Abstracts.

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ETIOLOGY.


The author has analyzed no less than 130 cases of juvenile tabes or taboparalysis, personal and from the literature, so that the documentary value of the paper is considerable. Of these 130 cases, 49 were in patients whose parents (one or both) had suffered from metalutic nervous disease. Since, however, in 37 no information as to the parents was forthcoming, the proportion is in reality 49 out of 93. As regards these 49 cases, in 39 the parents (one or both) were tabetics themselves, while 10 (one or both) were general paralytics. The female sex supplied 32 and the male 16 examples of the affection; in one case the sex was not stated; of the whole number of 130 cases, 74 belonged to the female sex. In addition to the marked hereditary element a large proportion of cases of juvenile tabes is characterized by a constitutional inferiority, seen more particularly in the persistence of stigmata of infantilism. To this the author attributes a pathogenic significance, but these characteristics may perhaps partake more of the nature of effect than of cause.

S. A. K. W.

PATHOLOGICAL ANATOMY.


Professor Marinesco gives us in this paper a minute and painstaking study of the nervous system from a case of amaurotic family idiocy which presented all the usual clinical features.

The results obtained by the methods of Nissl and of Bielschowsky and Cajal were in every way similar to what many others have reported, viz., swelling of the cell bodies and dendrites, disappearance of the intracellular fibrils except round the periphery, and so on. Special attention is drawn to the fact that the nuclei of the cells were relatively unaltered. Of greater novelty are his findings obtained by the methods of Perls for
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iron, Best for glycogen, Ciaecio and Dietrich for lipoids, Benda and Regaud for the minute protoplasmic granules known as mitochondria (chondriosomes, neurosomes), and the indophenol-blue method for oxydases.

Lipoid granules were found everywhere in the nerve-cells of the cortex, cerebellum, and spinal cord, in varying degree; by contrast, few were seen in the cytoplasm of the neuroglial cells. An intimate connection existed between the severity of the cell lesions and the amount of glycogen deposit therein; glycogen granules were massed in the most diseased cells and occasionally in their nuclei, also in their dendrites, and to a less extent in glial cells and processes. The quantity of oxydases was in inverse proportion to the amount of lipoids; where achromatosis was revealed by Nissl's method there also was poverty or absence of oxydases, but in the immediate vicinity of the nucleus, in the dendrites, and also round the nerve-cells, ferments were present in abundance. Generally speaking, the superficial layers of the cortex were poorer in this respect than the deeper. A similar relation between oxydases and lipoids was found in the Purkinje cells, and in the spinal ganglia and cord. The topography of the reaction for iron corresponds to that of the chromatic substance of Nissl; so that where achromatosis was noted the reaction for iron was negative. As for the mitochondria, a marked diminution in their quantity was noted in all the cells of the cortex, in the cell-bodies, dendrites, and sacellar dilatations, and they were more irregularly distributed than in normal preparations. Apparently none were to be seen in glial cells or their prolongations.

The physical and chemical phenomena of the disease are doubtless intimately related. The swelling of the cell-body, etc., is due to increase of intracellular osmotic tension; colloid particles disappear and lipid granules take their place. In comparison with the cytoplasmic changes, the relative conservation of the nucleus is of capital importance. The anatomical basis of the processes of heredity has usually been taken to be the nuclear chromatin, but its intactness in cases of amaurotic family idiocy militates against this conception; in this disease the histological changes are the expression of disturbance of function of intracellular ferments, and it is the cytoplasm which transmits the pathological peculiarities of the affection. Marinesco's conclusion is that the familial element is dependent on the diastasic activity of the mitochondria of the cytoplasm.

S. A. K. W.


Diffuse cerebral sclerosis, so-called, has always been a somewhat unsatisfactory nosological conception. From the histopathological viewpoint it may be regarded as the terminal stage of either inflammatory or degenerative processes in the white matter of the cerebrum; and in cases of diffuse glioma analogous histological changes may be found. In all of these the peculiar localized outfall of myelin sheaths is to be observed which is characteristic of the condition. A special form of inflammation, leading to
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diffuse brain sclerosis, is that involving the white matter of the cerebral hemispheres and known as encephalitis periaxialis diffusa (Schilder) or diffuse infiltrative encephalomyelitis (Jacob). It occurs usually in young people, and its clinical manifestations are remarkably variable. Pathologically it is distinguished by the appearance of larger or smaller areas of disease in the subcortical substance, with loss of myelin sheaths and of axis-cylinders; fatty changes; overgrowth of mesodermal tissues and development of thick glial network, sometimes with large glial cells, and secondary degeneration. Macroscopically the diseased areas are commonly yellow-grey in colour and of variable consistency. The etiology of the disease is still obscure, but it presents very close resemblances to disseminated sclerosis.

S. A. K. W.


The first case was that of a man of 60, who had had a 'stroke' with loss of consciousness. The ocular defect was as follows: In both left upper quadrants, excluding the macular and paramacular areas, loss of vision; in these, as in the whole of the left half fields, complete achromatopsia; in the right fields, complete blindness for colours except in a small sector in the lower quadrants extending from the macular area out to about 30°. In brief, the case was one of bilateral hemianopia.

The second case was that of a man of 62, who also had had a 'stroke' on the left side, and whose ocular condition was the following: Right field—a small central scotoma for white and colours, absolute loss of colour vision in the whole of the rest of the field, except that in a small area below and outside the macular field red and blue 5 cm. objects were occasionally recognized. Left field—no scotoma, occasional recognition of colours in one quarter of the macular field (right lower), extending out only about 4°; in the whole of the rest of the field of the left eye, complete achromatopsia. This case, similarly, was one of bilateral hemianopia.

