[85] The chemistry of degenerating nervous tissue (La chimie de la substance nerveuse au cours de sa dégradation).—Raoul M. May. L'Encéphale, 1930, xxv, 447.

The author reviews the older work of Koch, Mann, Mott and others on the gross chemistry of the brain and adds some experiments which he has himself performed by microchemical methods. Of special interest in his review of the literature are the observations of Delaville and Tcherniakofsky that in patients with anxiety states and in epileptics the chlorides in the blood rise with the crisis, the rise being especially in the intracorpuscular chlorides. Corresponding with this they found a great increase in intracerebral chlorides in a case of dementia paralytica and in a case of organic dementia. The author’s own experiments do little more than test the applicability to this form of research of the micro-methods of Pregl. Unilateral lesions of the cerebrum of a guineapig and of the sciatic nerves of rabbits were examined after varying intervals, and a similar sequence of chemical change was found in both series of experiments. The total sulphur and nitrogen rose and the total phosphorus fell, the water remaining constant or rising slightly on the injured side. In spite of the fall in the total phosphorus, there was found to be a rise in the fraction of phosphorus soluble in water which included the phosphates and the simple organic compounds of phosphoric acid. The chief fall was in the lipoid and protein phosphorus compounds, and in a fraction soluble in alcohol but insoluble in ether or water. Translated into the more familiar terms of histology, these experiments indicate no more than a disintegration of myelin and phosphorus-containing nervous elements; and while they appear to demonstrate the utility of microchemical methods for this form of research, they do not lend any support to the author’s claim that the chief advances in neuropathology in the future are likely to come from the gross chemical examination of diseased nervous tissues.

J. G. G.

SENSORIMOTOR NEUROLOGY.


A long and comprehensive review of the subject. A personal case is fully discussed and the author concludes that the ophthalmoplegic factors of these cases depend on lesions of the oculomotor nuclei and the fibres derived therefrom. The alterations come into the category which other authors have described in cerebral tumours and used to explain certain symptoms, viz., ‘action at a distance.’ This may be partly due to general toxic action, but may
also have reference to some selective factor evolved in the life of the tumour. The author concludes that the syndrome of temporal lobe tumour comprises the following:

1. The general symptoms of cerebral tumour.
2. The local symptoms of sensory aphasia if the tumour is on the left.
3. Neighbouring symptoms and signs of disturbance of the pyramidal tract.
4. Symptoms of distant effects, lesions of the third nerve and cerebellar components.

The syndrome of some of these tumours is very vague and confined to a few psychical disturbances, but the author thinks that such are in the minority, and that localisation in respect of temporal neoplasms is becoming more defined.

R. G. G.


Symptoms referable to the region of the third ventricle are apt to be overlooked or disregarded when the sella turcica is reported normal. The differential diagnosis between tumours of the third and of the fourth ventricle is difficult since each may give the symptoms of the other.

Polyuria, adiposity, hypersomnia are among the common indications of a third ventricle neoplasm, to which the authors now add the occurrence of Quincke’s oedema. Ventriculography is of great service in precise localisation. If the tumour is inoperable X-ray radiation sometimes effects remarkable clinical improvement.

S. A. K. W.


This long article contains matter of considerable neurological interest and may briefly be summarised.

Forced grasping arises through removal of inhibition in the motor system. It shows itself clinically when the pyramidal path is for practical purposes more or less intact physiologically. In all probability the inhibitory path is of cortical origin and appears to come from the first or upper frontal gyrus and perhaps also from the cortex of the neighbouring gyrus forniciatus—lying mainly on the mesial aspect of the hemisphere.

According to the authors’ researches the phenomenon develops in recognisable degree on one side as soon as the mechanism controlling grasping is inter-
ferred with from both frontal lobes. In the majority of their cases the lesion was so situate as to involve both frontal lobe and corpus callosum and hence to effect the double action just mentioned.

It makes its appearance on the side opposite to the 'blocked' hemisphere, but is sometimes accompanied by a much milder degree of the same disorder on the homolateral side. No clear evidence points to the predominance of one frontal lobe over the other in this connexion.

For the conduction of the inhibitory influence the occipito-frontal tract seems to be of significance, and impulses are transmitted to the opposite side via the corpus callosum.

S. A. K. W.


The case described is that of a woman of about 70 who in 1914 had a cerebral attack (right hemiplegia with some aphasia). A similar attack, with the same consequences, came 18 months later. Some three years before her admission to hospital the legs had become progressively weak and then powerless, and later the arms were affected. Symptoms of organic dementia also made their appearance.

