Abstracts.

Neurology.

NEUROPATHOLOGY.


The cortex in general paralysis of the insane has been the object of much more research than subjacent areas of the central nervous system, but in that disease there are numerous clinical symptoms—ataxia, dysarthria, tremor, etc.—for which no cortical pathogenesis is probable. Rossi has found, in the medulla of a number of typical cases of the disease, primary neuroglial overgrowth, of varying degree, round the upper end of the central canal, under the floor of the fourth ventricle, and round the nuclei of origin of the lower cranial nerves; in particular, throughout the inferior and accessory olives a marked amount of gliosis with hypertrophy of astrocytes is to be noted. Rossi emphasizes the clinical significance both of the sub-ependymal and the olivary gliosis, sometimes amounting to actual nodular glioma-formation, from the point of view of the symptomatology of the disease; the presence of this process entails a functional disturbance and eventually a degenerative atrophy of bulbar mechanisms: in fact, there is a bulbar ataxia as a consequence of impairment of olivo-cerebellar fibres, the significance of which can be better understood as a result of Rossi's investigations in a field hitherto largely neglected.

Wilson.


Twelve cases of dementia praecox were studied by the author. No case without a clear praecox history was dealt with, and the autopsies were usually held within an hour after death. Owing to the irregularity of the changes found in the nervous system in cases of dementia praecox it is worth while recording the findings of this researcher. She concludes that in the twelve cases regular and uniform pathological changes had occurred, which were due neither to arteriosclerosis, to senility, nor to a long-continued grave toxic process. The pathological process is essentially a chronic one, resulting in atrophy of the nerve-cell body and its nucleus and disappearance of the stainable substance, and an atrophy with distortion of the protoplasmic prolongations, the process terminating in extreme
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pyknotic atrophy or in fragmentation of the nerve-cell. Considered from the regional point of view, the organic changes observed were generally found to be most severe in the frontal regions, while of the two hemispheres the changes in the right one were usually much more recent and acute than those in the left. As regards nerve-cell strata, the first, second, and third nerve-cell layers showed the most severe involvement, the severity and diffuseness of the changes decreasing towards the third stratum. The author gains a general impression from her work that the initial pathological process is one of moderate swelling of both cell-body and nucleus, followed by a gradual breaking down of the normal nuclear chromatic structure, and later by atrophy and fragmentation of the neurofibrils. She believes that the changes she describes are pathognomonic of dementia praecox.

MAURICE NICOLL.


The case described is that of a married woman who, at the age of 40, began to have headaches, which became more severe as the disease progressed. She gradually lost her memory, and progressively became demented, disoriented, and showed stereotyped conversation and echolalia.

Physical examination revealed irregular and unequal pupils, which reacted slowly to light and failed to react to accommodation. Her reflexes were exaggerated, with ankle-clonus on the right side, but no Babinski sign was present. There was tremor of the hands, tongue, and lips. So far the case resembled progressive general paralysis, but the Wassermann reaction in the blood and cerebrospinal fluid was negative. The spinal fluid, however, showed 96 cells (per c.mm.). The patient died eight years after the onset of symptoms.

At the autopsy all the arteries supplying the brain were very sclerotic. The brain was shrunken, with widened sulci, and an excess of subarachnoid fluid. The atrophy of the gyri was almost symmetrical on the two sides, corresponding to the regions of supply of the middle and posterior cerebral arteries, but some areas supplied by the middle cerebral, such as the island of Reil, and the opercular portion of the left hemisphere, were intact. Over the areas of greatest atrophy the cortex showed a moth-eaten appearance, and on section no distinction could be made out between grey and white matter.

Microscopic examination showed two main types or stages of change in the atrophic parts of the brain: (1) At the point of greatest shrinkage of the cortex were seen wedge-shaped scarred areas, formed of spider-cells and granular corpuscles, along with overgrowth of the small vessels, and complete disappearance of the nerve-cells and their processes. (2) Side by side with these were cystic areas, where the section had a sponge-like appearance. These were made up of glial cells and fibres, the meshes of which were distended with fluid. In these areas also there were many granular corpuscles, and the blood-vessels were increased in number and showed ‘packet formation’. Areas showing such changes were present on
the cortex of the cerebellum as well as of the cerebrum, and in both cases they extended right up to the pial surface. In places a communication could be made out between the cystic spaces and the subarachnoid space. The ganglion-cells in the neighbourhood of the atrophied areas were in various stages of degeneration.

The author compares this condition to the 'spongijöser Rindenschwund' of Fischer and the 'état vermonuI' of Pierre Marie, to both of which it is closely allied.

