Abstracts.

Neurology.

NEUROPATHOLOGY.


The following is a résumé of the alterations in the nervous system in a case diagnosed as hereditary cerebellar ataxy with retinitis pigmentosa, but found to correspond with the juvenile type of amaurotic family idiocy.

A.—*The Nerve-cell.*—1. Marked distention of the cell-body and of its processes; spherical or ovoid outline; displacement of nucleus towards the apical extremity of the cell.

2. The cell-body is filled with granules, some powdery, some coarse, staining blue with Nile-blue and yellowish-red with Sudan; sections stained with silver show a fine honeycomb-like network, which is quite distinct from the neurofibrillar network. The rest of the cell-body preserves its normal structure as demonstrated by Nissl’s method, and the neurofibrils appear normal with Bielschowsky’s stain.

B.—*Neuroglia.*—Proliferation, especially in the region of the swollen neurons. The cytoplasm of the neuroglia cells contains yellowish granules.

C.—*Vessels.*—The lining cells of the capillaries are often laden with granules stained red with Sudan; similar granules are found in dementia praecox, chronic chorea, and senile dementia.

**Regional Changes:**

1. *Cerebrum.*—All the cells without exception show the above changes, the Betz cells particularly. The frontal lobes are most affected.

2. *Cerebellum.*—Purkinje cells are very much altered, and many have disappeared altogether.

3. *Cord.*—Similar changes in the cells, the nerve-fibres remaining intact.

4. *Retina.*—(a) Infiltration of the different layers with pigmented cells derived from the choroid. The infiltrated zone extends as far as the ganglion-cell layer, but never reaches the nerve-fibre layer. (b) Disappearance of many of the ganglion cells; those remaining are grouped in fives and sixes.

The nature of the granules is of considerable interest; from their chemical and staining reactions they appear to be of the nature of phosphatides or cerebrosides, derived probably from breakdown of complex lipo-proteins of the interfibrillary cytoplasm. The swelling of the cell is due to the hydrophilic action of phosphatides and cerebrosides within
it. The lipid material in the capillaries is evidence of the fate of the products of degeneration, as is the presence of pigment granules in the neuroglia cells.

E. O'Flynn.


The case described is that of a woman of 54, who for one year had suffered from headaches, and attacks of vertigo and vomiting. Her sight had failed, and she had had occasional periods of diplopia and of tinnitus. She had, in spite of these symptoms, continued her ordinary work until ten days before admission to hospital, when she suddenly developed double facial paralysis and dysarthria. Her previous symptoms at the same time became more acute, and she became progressively weak in her limbs. On admission her sight was poor, but she could count fingers at three yards, and see the hands of a watch at a foot; the fundus oculi was normal in both eyes, and the pupils reacted well. There was complete paralysis of the left, and incomplete of the right, internal rectus muscle, and complete paralysis of both external recti. Upward and downward movements of the eyeballs were retained. There was facial paralysis on both sides, greater on the left, without any fibrillation of the facial muscles. The Wassermann reaction in the blood was weakly positive. Lumbar puncture gave a fluid containing 26 cells per c.mm. and 0·2 per cent albumin. The patient became gradually weaker, and was for the most part in a state of low delirium; she died less than two months after admission to hospital.

At the autopsy the arachnoid was noticed to be somewhat thickened over cerebellum, medulla, and pons, and the oculomotor nerves where they entered the bony foramina were surrounded with an oedematous cone derived from this membrane. On section of the brain the walls of the ventricles and the iter of Sylvius were seen to be lined by a thick, soft, reddish-grey membrane, pieces of which had broken off and floated in the ventricular fluid. In thickness it varied from 3 to 10 mm., and covered the walls of the whole of the ventricular system, with the exception of the tips of the descending horns of the lateral ventricles. The left lateral ventricle was dilated; the right lateral ventricle was incompletely, and the third and fourth ventricles were completely, filled with tumour. On the floor of the fourth ventricle was a rounded tumour mass of the size of a small cherry which protruded slightly into the iter of Sylvius. This neoplastic lining not only protruded into the ventricles, but in some places, as over the caudate nucleus and optic thalamus, and on the outer surface of the left posterior horn, invaded the brain tissue to a small distance. The choroid plexuses were normal, and were not adherent to the tumour.

The microscopic appearances of the tumour were those of a glioma, formed of small round cells and larger multinucleated cells, with a fine fibrillar ground substance, and in addition there were many larger cells with a clear vesicular nucleus, closely resembling pyramidal nerve-cells. Numerous mitotic figures were seen, a rare finding in gliomata. The
ependyma lining the ventricles could only be seen in a few places, where it lay superficial to the tumour. In many places the adventitial spaces of the vessels, as they ran into the brain tissue, were infiltrated with tumour-cells. The authors consider the tumour to be a neuroblastoma, containing cells both of glial- and nerve-cell types, both in rudimentary forms. They comment on the rarity of diffuse tumours lining the walls of the ventricles, of which they have only found six cases in the literature.

