no effect, but fluids which the Wassermann reaction was positive caused an increase of the flow. This finding may depend on the presence of break-down products of the brain in the syphilitic fluid.

Clinical adaptation of some of this work is foreshadowed. J. P. M.


The burden of evidence of many workers suggests that the intraocular and cerebrospinal fluids are of the nature of dialyzates in simple membrane equilibrium with the plasma. The rate of dialysis depends on the balance between hydrostatic capillary pressure and the osmotic pressure of the plasma proteins; so that given a constant composition of plasma the filtration rate will vary directly with the capillary blood pressure.

Ludwig, and later, Leonard Hill and others have shown that capillary pressure depends far more on venous than on arterial blood pressure, and hence both the formation and absorption rates of cerebrospinal fluid and of the aqueous humour are governed by the venous blood pressure. In an elaborate series of experiments these arguments are tested and the authors conclude that although the intraocular and intracranial pressures are not directly interdependent, changes in hydrostatic or osmotic blood pressure produce parallel pressure changes in the eye and in the cranium.

C. J. E. C.

**NEUROPATHOLOGY.**


After referring to methods already in use, the author offers the following technique of his own as giving excellent results:

1. Fix pieces in pyridine 5 c.c., acetone 5 c.c., formol 15 c.c., ammonium bromide 3 gr., distilled water 75 c.c.
2. Cut frozen sections of about 15 μ thick.
3. Rapid washing in distilled water.
4. Immersion for five minutes or longer in a solution consisting of strong ammonia 10 drops, glycerine 4 c.c., distilled water 16 c.c.
5. Without washing, immerse in a 2 per cent. solution of silver nitrate, for about one minute, moving the section meanwhile.
6. Without washing, place in a 1-2 per cent. solution of formol for about 3-5 minutes.
7. After rapid washing, fix in an alcoholic solution of sodium hyposulphite.
8. Dehydrate in 95 per cent. alcohol, phenol-xylol, balsam.

The addition of 2-3 drops of gum arabic (5 per cent.) either to the nitrate bath or the reducing solution (for every 15 c.c. of the solution) improves the picture. J. V.
Method of demonstration of *Spirochaeta pallida* in single microscopic sections.—R. R. DIETERLE. *Arch. Neurol. and Psychiat.*, 1927, xviii, 73.

The author has evolved a method of staining *Spirochaeta pallida* in sections, which on account of its reliability, simplicity and time-saving features will be welcomed by laboratory workers.

1. After fixation and cutting, place the preparations in a 1 per cent. solution of uranium nitrate in 70 per cent. alcohol at 55° C. for one half hour.
2. Wash for a moment in distilled water.
3. Pass the sections through 96 per cent. alcohol.
4. Handling the sections individually, place them in an absolute-alcoholic solution of gum mastic (10 per cent.) long enough to allow thorough infiltration—about thirty seconds. Immerse sections for an instant in 96 per cent. alcohol.
5. Transfer them to distilled water.
6. Silver for from one to six hours at 55° C. in 1 per cent. aqueous silver nitrate solution, carrying out this procedure in the dark.
7. Wash for a moment in water.
8. Develop in the following reducing solution for from five to fifteen minutes:

   Hydrochinone . . . . . 1.5 gm.
   Sodium sulphite . . . . 0.25 gm.
   Neutral formaldehyde . . . 10.0 c.c.
   Acetone . . . . . 10.0 c.c.
   Pyridine . . . . . 10.0 c.c.
   Water, to make . . . . 90.0 c.c.

Mix and dissolve these and then add 10 c.c. of absolute-alcoholic mastic solution to make the mixture milky.
9. Wash for a moment in distilled water.
10. Dissolve out the mastic and dehydrate by transferring the sections to 96 per cent. alcohol, and then to acetone. Clear in xylol and mount in canada balsam.

The distilled water must be free from chlorides and all glassware must be likewise clean. Frozen and celloidin sections are handled with glass hooks and cover glasses with ordinary forceps. The method is also applicable to paraffin sections.

R. M. S.

The Cajal and Hortega glia staining methods.—J. H. GLOBUS. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 263.

