gonadotropic hormone of the pituitary body. Among other results glycogen was stored up in the liver and in the muscles. Starting from these observations several human cases of so-called 'glycogen-storage disease' were examined for signs of endocrine disorder, especially of the pituitary body. The clinical picture in these cases proved very similar to the symptoms observed in animals by Thompson and Cushing. In consequence of these observations the condition characterized by a storage of glycogen (hepatomegalia glycogenetica) is assumed to be the result of disordered internal secretion, especially of the pituitary body. It was not possible to decide if disordered carbohydrate metabolism in cases of diminished pituitary function is always accompanied by defective absorption of glycogen.

M.


The syndrome usually associated with syringomyelia may result not only from cavity-formation but also from other lesions such as luetic meningomyelitis and thrombosis of the anterior spinal artery. There is little evidence that syringomyelia cavities are due to necrosis in a glial tumour, the cavity being surrounded by a glial scar rather than by tumour cells. In most cases cavities in the spinal cord are dependent on abnormalities of circulation. War cases show that cavities may be produced by concussion with no direct injury of the cord by the missile. The authors were able to produce cavities similar to those found in syringomyelia by interfering with the arterial supply.

R. G. G.

SENSORIMOTOR NEUROLOGY

[107] Hemiplegia with the leg in flexion.—Orthello R. Langworthy, Elmer Higberger, and Ruth Foster. Arch. of Neurol. and Psychiat., 1935, 34, 520.

Patients with injury of the pyramidal tract producing hemiplegia show abnormalities of tone that usually conform to a stereotyped pattern: the