J. G. GREENFIELD.


The authors describe under the name ‘subacute leuco-encephalitis’ a pathological condition hitherto unrecognized, characterized by inflammatory foci in the centrum ovale, invading the basal ganglia to some extent, but leaving the cortex altogether intact.

The case is that of a young soldier of 19, who, after a slight fever, began in August, 1915, to have progressive weakness of his legs, which increased until, in October, he was unable to stand up. At this time he was also found to have inco-ordination and dysdiadochokinesis of the upper limbs. The deep reflexes were absent, and the plantar reflexes flexor. The cerebrospinal fluid was normal, and remained so throughout. After slight improvement he got rapidly worse, and in December, 1915, he became bedridden, stuporose, and incontinent, with contractures of all the limbs. Thereafter he improved considerably, so that by February, 1916, he could walk. In April he again had a rise of temperature, and again lost the use of his limbs. He also became very dysarthric in speech. Again improvement set in, and by May he could walk with some support. In June his vision became affected, he was very inco-ordinate in his movements, and showed a considerable degree of apraxia, especially with the left hand. The deep reflexes were now increased, with Babinski’s sign on both sides. No papilloedema was found. In October he developed ideas of persecution and auditory hallucinations, but could still walk with help. At this period he showed no motor or sensory aphasia, but had gross apraxia in the left hand. There was, however, no inco-ordination in the movement of either hand. In January, 1917, he became again stuporose, taking no heed of his surroundings, but in answering questions he gave no evidence of disorientation. He died on Jan. 10, 1917.

At the autopsy the only pathological changes, apart from some fatty degeneration of the liver, were found in the centrum ovale and basal ganglia of the brain. These were congested, especially in their posterior part, but showed no haemorrhages or areas of softening. Microscopically the greatest degree of abnormality was found in the white matter of the centrum ovale, corpus callosum, and internal capsule. The lenticular and caudate nuclei showed similar though less severe changes, and the optic thalamus was slightly affected. The rest of the brain, including the whole of the cortex and brain stem, was absolutely normal. The changes in the centrum ovale consisted of great dilatation of vessels, some of which were
thrombosed while others had small haemorrhages surrounding them. Their perivascular sheaths were filled with small cells of lymphocyte type. There were numerous patches of myelin destruction, with corresponding damage to the axis cylinders, and in some of these there was very intense overgrowth of neuropilial tissue, giving rise to many giant forms of glial cells.

The authors draw attention to the obviously inflammatory nature of these changes, and deduce from them, and the clinical history of remissions, that the disease was due to some microbial agent. But as they were unable to undertake any injections into animals, the etiology s for the present purely conjectural.

J. G. Greenfield.


The authors claim that this is the first case of lipodystrophia progressiva followed to autopsy. The patient, a girl of 13 years, died as a result of a prolonged septicemia which caused considerable emaciation in the parts of the body unaffected by the disease. In spite of this, a moderate amount of fat was present in the gluteal regions, orbits, omentum, about the kidneys, heart, and pericardium, and under the serous membranes. There was no evidence of tuberculosis. The thyroid gland was enlarged and very rich in colloid material. The thymus gland was very scanty. No other special abnormality was found in the endocrine glands, though the suprarenal cortex was considered to contain less lipid than usual. Microscopic examination of a piece of anterior abdominal wall and of scalp from the occipital region showed complete or almost complete absence of fat-cells in the subcutaneous tissue. There was no evidence of inflammatory fibrosis. Little importance is to be attached to any of these changes, as the excess of colloid in the thyroid gland was less than often occurs at puberty, and changes in the suprarenal might be due to the prolonged infection.

J. G. Greenfield.


The patients studied, in number 101, were cases of resistant infection which had still a persistently positive Wassermann reaction after more than twelve intravenous injections of arsphenamine and interval mercurialization. The average dosage was fourteen intravenous injections and ninety injections with mercury. Of the whole number, only 4 had come to notice because of definite secondary syphilitic lesions; the rest were Wassermann-positive cases with latent skin, visceral, and osseous manifestations. The tests consisted in the use of single Noguchi antigen with rabbit-human haemolytic system, with active serum and incubator fixation. This conservative method the writers calculated would give certain positives with the multiple antigen and cold-fixation modifications, so that negatives would be detected with greater certainty by the method used than by the others.
ABSTRACTS

An analysis of the persistently positive cases gave the following: Cardiovascular syphilis, 44 per cent; neurosyphilis, 30 per cent; osseous lesions, 30 per cent; visceral lesions, 21 per cent; other types, 10 to 17 per cent. Of the neurosyphilities, 40 per cent were cases of paresis and 50 per cent of tabes. Fifty per cent of the neurosyphilities had also cardiovascular syphilis. Patients with cutaneous syphilis showed the familiar immunity from neurosyphilis, and vice versa.