Sections obtained from brains or spinal cords which have been kept for months or years in formalin can be successfully stained by any of the methods based on the Cajal gold chloride-sublimate principle after a preliminary saturation of the tissue with ammonium bromide.
The technique is as follows:

1. Prepare frozen sections of formaldehyde-fixed material at a thickness of from 15 to 30 microns.
2. Wash quickly in several changes of distilled water.
3. Place in a ten per cent. solution of strong ammonia water for twenty-four hours at room temperature, or for shorter periods in an incubator.
4. Carry rapidly through two changes of distilled water.
5. Place in a ten per cent. solution of pure hydrobromic acid, and let it remain there for from two to four hours.
6. Wash quickly in two changes of distilled water, to which a few drops of ammonia water is added.
7. Treat in accordance with the method selected. If it should be necessary to delay the final staining for some time, sections may be put into a 2 per cent. aqueous solution of ammonium bromide to which a few drops of neutralised formaldehyde have been added.

R. M. S.


The xanthoprotein and indican content was estimated in 230 sera and 270 spinal fluids, and in some cases the results obtained were correlated with the amount of other nitrogen-containing substances present.

The normal xanthoprotein content of the fluid was found to be less than ten units. The indican test (Hans) was normally negative in the fluid.

In meningitis, there is usually an increase of xanthoprotein in the spinal fluid, depending on increased permeability of the meninges.

In cases of contracted kidney (even without uremic symptoms) xanthoprotein may pass in increased quantities into the fluid. With uræmia there is nearly always an increase in the amount in the fluid, but this is not the cause of the uremic symptoms.

J. P. M.


In this peculiar case, that of a woman of 53, autopsy revealed a small tumour (size of a cherry) in the extreme left occipital region. Apparently a metastatic carcinoma (from the thorax), it was the solitary neoplastic lesion of the whole brain, yet it had given rise to no symptoms by itself. The clinical picture was one of right-sided cerebellar disease, with slight pyramidal symptoms on the same side. But apart from the small tumour, the whole of the left cerebral hemisphere was in a state of marked acute swelling or œdema (well seen in the...
photographs accompanying the article), and so was the mesencephalon. This latter region also exhibited numerous small block extravasations by diapedesis. Thus, as the author remarks, his case is one of pseudotumor cerebri occurring in tumor cerebri. Not one of the clinical symptoms could be attributed to the neoplasm directly, and the problem remains, to explain the development of local edema in two specific areas. It was the edema that had caused all the clinical symptoms. The supposition is that the swelling of the left hemisphere had produced the changes in the mesencephalon by pressure on the vessel system of the latter, and it is suggested that, as compared with other vascular areas, the mesencephalic is one that is unduly vulnerable. The swelling of the left hemisphere as a whole must somehow be related to the presence of a tumour within it, but little is stated as to the mechanism of its production.

J. V.


For the purpose of these investigations an examination was made of some ten human cases of varied cerebral lesions, both involving and separate from the basal ganglia, and reference is made further to three experimental animals (one dog and two cats).

The author considers himself justified in stating, contrary to the views of various workers, that cortical connexions with the basal ganglia are more definite than some of his predecessors have found. He thinks that there is a connexion between the cortex and the caudate which is definite but slight ("wenn auch schwach"). In lesions of the hemispheres he considers a slight outfall of the finest myelinated fibres of the caudate occurs.

As for the putamen, in lesions of the temporal lobe and region of the insula a slight degeneration is found in its lateral division. The globus pallidus is stated to degenerate in its posterior part from lesions of the temporal lobe; cortical lesions in the parietal region are followed by a fine degeneration in the inner and outer laminae of the globus pallidus and in its ground-substance and ganglion cells.

In regard to the corpus Luysii and substantia nigra, a cortical connexion for the former is still doubtful, and for the latter similarly. On the other hand, degeneration of the stratum intermedium is said to follow degeneration in the cerebral peduncle, which is not the case with the substantia nigra.

It is difficult to represent in illustrations the presence of fine degenerations, and those given with the paper are not particularly clear. Further, questions of interpretation of histopathological changes require to be investigated with particular care if the cases selected are such as to render the occurrence of more than single lesions possible. The experimental evidence adduced is quantitatively insufficient.