J. G. Greenfield.


The patient was a man, age 36, obviously retarded in development from early years, who was regarded, briefly, as an epileptic idiot. He showed various of the cutaneous formations associated with tuberose sclerosis. Voluntary movements were slow and awkward, a variable degree of hypertonus was present in the limbs, and a form of pseudoflexibilitas cerea. Sometimes, further, the hands assumed the attitude of paralysis agitans, with the terminal phalanges, if anything, over-extended.

At autopsy, the diagnosis of tuberose sclerosis was fully established. In addition to the characteristic changes, which need not be particularized, pathological glia-overgrowth was found in the cortex in gyri which appeared normal macroscopically; and in the caudate nucleus minute 'tumours' were present, of the usual tuberose structure. In the corpus striatum, further, numbers of giant ganglion-cells were observed, and of 'monster' glia-cells, as well as undifferentiated cells (spongioblasts). Whereas in the cortical lesions these elements were more or less collected together, in the corpus striatum they were scattered diffusely. In putamen and nucleus caudatus, finally, calcareous nodular deposits were met with. A second case of tuberose sclerosis showed a lesser degree of identical alterations in the corpus striatum.

The authors consider there are analogies between this condition of the corpus striatum in tuberose sclerosis and what has been described by Alzheimer and others in so-called pseudosclerosis, viz., increase of glia in cortex, basal ganglia, pons, cerebellum, and elsewhere, the occurrence of giant glia-cells and of glia-cells with multiple nuclei. Westphal also is inclined to link pseudosclerosis to tuberose sclerosis. The authors think it probable that the former is a diffuse form of the latter, that the glial overgrowth is in no way dependent on degeneration of nerve parenchyma, and that it may be a kind of blastoma-formation. They are sceptical, however, of the suggestion that progressive lenticular degeneration is identical with pseudosclerosis, and cite a case of the former minutely examined by them in which the glial peculiarities of the latter were completely absent, notwithstanding careful search. They consider that progressive lenticular
Degeneration is a mainly local parenchymatous degeneration with secondary glial changes, whereas pseudosclerosis consists of a primary glial alteration of a quite diffuse character.

Wilson.


The histology of tuberose sclerosis is well known, and its association with other lesions, such as hypernephroma, persistence of the foramen Botalli, and sebaceous cysts of the skin, has been frequently noted. The author's conception of the disease is: (1) That there is no evidence of inflammation in the brain; (2) That the fundamental lesions in the tuberosities are (a) a partial fusion of the grey and white matter, (b) the presence of giant-cells of neuroglial origin, (c) a mingling of the various layers of the grey matter, a diminution in the number of the large pyramidal cells, which are poorly differentiated and abnormally oriented, and a poor formation of tangential fibres; (3) That the tuberosities in the cortex and corpus striatum do not constitute the whole disease, but are constantly associated with malformation or neoplasm of various organs of the body, of which the following list is given:

Malformations.—Macroscopic changes in the nervous system, such as agenesis of one hemisphere, a lobe, a convolution, the corpus callosum, the olives, one corpus mamillare or one cerebral peduncle; fusion of the olive with the pyramid, congenital hydrocephalus, spina bifida, or non-pigmentation of the iris.

The skull may show various malformations, and abnormalities may be found in face, ears, and palate, and also in the digestive organs. In the circulatory system there may be congenital malformations of the heart and aorta. The testicles may be small and undescended, and there may be incomplete development of the penis, or total or partial lack of hair.

Tumours.—In the heart there may be pure rhabdomyomata or mixed simple tumours containing striped muscle fibres, columnar epithelium, or cartilage. In the kidneys the commonest tumour is a typical hypernephroma; but mixed simple tumours may also occur. In the skin a variety of tumours may be present—pigmented or hairy moles and nævi, sebaceous cysts and adenomata, and diffuse papillomata, in addition to molluseum fibrosum or neurofibromata similar to those of Recklinghausen's disease.

The author accounts for these abnormalities on the theory of developmental error affecting the three primitive embryonic layers, especially the ectoderm. This appears to come on late in foetal life. He considers that there is a strong probability that syphilis plays some part in the etiology of the disease. The evidence for this rests both on the similarity of some of the lesions to those found in congenital syphilis, and on a history of premature birth, repeated abortions, or other symptoms of syphilis in the parents. He does not, however, consider the evidence so far conclusive.