There was no evidence that a Wassermann-fast condition resulted from infection with any special strain of spirochaete; in fact, the multiplicity of the involvement pointed rather in the opposite direction.

Of these resistant cases, 84 per cent have undergone symptomatic arrest to-day as a result of treatment. The cases of paresis and tabes with gastric crises account for more than half of the failures.

In the discussion on treatment the writers urge that, while it is desirable that the positive Wassermann be reversed, this object should not be the primary object of the therapy. Symptomatic arrest within the bounds of tolerance is the most important point. As regards prognosis, a persistently positive serum Wassermann test seems to accompany a grave, rather than a trivial, syphilis. The cardiovascular and nervous systems should be most carefully examined in all Wassermann-fast cases.

J. LE F. B.


This article is based on the systematic examination of 310 cases of general paralysis and of taboparesis, 84 cases of tabes, and 72 cases of cerebrospinal syphilis. In each case the 'four reactions' were investigated, viz., the Wassermann test in the blood, and, in the fluid, the Wassermann test, the cell-count, and the globulin test.

General Paralysis and Taboparesis.—In 90 per cent of the 310 cases all four reactions were positive, and in no single one of them were all the reactions simultaneously negative. Only in 1 case was a pleocytosis wanting, and only in 3 was the globulin test negative. In 15 cases of the series the Wassermann test was negative in the fluid. Incompleteness of the four tests is suggestive either of an early stage of general paralysis, or of some interference as the result of treatment with salvarsan, although it is possible that these two factors may not explain every case where one or other of the four tests is negative.

Tabes.—In the tabetic series all four reactions were positive in about 70 per cent of the cases, and all four were negative in 3½ per cent. The Wassermann reaction in the blood was positive in 85 per cent, and in the fluid in 77 per cent. In about 10 per cent no pleocytosis was found, and in a slightly smaller percentage no increase of globulin. The explanation of the negativity of all four reactions in a few cases is to be assigned either to an early stage of the disease or to the result of treatment by salvarsan.
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*Cerebrospinal Syphilis.*—In roughly one-half of the cases of the author's series all four reactions were positive; in two-thirds of the cases the Wassermann reaction was positive in the blood, and in about 80 per cent the positive Wassermann test in the blood, the pleocytosis, and the positive globulin reaction occurred in a quite independent way. There can be no doubt that in this group the result of salvarsan treatment in reducing the positive states of the tests is much more marked than in either of the two former groups.

A more difficult point to determine is whether, from the tests, general paralysis and tabes on the one hand can be separated from meningitic, gummatous, or vascular neurosyphilis on the other. From the author's figures it is clear that the tests are not of much assistance in this respect, except in so far as the conclusion is justified that, if the reactions improve rapidly under salvarsan treatment, the condition is likely to be one of cerebrospinal syphilis. In tabes, and still more in general paralysis, the pathological state of which the tests are the expression proves much more refractory even to persistent treatment.

S. A. K. W.

SENSORIMOTOR NEUROLOGY.


In well-established hemiplegia the crossed plantar response is usually bilaterally flexor, though it may be extensor in a few cases. In cases of myelitis and transient intermittent paralysis there may be no plantar reflex obtainable on the paretic side, but the crossed reflex may be extensor, showing that serious change may take place in the pyramidal system and be confirmed by the subsequent development of paralysis. To produce the crossed reflex the stimulus must be strong, whether applied by the methods of Babinski, Oppenheim, or Gordon.

R. G. Gordon.


The author describes a sign which he claims has not been noted previously, and which consists of a small point of tenderness just to the left of the spinal column, corresponding to the 5th dorsal interspace, or about that level. It is always found on the same side as the stomach, and may be an area covering more than one space. The tips of the thumbs or fingers are stroked across the areas in question, and a positive sign is elicited if the patient winces or complains of discomfort. The sign persists for a few days before and after a gastric attack, and in the intervals the tenderness subsides. The origin of gastric crises is shortly discussed, the author suggesting that "the 'central' (cerebral) origin heretofore largely assumed" is unlikely to be the true origin of the attacks. The above statement is unexpected, as since the days of Charcot the spinal origin of lightning pains and other 'crises' has held the field to the exclusion of all others in medical opinion. Treatment by counter-irritation over the hyperalgesic