Towards the end the clinical condition was as follows: the patient lay half on her left side, both legs sharply flexed at knee and hip, the right arm adducted and flexed at the elbow. Only the left arm was capable of any movement, the other limbs being paralysed. Tonus was everywhere increased; the knee-jerks could be obtained but were very slight; the plantar reflexes were always doubtful. No ankle clonus could be elicited. On the other hand, the reflexes of 'defence' were always obtainable in the legs.

The pathological condition proved to be one of severe cerebral arteriosclerosis, with a general cortical and subcortical atrophy—mainly the result of numerous small necrotic foci and haemorrhages. Lenticular nuclei and to a less extent the thalami were greatly reduced in volume. On the other hand, the anterior thalamic nuclei, nucleus ruber, corpus Luysii, and substantia nigra were for practical purposes normal, while structures at lower levels showed no primary injury.

The case may be considered one of combined pyramidal and extrapyramidal character. Decerebration down to the level of the pars magnocellularis of the nucleus ruber allows of paraplegia in flexion; if the lesions extend further to include the whole of the nucleus, decerebrate rigidity of the usual type will develop. But the same paraplegia in flexion may be derived from lesions involving frontal and central gyri only, so that the basal ganglia presumably take no part in its onset. Regression to the flexed type is caused by
destruction of the frontal lobes and is accompanied by paralysis if the pyramidal systems are also involved. Lesions lower down effect no change in this condition till the level of the pars magnocellularis is reached, when the type changes to that of full decerebrate rigidity.

S. A. K. W.


This interesting communication is based on a case of absolute crossed pyramidal degeneration on one side (infantile cerebral hemiplegia), in which none the less some functional power and movement existed on the affected side. There was definite evidence of hypertrophy of the sound pyramidal system. Several modes of functional substitution are sketched; these include more use of the direct pyramidal tract, or of homolateral pyramidal fibres; a further suggestion is abnormal division at the crossing. A number of references to similar cases are given.

S. A. K. W.


Thirteen cases are reported from the literature of hemichorea, 11 of which showed lesions in the subthalamic body of Luys. In these the chorea was of great violence, a feature described as 'hemiballismus.' This particular variety of chorea is ascribed to the above lesion. In six there seemed to be a diffuse lesion of the extrapyramidal motor system and especially of the cerebello-rubro-thalamic system. The author thinks that lesions of the subthalamic region may cause different symptoms in experimental animals and in man; and that vegetative functions belong to many of the nuclei, including that of Luys, in this region. He thinks that the violent chorea is probably due to interference with a neuronic system, possibly the cerebello-rubro-thalamic tract.

R. G. G.


The case described is that of a woman of 65 who exhibited the syndrome of hemiballismus on a basis of cerebral arteriosclerosis. A softening was found
involving the right corpus Luysii but spreading beyond its anatomical limits. The writer argues for that structure being considered the essential anatomical factor in the production of the syndrome. Yet he admits that not every case of disease of the corpus is followed by hemiballismus, and contends that only strictly limited lesions will have that consequence. He does not accept the view that the clinical phenomena belong to the 'irritative' class, but considers them in the 'release' category. The normal function of the corpus is thought to be one of 'special movement-activities within the extrapyramidal mechanism'—whatever that may mean.

S. A. K. W.


The clinical features of the case, that of a woman of 46, include: gradual onset, after indefinite neurasthenia-like complaints, of pyramidal signs, coupled later with symptoms of striatal type; amnesia, severe dementia, and complete helplessness, mental and physical.

Its pathological basis is described in the paper by Stender (see below).

S. A. K. W.


The examination in the case reported by Meggendorfer (above), and in two others, is summarised as follows: severe degenerative processes throughout the cerebral cortex and the basal ganglia, leading to sclerosis: marked fatty changes in cells, with remarkable proliferation of the protoplasmic glia. On the whole, the motor areas, frontal lobes, striatum and medial nuclei of thalamus are most involved; to a less extent, parietal and temporal cortex, globus pallidus and insula.

For anatomical reasons the condition should be separated from senile parenchymatous affections and from general paralysis. Of its etiology the author remarks that it may arise on a basis of heredo-degeneration, but cannot say whether this of itself suffices.