J. G. Greenfield.

The paper summarizes the results found in a total of forty-two cases of herpes zoster in which the cerebrospinal fluid was examined in its uncentrifuged state. Attention was directed to cell count, albumin content, globulin content, and Wassermann reaction. The results were as follows: Lymphoeytosis in 28 cases, most marked in cases with slight eruption. No polymorphs seen throughout the series. Albumin: a slight excess in the majority of cases, most in those with large cell counts. Globulin: one case only clear excess, and this one had a marked lymphoeytosis. Wassermann test negative, except in clinically syphilitic cases.

In a large lock department, occurrence of herpes zoster was 4 per 1000, as against 1 per 1000 ordinary non-syphilitic cases, the latter compiled from general hospital wards.

J. le Fleming Burrow.

Histopathologic study on two cases of 'central neuritis'; demonstration of a new granule ('nucleoproteid-like granule') in the neuroglia cells.—K. Marui. Arch. of Neurol. and Psychiat., 1919, ii, 1.

In 1901, Adolph Meyer described, under the designation 'central neuritis', a widely distributed and strikingly symmetrical parenchymatous degeneration of numerous nerve elements, occurring in peculiar forms of depressive disorders, senile and other psychoses. The cortical nerve-cells show axonal reaction, with decay of the medullary sheaths of some of the corresponding sets of fibres, but there is no gliosis or vascular infiltration.

Almost all the Betz cells in Marui's two cases of central neuritis were in a more or less advanced stage of axonal reaction. The intracellular neurofibrils were found to be fragmented, their alteration keeping pace with the dissolution of the Nissl bodies. Marchi preparations showed definite but scanty myelin-sheath degeneration in the paracentral lobules and anterior central convolutions. The axis-cylinders in Alzheimer and Mann preparations, where normal, stained a deep-blue colour; others, apparently morbid, showed an interesting alteration in their affinity for stains. Of these, some showed red-stained parts, while others remained unstained for certain lengths. The appearance of some axis-cylinders was that of strings of changing colour: the red-stained parts were regarded as representing a more advanced stage of alteration than those which were uncoloured. Ameoboid glia-cells carrying different kinds of granules were also observed; some were identified as Alzheimer's methyl-blue granules: others as fuchsinophil granules. In the protoplasm of amöeboid glia-cells peculiar 'nucleoproteid-like granules' were discovered. They do not appear to have been previously noted. They were rather small, rounded, and of almost uniform size, and were never observed lying free in the tissues or in the perivascular spaces. In thionin-blue preparations of both formalin- and alcohol-fixed material they stained blue or an exquisite
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metachromatic colour; illumination by electric light rendered them especially visible. In their microchemical reactions they behaved like the Nissl-bodies of nerve-cells, and could not be mistaken for Reich π-granules. Nervous tissue taken from 70 insane and general hospital cases was examined for the presence of these granules; they were found in 45 cases, and thus cannot be regarded as peculiar to this condition.

The author concludes that the presence of ‘nucleoproteid-like granules’ in amœboid glia-cells indicates that neuroglia has a constructive function as well as a scavenger function, and that this granule is given to the neuroglia cells in an afferent direction.

R. M. S.


The material used in this investigation was obtained from apparently normal brains of young adult male subjects. In one case death was caused by trauma; in the remaining two cases death was due to measles and bronchopneumonia, with which there was associated high fever. Fatty pigment, in the form of minute discrete droplets, was present in some degree in the nerve-cells of all cortical layers. It occurred most abundantly in the fourth and sixth layers (Campbell's nomenclature), the next most frequent locality being the seventh zone. Fat was found in negligible amounts in the first, second, and fifth layers. It was also found in variously-shaped and irregular masses—never in droplets—in the walls of the cortical vessels, and to a lesser extent in those of the white matter. In the two cases with terminal infection there was a remarkable increase of fat in the third and seventh layers.

R. M. S.

[56] Pathogenesis of homolateral hemiplegia (Recherches sur la pathogénie de l'hémiplégie homolatérale).—V. Demole. Revue Neurol., 1918, xxv, 100.

A case is recorded of cerebral glioma in the left frontal lobe in which the paralysis of the limbs was greater on the left side, with a Babinski sign in the left foot only. It was noted at the autopsy that the right frontal lobe was compressed by the tumour, which considerably passed the middle line, and also that the falx cerebri was rudimentary, being represented only by a ridge 5 mm. in depth.

