and Sherrington and to demonstrate how the nervous system integrates and controls the functions of the body.

R. G. Gordon.

**NEUROPATHOLOGY.**


The material investigated consisted of the brain and spinal cord of a young girl, who died in an attack of acute chorea on the tenth day of her illness. The authors summarize the results of their researches as showing infiltration by inflammatory products both of the perivascular sheaths and the nervous substance, together with destruction of the nerve-cells in certain areas. The inflammatory process is very diffuse and irregular, and beginning around the vessels involves the whole cerebrospinal axis with the exception of the medulla and cerebellum, which appear absolutely intact. Thus the spinal cord, pons, cerebral peduncles, the basal ganglia, and the cerebral cortex are the sites affected. The changes are most marked in the grey matter of the basal ganglia. The process has a selective incidence upon the grey matter; in contrast, the centrum ovale, corpus callosum, and cerebellar peduncles are intact. The process, therefore, is to be considered as a polio-encephalitis.

The writers describe the nature of the lesions in detail, with appropriate figures, and point out their close resemblance to those found in encephalitis lethargica. They consider, however, that in the case under investigation the cerebral cortex was more extensively involved than is usual in the other disease.

Commenting upon the similarity of their findings to those described by other authors, they consider it as established that histological changes of this type are found in children dead of acute chorea. They further draw attention to the correspondence between the especial localization of the lesions in these cases and the findings of Marie and Lhermitte in Huntington's chorea, the areas most affected in both instances being the cerebral cortex and the corpus striatum. No clinical details are offered in proof of the accuracy of the diagnosis in the case which is the subject of this paper, and, as the authors themselves point out, the differentiation of a case of Sydenham's chorea from one of encephalitis lethargica with choreiform movements may be no easy matter. In view, therefore, of the histological picture described, the reader may perhaps be excused for wondering whether they were not dealing with a case of the latter disease.

C. P. Symonds.


The object of these investigations was to determine, first, the extent to
which polymorphonuclear leucocytes participate in inflammatory processes in the nervous system; and secondly, the origin and nature of the cells which are found in the infiltrations around the vessels and in the nervous substance. To this end, using a method which is free from objection—the oxydase reaction—the author examined a number of cases of poliomyelitis and encephalitis lethargica. His most important conclusion is that polymorphonuclear leucocytes play a much greater part than recent writings have lead us to believe. He found that up to the third or fourth day the great majority of the cells, both perivascular and within the nervous substance, were polymorphonuclear leucocytes; in other words, that the cells described by Wickman as polyblasts, and by Wallgren as polyblasts, polymorphonuclear glia cells, and degenerated leucocytes, are in reality almost all derived from them. After the fourth day altered polymorphonuclear leucocytes are replaced by polymorphonuclear cells of glial origin in the intranervous infiltrations. In the lymph spaces the polymorphs also disappear rapidly, and in a few days lymphocytes and plasma-cells alone are found around the larger vessels. Around the smallest vessels a few polymorphs of glial origin may be found in the later stages. In agreement with Marchand and others it was found that the Fettkörnchenzellen were derived from the glia and not from leucocytes. It is probable that the lymphocytes do not migrate from the blood-vessels, but that they arise from the lymphatic channels. Neuronophagia was prominent in all the cases. In the acute stage the neurophages are polymorphonuclear leucocytes, later glial cells. Plasma-cells arise from lymphocytes and not from glia.

Hauptli finds himself in agreement with the older writers, Harbitz, Richter, and others, who gave an important rôle to polymorphonuclear leucocytes in the acute stage of inflammation of the nervous system. He cannot support Wickman, who derives the polymorphs of the later stage from lymphocytes, and likens them to the polyblasts of Maximow, but agrees with Wallgren, Homén, and others that they are of glial origin. He agrees with Wallgren that polymorphonuclear leucocytes are present in large numbers, but during the first few days only.

In encephalitis epidemica the findings were identical in every respect with those in poliomyelitis. The differential diagnosis cannot be made with certainty on the histological changes alone.

W. J. Adie.


Bielschowsky's admirable paper deserves full consideration by all who are interested in or working at the subject. It concerns three cases of the disease in a Gentile family (N.B., not Jewish), occurring in each of their three children, beginning at the age of four in them all, and leading to profound idiocy and marasmus after a course of some three and a half
years. Its place, therefore, is intermediate between the infantile and the juvenile varieties: it is designated by Bielschowsky 'late infantile'.