S. A. K. W.


The authors deal only with the referred part of visceral pain and describe how in cases of angina pectoris, cholecystitis, duodenal ulcer and subacute appendi-
NEUROLOGY 267

citis this was completely relieved by local intracutaneous injections of a 2 per cent. solution of novocain.; in many of their cases the true visceral part of the pain seemed also to be lessened at the same time. Thus superficially referred pain from a viscus is not a matter of central nervous projection, for it is abolished by local anaesthesia. They conclude that for the appreciation of referred pain from a viscus an intact reflex arc including the somatic portion is essential—the somatic neurones must be able to conduct normal afferent impulses from their periphery before referred visceral pain can be experienced in their distribution.

They noted too that deep discomfort was often lessened by surface anaesthesia, and suggest that the absence of parietal afferent impulses lessens the excitability of the visceral neurones—that afferent impulses from the source of visceral irritation are amplified to some extent by cutaneous impulses from the corresponding somatic segment.

A. G.


With Bailliart’s ophthalmodynamometer, applied on the globe of the eye, it is possible so to augment intraocular pressure as to obtain pulsations of the central artery of the retina and by increasing it to cause them to cease. These diastolic and systolic pressures provide an index to intracranial pressure.

Full details are supplied of the instrument itself and of the necessary technique. By its means valuable corroboration of results obtained by spinal manometry can easily be procured. Many clinical data are recorded in the article.

J. V.


By means of their instrument—the nystagmograph—the authors studied the characteristics of the slow phase of optic nystagmus. It was found that the slow phases are essentially eye-movements of pursuit, and may be termed adequate when the point of regard consistently falls on one of the moving objects of interest; inadequate when the point of regard overshoots or lags behind the visual object.

With increasing speed of oscillation the rapid corrective eye-movements first increase and then decrease in both number and amplitude, occasionally reaching still fixation.

R. M. S.

This paper is divided into two parts, the first of which is the report of a case with pathological findings. At the age of 36, following a fall from a balloon in which, in spite of a parachute, he suffered some injury, the patient began to have constant choreiform movements. He became progressively insane and eventually demented so that he had to be certified at the age of 47. When examined soon after this he was quite unable to walk, to speak, or to feed himself. His limbs and trunk were rigid, but the muscles were not hard and the contraction was variable. When he stood on his feet choreiform movements were much more evident. There was no propulsion or retropulsion but when pushed in any direction the patient fell to the ground unless supported. Although the tendon reflexes were brisk and perhaps exaggerated the plantars were definitely flexor in character. None of the Magnus and de Kleijn reflexes could be elicited.

In the month which elapsed between this examination and the death of the patient there was still more disappearance of the choreiform movements and a greater tendency to fixed symmetry of the position of the limbs.

At autopsy there was general shrinkage of the brain, affecting the cortex diffusely so that all the sulci were widened. The basal ganglia also were noticeably shrunken, but no softenings were seen anywhere. Histologically a considerable loss and sclerosis of nerve cells was seen throughout the cortex. Still more evident was the disappearance of the small nerve-cells of the corpus striatum. The larger nerve-cells in the putamen-caudate and in the globus pallidus were also greatly diminished in number, but not to the same extent. There were moreover in the corpus striatum and globus pallidus inflammatory lesions of the nature of perivascular infiltration and inflammatory nodules. Apart from some degeneration of the cells of the substantia nigra the brain-stem was normal and the changes in the optic thalamus were slight.

In the second half of their paper the authors discuss the relationship of chorea and rigidity to lesions of the basal ganglia. Two other cases of rigidity following on chorea have been described, one by Bielschowsky, the other by Jakob. In Bielschowsky’s case the disease began at the age of six. Rigidity supervened on choreiform movements after the age of nine and death occurred at the age of 14. The most striking histological feature in this case was the loss of the small cells of the putamen-caudate, but there was also considerable atrophy of the large cells of the corpus striatum and globus pallidus. Cellular lesions were found in the corpus Luysii, the red nucleus and the substantia nigra, and degeneration of the cortical nerve-cells was also manifest.