The author considers that in this case, and in the majority of the recorded cases, the presence of homolateral hemiplegia may be explained by pressure of the tumour on the opposite hemisphere. This is particularly likely to occur when the falx cerebri is narrow, lax, or defective. He rejects the other theories, such as non-decussion of the pyramids or compression of the opposite pyramid against the edge of the foramen magnum, œdema affecting the opposite hemisphere, and diascisis. In the case described, epileptiform fits were present, with deviation of the head and eyes to the right, which pointed to a lesion of the pyramidal tracts in their cerebral rather than in their medullary course.
Further, as any tumour in the anterior or upper part of one cerebral hemisphere will cause the other hemisphere to be pushed upwards towards the vertex, the corpus striatum of that side may come to lie in closer relation to the tumour than that of the homolateral side, which will be pushed backwards or downwards. It is therefore possible for a tumour to compress the internal capsule of the opposite hemisphere more than that of the hemisphere in which it arises.

J. G. Greenfield.

[57] The relation of papilloedema ('optic neuritis') to ventricular dilatation in the course of cerebral tumours (Stase papillaire et dilatation des ventricules au cours des tumeurs cérébrales).—Bollack. Ann. d'Oculist., 1919, clvi, 538.

It has long been recognized that tumours in certain cerebral areas are more likely to give rise to papilloedema than others, and Bollack has come to the conclusion that the condition of dilatation of the ventricular system is the intermediate factor between the position of the tumour and the ocular disturbance. His research is based on 27 cases of cerebral tumour, 23 with papilloedema; 14 out of 15 cases of subtentorial tumour were accompanied by it. Dilatation of the third ventricle invariably is associated with papilloedema, and the reverse is usually true, whereas dilatation of one or other lateral ventricle is much less likely to be so accompanied. In almost every case of papilloedema (16 out of 20) the aqueduct of Sylvius was distorted in one form or another, while it was normal in cases without papilloedema. Ventricular dilatation is the sequel to ventricular hypertension, and this occurring in the course of cerebral tumours may be caused by disorders in the production, resorption, or circulation of the cerebrospinal fluid. Ventricular hypertension affects the third ventricle before any other part of the system.

Bollack proceeds to examine the relation of ventricular distention and papilloedema, and criticizes several well-known theories. His own view, supported by minute and ingenious histologico-anatomical investigations, is that the pressure acts directly on the optic chiasma at a spot which he calls the ependymo-prechiasmatic triangle; this is the area where the layer of grey matter forming the antero-inferior limit of the third ventricle abuts on the antero-superior aspect of the chiasma, at an acute angle. Here Bollack has found constant pathological alterations, absent in cases of cerebral tumour without ventricular dilatation and papilloedema.

Wilson.


The affected pigeons (four in number), whose descent from the originally affected female bird is traced with exactitude through four generations, presented typical static disequilibrium, ataxia of gait, "like a drunken man", and ataxia of wing muscles, rendering flying impossible. Nystagmus was not present, and rotatory tests had the same result as in normal birds.

Pathologically, the cerebellum, medulla, and cord were obviously small
in proportion to the rest of the brain, in all the birds, and reduced in weight considerably below the average. Microscopically, the lesions consisted of decided reduction in thickness of the molecular layer of the cerebellar cortex, marked reduction of the cerebellar peduncles, formatio reticularis of the medulla, and medullary olives; in the cord, defective development of the dorsal columns, spinocerebellar tracts, and Clarke's columns.

This congenital hypoplasia, with no evidence of definite degeneration and neuroglial overgrowth, is of interest in view of its close analogy, both in the segments of the neuraxis affected and the special incidence of the atrophy in these segments, to the hypoplasia frequently met with in Friedreich's disease and the spinal-cerebellar form of degenerative ataxia (Marie's 'hereditary cerebellar ataxia').

WILSON.

[59] Regeneration of the posterior roots in cases of complete division of the dorsal cord (Sur la régénération des racines postérieures dans la section complète de la moelle dorsale).—J. Lhermitte. Revue Neurol., 1919, xxvi, 129.

It has long been disputed whether or not the fibres of the spinal cord are able to regenerate. Lhermitte took advantage of the wonderful opportunities for histological investigation afforded by traumatic injuries of the cord in the recent war, and has done much to settle the question.

Using Bielschowsky's method, the author has obtained evidence of regeneration in four cases of complete transection of the spinal cord. In two of the examples the injury was due to crushing of one or more segments in the dorsal region; in the third the cord was divided by a bullet, the meningeal sheath being preserved; and in the fourth the ends of the cord had been sutured together at operation.