The weight of the brains was 760 grm., 670 grm., and 685 grm. respectively—some 250 grn. below the normal weight for the age. Covered by a fibrous leptomeningitis, the brains were normally convoluted, but there was a tendency for the superficial cortical layers to split off from the deeper, especially in the calcarine area. The cortex was universally reduced, again especially in the occipital region. White matter was distinctly below the normal proportion. In all three the cerebellum was notably atrophic, not merely absolutely, but also in proportion to the already reduced size of the cerebrum, and this atrophy implicated both vermis and lateral lobes. No evidence of inflammatory processes was found in the meninges.

Throughout the whole of the nervous system the nerve-cells showed the characteristic swelling of the cytoplasm, with granular degeneration; the denarites, on the other hand, were much less involved than in the pure infantile variety, in comparison with which, further, the cell-degeneration was more restricted in degree to the cells of the cortex. In the basal ganglia, mid-brain, pons, medulla, and cord, many more or less normal cells existed alongside degenerated ones. Bielschowsky shows that the granular change in the cytoplasm is not derived from the breaking up of Nissl bodies. It is common to find a plasmatic network in the cell protoplasm, enclosing the degenerated granules, and distinct from the cell fibrillae.

The occipital cortex was the seat of a spongy disintegration, especially in the calcarine area, largely restricted to the lamina pyramidalis. Yet the myelo- and cyto-architectonic structure of the calcarine cortex was not otherwise grossly altered. Glial changes were prominent throughout the brain. The reduction of the white matter was presumably in part a hypoplasia and in part a degeneration. Striking changes were seen in the retina—loss of rods and cones and of the outer granular layer, glial degeneration, pigment deposits, etc. Yet the optic nerves were but slightly atrophic.

In the cerebellum the chief alteration was one of sclerosis; i.e., there was an immense overgrowth throughout the organ of fibrous glia. The granules of the granular layer had vanished. The Purkinje cells were largely present, but nearly all were degenerated. Many astrocytes were to be seen in the molecular layer; in it and in the layer of Purkinje cells no trace of nerve-fibres remained. Afferent and association fibre-systems alike, in the cerebellum, were conspicuously defective. Bielschowsky considers that the changes in the Purkinje cells were not those of the ordinary cell degeneration in amaurotic idiocy, but rather akin to what is found in multiple sclerosis, juvenile general paralysis, etc. All the cerebellar nuclei were atrophic. Degeneration in the peduncles and in the olivo-cerebellar tracts suggests an analogy with the form of cerebellar atrophy known as olivo-ponto-cerebellar (Dejerine and Thomas); but on the whole the author is inclined to regard the cerebellar alterations as the result of the general morbid process acting on that organ.

The discussion of the significance of his material and the correlation with analogous cases from the literature are particularly informative.

S. A. K. W.
[110] Sensory disturbances from lesions in the oblongata and the pons.—C. Bergmark. Upsala Läk. Forhandl., 1921, xxvi, Häft 5–6, contrib. no. 4.

A fine paper, based on minute examination, clinical and pathological, of three cases; one of tumour at the level of the foramen magnum, one of occlusion of the posterior inferior cerebellar artery, and one of tumour of the medulla oblongata. The original should be consulted for many details which cannot here be referred to.

1. Disturbances of sensibility in the trigeminal area on the side of the lesion.—Much evidence from cases of thrombosis of the posterior inferior artery shows that touch cannot be conveyed solely by the descending root of the fifth and that it must have a localization on the central side of the Gasserian ganglion different from that of the other cutaneous senses. The author's finding is that it is partly localized within the descending root of the fifth, and partly (possibly following the motor fifth fibres) in the sensory nucleus of the nerve in the pons.

2. Dissociated disturbances of the senses of pain, cold, and heat in the crossed half of the body.—The author's evidence is against the somewhat dogmatic conclusions of Marburg, that in the conduction of pain and temperature impulses by the spinoctectal and spinothalamic tracts pain is mesially situated, heat laterally, and cold between the two. He can only say that "the sense of pain is situated relatively mesially more often than the other qualities". He further emphasizes the somewhat important point that in not a few cases the disturbance of sensibility is of the same type in the uncrossed half of the face and the crossed half of the body, which suggests the desirability of caution in drawing conclusions as to the topography of the different tracts for the cutaneous senses.