S. A. K. W.

Changes in the central nervous system in Recklinghausen’s disease can be regarded as falling into two groups: (1) developmental anomalies or dysgeneses, and (2) over-production of cell-elements of glial origin. Included among the former are changes in the vicinity of the central canal, sometimes amounting to a syringomyelia, heterotopias of white in grey substance and vice versa (spinal cord, cerebellum), nests or whorls of entangled myelinated fibres in the cerebral cortex (plaques fibromlyeliniques). The latter take the form of blastoma-formations, either as neuro-epithelial tumours or as neurinomata.

The possibility of the occurrence of cases in which the changes are more or less confined to the neuraxis, peripheral nerve changes being minimal or absent, has been mooted by several workers, and a case apparently belonging to such a class is here described with much detail. The patient was a girl of 22, whose clinical symptoms were those of a nearly complete transverse lesion at the level of the lower cervical enlargement, commencing with a kind of Brown-Séquard syndrome. In addition, numerous brown pigmented flecks were visible on the skin in various areas.

Pathologically, a tumour was found within the spinal cord at the level mentioned, and it turned out to be a neurinoma. While no recognisable peripheral fibroneuromata were found, microscopical examination showed the presence of collections of cells in the cutaneous pigmented areas, suggestive of early new-formations. A long account is provided of the actual histological findings, with fine plates, and a discussion of the bearing of the case on the nature of Recklinghausen’s disease is given. Briefly, the spinal tumour consisted of spindle-shaped nuclei of varying size, arranged in rows in many places though by no means everywhere, and embedded in a ground-substance staining orange with van Gieson’s stain. Changes in this ground-substance had led here and there to sponge-like or cystic formations; at other spots it exhibited hyaline degeneration. Nuclei and fibres in some places tended to a whorl-arrangement. Numerous branching and intertwisting nerve-fibrils were found, almost all unmyelinated. They could be demonstrated to be encytial, that is, to run in the plasma of the tumour cells. Ganglion-cells were also found, either from the anterior cells, enclosed and degenerated, or else partaking of the nature of a new-formation (ganglioneuroma). Further, the main elements in the tumour were undoubtedly of gliogenic derivation. Thus its histology was closely allied to that of the neurinoma of peripheral nerves.

S. A. K. W.
Studies of metabolism in epilepsy. II. The sugar content of the blood. III. The blood sugar curve.—W. G. Lennox.

Psychiat., 1927, xviii, 383 and 395.

Under the above title the author publishes two papers on sugar in epilepsy.

The first is concerned with a study of 509 estimations of the fasting sugar level in 267 cases. The Folin-Wu method for venous blood was used and samples were collected as a rule before breakfast. The observations were made in every conceivable circumstance with relation to the seizures and included estimations made before, during and after a simulated fit in a healthy man. After review of the literature and a full discussion with special reference to any possible connection between epileptic convulsions and those of insulin hypoglycaemia, the author concludes that there is no abnormality in the fasting blood sugar level of his patients and no direct relation between the blood sugar level and the convulsive attacks. Increase in blood sugar during the attack depends, he thinks upon the available glucose in the body.

The second paper deals with the sugar tolerance curves of 140 cases. Out of this number 24 per cent. showed curves abnormally high, 6 per cent. curves abnormally low and 70 per cent. within normal limits. In several patients there was a considerable variation both in shape and in level between successive curves. Possible reasons for this are mentioned and a further paper dealing with similar variations in normal persons is promised.

The author considers that his results fail to show any abnormality of carbohydrate metabolism which actually induces seizures. Such variations as he observed are taken to be rather a reflection of an underlying "instability of physiologic function" as to whose nature he can give no clue.

C. J. C. E.

SENSORIMOTOR NEUROLOGY.


Professor Pette at Nonne's clinic has seen during the past year a number of cases with symptoms of encephalomyelitis which are difficult to classify. The onset is acute or subacute: the symptoms are often vague and can seldom be referred to a circumscribed focus: there is a tendency to great recession of the clinical manifestations. Among 25 cases seen in the last two years there were only two deaths.

These two patients died in the acute stage: both clinically and pathologically the cases were very similar. In both the disease began subacutely with rapidly progressive spastic paresis in the extremities, quickly followed by cranial nerve disturbances and, in one case, bulbar symptoms: in both cases...