If regeneration of fibres has been going on, the stumps of upper and lower portions of the divided cord are different from one another in appearance even to naked-eye examination. The stump of the upper portion looks clean-cut, while that of the lower portion is irregular and ragged, and budlike processes protrude from its surface. When examined histologically the regenerated fibres are almost invariably grouped in distinct bundles, which may be situated in the pia mater, the sheath of a vessel, or degenerated tissue. In contradistinction to the fibres which compose spinal tracts, new axis-cylinders rarely run parallel to one another; they twist about and form a regular entanglement. Newly-formed fibres can be recognized by the regularity of their outline, their small calibre, their waviness, and segregation into long spiral bundles. These fibres originate from the posterior roots. Sections at the level of entry through the pia mater show that the bundles of sensory fibres penetrate the deep layer of the leptomeninges, disperse in this region, and become continuous without interruption with the scattered bundles of regenerated fibres. In the sclerosed tissue, which in places surrounds the posterior roots at their point of entry in the pia mater, some fibres which have met the obstacle formed by the pia mater can be seen to have turned back and travelled round the posterior surface of the meninges.
The fibres of the posterior roots are endowed with great regenerative power. Even in the middle of dense scar-tissue a few axis-cylinders showing the phenomena of regeneration can usually be seen. This inherent power of growth of posterior root fibres is in marked contrast to the regenerative inertia of fibres in the long spinal tracts. In no case was there the least evidence of proliferation of the pyramidal bundles in the upper segment of the cord, or of the cerebral or posterior bundles in the lower segment.

Lhermitte believes that regeneration of motor fibres in the spinal cord never occurs in man, and in consequence suture of the transected cord, however exact, is useless. As to recovery of sensation he is uncertain, but he states that in two patients, who were under observation for fifteen and eighteen months respectively, there appeared to be some return of sensibility in the lower limbs.

G. RIDDOCH.


The author's argument in this interesting paper is that the more recent acquisitions of the central nervous system are less resistant to noxious agents than older parts, and he illustrates his contention by a consideration of disseminated sclerosis, a disease in which, despite the irregularity of the foci, for some reason a certain group of symptoms appears to predominate.

The abdominal reflexes occur in primates only, and in man they do not appear till some months after birth; they are phylogenetically and ontogenetically recent, and therefore lost early. When we consider that synchronous lateral movement of both eyes in a horizontal direction is a function which is only present in higher mammals, where the position of the eyes and the shape of the face make it possible, we can understand why horizontal nystagmus is an early and regular symptom. Similar considerations explain the pallor of the temporal half of the optic disc. Two changes have occurred in phylogensis—a large group of uncrossed fibres has appeared, and the macular bundle has increased. These new fibres arise from the temporal half of the retina, and remain in the lateral part of the optic nerve; atrophy therefore occurs in the phylogenetically younger part of the disc. The mental changes, speech defects, and sensory and motor disturbances of disseminated sclerosis are considered from the same standpoint.

In the second part of the paper fifteen cases of median neuritis, with isolated wasting of the abductor brevis and opponens pollicis are described and discussed. The author rejects so-called occupation-neuritis and over-functioning as adequate explanation of the selective action of the morbid process; he holds that these muscles, and these alone, were picked out first and most, because they are phylogenetically younger than other muscles, which, though submitted to the same strain, remained normal.

W. J. ADIE.
ABSTRACTS


Bielschowsky's proposed classification of the inherited neural degenerations is as follows, incomplete though its author allows it to be in the present state of knowledge:

I. Pure Dysplasias.
   A. Cortical malformations; micropyogryia, pachygyria, agyria.
   B. Malformation of corpus striatum; état marbré.
   C. Malformation of more caudal parts of neuraxis; micromyelia, syringomyelia and its analogues.

II. Dysplasias with a Blastoma Element. Tuberose sclerosis (related to glioma and Recklinghausen's disease).

III. Abiotrophies.
   A. Abiotrophies with a blastoma element; pseudosclerosis (and certain forms of diffuse sclerosis?).
   B. Abiotrophies with local total necrosis of the parenchyma:
      2. Necrosis of globus pallidus: a solitary case recorded by Fischer.
   C. Abiotrophies with selective necrobiosis of ganglion cells:
         b. Ditto with special involvement of certain systems: juvenile (chronic) amaurotic idiocy with cerebellar atrophy.
      2. Selective degeneration of certain cell-systems:
         a. Of nucleus caudatus and lentiformis: chronic chorea.
         b. Of cerebellar systems: cerebellar heredo-ataxia in its many forms.
         c. Of corticospinal system: spastic spinal paralysis, spinal amyotrophy, amyotrophic lateral sclerosis.

Wilson.

SENSORIMOTOR NEUROLOGY.


The case was that of a man of 57, who suffered at the age of 17 from a severe illness, apparently of a toxic or infective nature, as a result of which he showed slight but definite signs of defective cerebellar function. Very gradually these signs became intensified, and when he came under observation his condition was one of advanced cerebellar disturbance. In addition, there was a definite rigidity of certain muscular groups, and the