3. Localization and the so-called space sense.—Evidence is adduced which tends to show that localization of stimuli is effected not only by the dorsal column of the cord, but also by the lateral column on the opposite side. The topoipnostic sense (Ortsinn) is mediated, apparently largely though possibly not entirely, by the dorsal columns.

4. The muscular sense.—This sense has at its disposal two tracts, which can replace each other functionally. One is represented by the dorsal column of the cord, fibres arcuate internae, and lemniscus. The other runs in the spinocerebellar tract of Flechsig and is still uncrossed when it reaches the cerebellar hemisphere, but then crosses to the other side and reaches the brain stem through one of the large cerebellar commissures of the crossed side. Under ordinary conditions the impulses of the muscular sense are probably transmitted mainly in the dorsal column system.

S. A. K. W.


The author starts out on an endeavour to explain the processes of functional nervous disease on a basis of physiology. After referring to the current views with regard to such diseases, he recapitulates Sherrington's conclusions as enunciated in his "Integrative Action of the Nervous
System” and elsewhere. He then attempts to show how the laws laid down by this author operate on the ‘emotional’ level. Here it would have been better if the author had been clearer in the definition of terms which he uses; the word ‘emotion’, e.g., is used in so many different senses that it is difficult for the reader to realize what exactly is meant. Finally the author refers to the various methods of therapy in relation to this point of view.

The paper certainly merits attention as suggesting an important line of investigation, but it is impossible to abstract a short communication dealing with such a large subject, for it is bound to be reduced to the full limits of abstraction already. The general impression given is that the author has somewhat wearied of well doing, and the article does not quite fulfil the high expectations which are aroused in the first paragraphs.

R. G. Gordon.

[112] The mechanism of referred pain, hyperalgesia (causalgia), and of alcoholic injections for the relief of neuralgia.—Joseph Byrne. Jour. of Nerv. and Ment. Dis., 1921, liii, 483.

Functional or anatomical interruption of the afferent system of pathways, and more especially of those in the peripheral nerves, is associated with more or less persistent pain and tenderness which are referred to the area of distribution of the injured pathways. This pain is to be distinguished from reflected pain due to lesions of visceral organs, with which this paper does not deal. From periphery to brain two separate systems of afferent paths serve the purposes of sensation—the affective and the critical; but in testing sensory function there are four sets of afferent pathways to be kept in mind—superficial critical, superficial affective, deep critical, and deep affective.

The thalamus is concerned with affective perception and the cerebral cortex with critical perception, but the latter influences and controls the former at the level of the thalamus. Lesions at various levels illustrate the function of these systems. Injury to a nerve trunk, e.g., by compression, does two things—(1) more or less completely interrupts the function of the critical system, thereby abolishing its control over the affective system; (2) partially interrupts function of the affective system, thereby stimulating it to increased metabolic activity in the interests of functional restoration and repair. At first the energy is directed to repair, but if this is impossible the hypermetabolism gives rise to accumulation of energy which, no longer concerning itself with the more primitive embryonic function of growth and repair, becomes diverted to the more highly specialized function of preparing the materials necessary for the elaboration and discharge of nerve impulses. This results in the spontaneous or readily elicited discharge brainwards of impulses which are the immediate cause of pathological pain and tenderness. A useful diagram is given showing possible lesions on the sensory paths and the effects on the two systems. A description is given of the ordinary changes which take place in the myelinated and unmyelinated fibres after section of a sensory nerve.

After discussing the various forms of referred pains with illustrative
cases, and the relative influence of the affective and critical systems, the
author concludes that: (1) All pain, whatever be the cause, is in reality
referred pain. (2) The great causative factors in referred pain are (a)
mechanical trauma such as contusion, over-stretching, compression; and
(b) infection, the mechanism essentially consisting of the resulting hyper-
functioning of the related affective neurones as set forth in the text. (8)
The incubation period, during which pain and tenderness are absent for
some time following severe injuries of nerve trunks, is the result of axonal
reaction in the related neurone bodies of the spinal ganglia, with consequent
cessation of specific function. (4) Alcoholic and other injections into the
nerve-trunks for the relief of neuralgic pains act by inducing axonal
reaction phenomena and suspension of specific function in the related
affective neurone bodies in the spinal ganglia. (5) In all operative
procedures, crushing, stretching, or otherwise injuring portions of nerves
that are to remain connected with the spinal cord should be scrupu-
ously avoided, as these are great factors in the causation of interstitial
neuritis, neuromata, and other conditions well known to be vital sources
of referred pain. (6) In assessing the value of etiological factors, with a
view to therapy in neuralgia and neuritis, due weight must be attached to
occupational and postural trauma of the nerves, muscles, tendons, and
ligaments, as well as to exposure and faulty function from whatever cause.
(7) In the investigation of painful conditions the afferent pathways from
the periphery to the thalamus must be kept in mind and not merely the
locus of the pain and tenderness. It is only in this way that the location
and modus operandi of the causative factors can be appreciated and
effectively attacked.