Pathologically, there was found in the first case an area of softening of the right lower calcarine cortex, beginning some 2 cm. from the occipital pole and extending forward to the anterior fourth of the gyrus; on the left side, a somewhat smaller area of softening was found in the fusiform gyrus, beginning about 1 cm. from the occipital pole and extending to about 2 cm. from the junction of the calcarine with the parieto-occipital fissures. In the second case there were diffuse lesions of the white matter underlying both visual areas. A very painstaking microscopical examination of cells and fibres in both cases was undertaken, and a large number of beautiful photographic illustrations are given in the paper. The details are so numerous that they cannot well be given in abstract, and for them the original should be consulted. Suffice it to indicate here some of the author’s conclusions.

The cases support the view that the lower calcarine cortex represents the upper visual fields, i.e. the lower retina, and that the macula is repre-
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sented posteriorly towards and in the occipital poles. They furnish strong evidence for the hypothesis that disturbance of colour vision depends on defect of conduction of the white matter of the calcarine region generally, and that the degree of the latter is parallel to the degree of the former. The law of 'vertical projection' holds good for colour as well as for white. Further, the cases suggest that it is the outer cell layers of the visual cortex that are concerned with the appreciation of colour. There is no evidence of the existence of a special tract or path among the optic radiations which stands for the conduction of the impulses underlying the colour sense.

No worker at this difficult subject should neglect to study this important communication.

S. A. K. W.


Under the above title the authors publish full details, both clinical and histological, of the following cases:—

Case 1 was that of a girl, age 16, who died after seventeen days' illness which began suddenly with intense headache, high fever, and ophthalmoplegia. The temperature fell on the third day, and thereafter remained irregular, with a brief ante-mortem rise. After the onset she had seemed to be recovering, but at the end of the first week she complained of numbness in the legs and difficulty in micturition. The paraesthesia spread upward to the trunk, and were accompanied by weakness, and on the twelfth day of the disease, when a detailed examination was first made, the arms also were involved. At this time her mental condition was quite clear. She could make no movements except of the upper extremities (which were weak) and the head and neck. The deep reflexes were abolished in the lower limbs, the abdominal reflexes absent, and the plantar responses doubtfully flexor. There was some loss of sensibility up to the nipple line, and burns caused by hot bottles on the abdomen had not been felt. The bladder was distended, requiring the catheter. The patient complained of intense headache. The cerebrospinal fluid contained 0.03 albumin and 20 leucocytes per c.mm. The main features of the microscopic examination, which are clearly illustrated in the paper by drawings, may be summarized as follows. The whole cerebrospinal axis was peppered with minute inflammatory foci, which were entirely confined to the white matter and were most intense in the mid-dorsal region. The lesions consisted of perivascular small-celled infiltration, degeneration of the axis cylinders, and increase of glia cells.

Case 2 was that of a woman, age 35, who died after an illness of three weeks' duration which took the form of an ascending flaccid paralysis. Histological examination revealed acute inflammatory changes of the type commonly found in acute anterior poliomyelitis. These were confined to the anterior horn cells in the cord and to the grey matter of the brain stem and basal ganglia. Two small zones of degeneration were apparent in the posterior columns.
Case 8 was that of a girl, age 14, the subject of bony tuberculosis. The illness commenced with paralysis of the lower limbs, which extended upwards so that on the fourth day the condition was one of flaccid quadriplegia with abolition of all reflexes, and in addition paralysis of the palate and loss of sphincter control. Intense vullovaginitis and bronchopneumonia were also noted during life. The disease terminated fatally in six days. Histological research demonstrated lesions which were necrotic rather than inflammatory, occupying the anterior horns in the spinal cord. These masses of grey matter appeared to be distended in some places by an exudate from which cellular elements were singularly lacking.

In their discussion of these cases, the authors make it clear that their conception of Landry’s disease is more generous than that commonly accepted in this country. They would include under this heading every case of ascending paralysis, distinguishing first those cases of this condition which are due to an acute peripheral neuritis, and subdividing the cases in which the spinal cord is affected into poliomyelitis, leucomyelitis (the white matter being chiefly involved), and diffuse ascending myelitis. They point out the striking discrepancies between the histological appearances of the three cases reported, and conclude that it remains for the bacteriologist to establish a more rational classification in this group of diseases.

C. P. Symonds.

CLINICAL PATHOLOGY.


This paper aims at correlating the serum and spinal-fluid findings in a large number of cases with the clinical signs and symptoms of the patient. It must never be forgotten that syphilis is a constitutional disease, and signs of neurological and visceral involvement may be discovered if looked for in cases which come up for quite other manifestations of the disease. The authors group as secondary all cases presenting themselves for treatment within two years of the original infection, and as tertiary all others. Of 243 secondary cases 64, and of 821 tertiary cases 480 had abnormal findings in the spinal fluid. These results showed a higher proportion of positives than normal because they included several cases of obvious neurosyphilis specially sent to the clinic. Elaborate statistical analyses of these figures are given. The cases seem to fall into groups:—

1. Mild reactions with no complaints. These yield to treatment, and later give negative results.

2. Strong reactions with indeterminate complaints of headache, giddiness, fatigue, lack of concentration, pains in the limbs, and slight neurological signs such as pupillary changes or inequalities in reflexes. These often resist treatment.

3. Malignant type simulating meningitis and often terminating fatally. These require very small doses of antisyphilitic remedies until the spinal canal has been drained several times to relieve pressure.