Jakob’s case was that of a man who came into hospital at the age of 47 with chorea and died five years later with progressive rigidity during the last year of his life. In this case also there was considerable loss of cells throughout
the globus pallidus and corpus striatum but especially in the caudate nucleus. The substantia nigra was also affected. The authors in a review of the literature of progressive chorea discuss the various views which have been held as to the causation of chorea by lesions of one or other part of the basal ganglia. They are inclined to accept the view that rigidity is brought on by a lesion of the large cells of the globus pallidus and corpus striatum and that choreiform movements may be related to loss of the small nerve-cells of the corpus striatum. In the cases cited the spread of the disease to the globus pallidus seems to have produced muscular rigidity and at the same time to have abolished the choreiform movements. They hold that Foerster’s observations disprove the relationship of cortical lesions to chorea. The mechanism by which these modifications of muscular control are brought about is not yet at all fully understood, although various theories have been put forward to explain them. They accept, however, as broadly true, the view that the corpus striatum and globus pallidus are motor organs subserving automatic movements; the corpus striatum being a complex organ controls the activity of the more simply constituted globus pallidus, and both control the lower centres. But the results of lesions of these centres may depend not only on their topography but also on their quality. Some may be due to excitation of nerve-cells, others to their destruction, and the same or similar symptoms may arise from excitation of one group of cells or from destruction of the cells which normally control them.

Although in this part of their paper the authors have done little more than discuss theories, the association of their case with others in which a similar clinical picture was associated with a lesion of similar nature and topography makes it unusually interesting and important.

J. G. G.

[99] A case of ‘hormetonia’ [Davidenkoff] resulting from an intraventricular hæmorrhage (Un cas d’hormétonie de Davidenkoff consécutif à une hémorragie cérébrale ventriculaire).—TEODORE DOSUZKOV. L’Encéphale, 1930, xxv, 302.

The case reported is that of a woman of 63 who two years before her final attack had suffered from a stroke with right hemiplegia and some aphasia. The final attack, which was fatal in two and a half hours, was unusual in that within a few minutes of the stroke the limbs on both sides, but particularly on the right, were in a state of considerable hypertonus. A ‘fan sign’ appeared on both sides and a grasp reflex in the right hand. After half an hour hypertonus was replaced by hypotonus: this again was later replaced by hypertonus, and so on until her death. Post mortem the old lesion was present as a focus of softening near the vertex of the parietal lobe, which spared the internal capsule. Old hemorrhages were also present in the cerebellar hemispheres, affecting among other structures both dentate nuclei. The new
haemorrhage had burst through the head of the right caudate nucleus and optic thalamus into the third ventricle, destroying also the inner surface of the left optic thalamus, and spreading into the iter and third ventricle.

The author discusses the syndrome of hormetonia described by Davidenkoff in 1919. This consists of alternating hyper- and hypotonus in the paralysed limbs, coming on immediately after a stroke. It is associated with defence reflexes and postural neck reflexes and a loss or at least great diminution of consciousness. It appears to be caused by various severe lesions of the hemispheres or brainstem, and is considered by Davidenkoff to be the human analogy of decerebrate rigidity in animals.

J. G. G.


The case described here is that of a girl of 19 whose movements had become progressively choreiform for six weeks before death. It is noteworthy in view of the pathological findings that the patient had been given intravenous injections of 2 and 3 grammes of sodium salicylate, and that during the 48 hours which preceded death the temperature had risen gradually to 40°C. The most noteworthy alteration in the nervous system was a diffuse chromatolytic lesion of the nerve-cells, which affected in greater or less degree all the cells examined from the medulla to the cortex. Slight neuroglial changes and some perivascular oedema were also noted, but no inflammatory changes were found. The case thus gives little help in the elucidation of the vexed problem of the pathogenesis of chorea minor. There appear to be two groups of cases, one in which inflammatory lesions have been found in the basal ganglia and cortex, the other in which the lesions are of a purely toxic or degenerative character. In the present case they are so diffuse as to suggest that they were rather due to the manner of death than that they bore any relationship to the preceding symptoms. The authors criticise at some length the theories of Kinnier Wilson on the cortical origin of chorea, and consider that lesions of the basal ganglia or of the cerebellum may play a large part in the production of choreiform movements.

J. G. G.

PROGNOSIS AND TREATMENT.

[101] Further contributions to the chemical induction of hyperpyrexia in the treatment of general paralysis and other diseases of the neuraxis (Ulteriore contributo alla piretoterapia chimica nella paralisi progressiva ed in altre malattie del nevrasse).—P. ARMENISE. Riv. di pat. nerv. e ment., 1930, xxxv, 326.

The author has treated 12 cases of general paralysis, 3 of cerebral syphilis and 7 of dementia praecox by supraperiosteal injection of oleated sublimed