R. G. Gordon.

[113] Some observations on cistern punctures.—Henry McCusker.
Jour. of Nerv. and Ment. Dis., 1921, liii, 453.

The author describes the puncture of the cisterna magna on 55 occasions
in 9 patients, of whom 6 were cases of neurosyphilis, 1 spinal tumour, 1
lumbosacral meningitis, 1 of undiagnosed head symptoms. The head and
neck of the patient are shaved from the occipital protuberance to the 6th
cervical vertebra and prepared as for other operations. The patient is
placed on the operating table lying directly on his left side; the head is
flexed on the chest and placed on a pillow so as to bring the occipital
protuberance on the same horizontal plane as the vertebral spines. By
palpation the depression between the occipital protuberance and the spine
of the axis is located, and this area is well cocainized. The outer skin is
punctured by a sharp lumbar-puncture needle, which is then discarded,
and another of 18 gauge and graduated in centimetres is introduced. The
direction of the needle is slightly forward, and along a plane passing through
the glabella and upper edge of the external auditory meatus. At a depth
of 4 to 5 cm. it will have pierced the dura and the posterior occipito-
atlantoid ligament and entered the cistern. The needle is never introduced
more than 5 cm. for fear of injuring the medulla. After withdrawal,
pressure should be maintained over the puncture for some minutes and a
sterile dressing be applied.
NEUROLOGY

In the 55 punctures the author claims there was no evidence of injury to the medulla, no deaths, and no blood in the spinal fluid. The nuchal area was less sensitive than the lumbar area, and there was no sudden extension of the head. There was a consistent absence of pain on piercing the dura. There was no puncture headache, serum reaction, or backache, and no root pains or medullary symptoms, such as slowing of the pulse or respiratory changes. The punctures were made without difficulty, and with less discomfort to the patient than is generally found after lumbar puncture.

R. G. Gordon.


The author has performed experiments on rats exposed to varying degrees of cold, using his own method of staining to demonstrate Golgi's apparatus. The relatively protracted exposure of small animals to a moderate degree of cold is sufficient to cause peculiar changes of Golgi's internal apparatus in the nerve-cells of the dorsal horns, intermediolateral and middle cell column, and grey matter surrounding the central canal. Such changes are characterized by an unusually robust aspect of the whole apparatus and of its constituent parts, which show a tendency to occupy a tight perinuclear situation. In more affected specimens the apparatus gradually loses its characteristic structure, and becomes transformed into irregular and variously-arranged masses and pieces. These alterations are accompanied by an appearance of canaliculi and spaces similar to those generally spoken of as Holmgren's trophospongium. If the exposure to cold has ultimately been such as to lower considerably the normal temperature of the animals experimented upon, the same apparatus undergoes a further form of almost complete disintegration.

R. G. Gordon.


This paper is in the form of a clinical lecture dealing with nervous and mental disease due to congenital syphilis. The author points out that in such children the disease attacks the brain more often than the spinal cord, whereas the reverse is probably the case in acquired syphilis. He considers diabetes insipidus to be a characteristic symptom of syphilitic involvement of the pituitary body. We do not altogether agree with the value he places on the Lange test in the cerebrospinal fluid. A curve such as 1111050000 would convey nothing to us except that one tube was dirty, and would certainly not be accepted as evidence of meningeal involvement; and we should like to know the type of curve obtained by the author in normal children and non-syphilitic epileptics before accepting changes in the colloidal gold of the order of 1, 1½, and 2 in a few tubes of the test as evidence of syphilitic irritation of the meninges.

J. G. Greenfield.