ABSTRACTS

SENSORIMOTOR NEUROLOGY.


A description is given of five personal cases in which the prominent clinical feature was so-called mindblindness, a variety of agnosia. In four of the five definite hemianopia was present; in the fifth, it was probable. Acuity of vision was unimpaired. Disorientation in space was usually more or less pronounced, and, as in common enough, was more noticeable when the patient was being tested than in his ordinary spontaneous activities. As for the mindblindness (visual agnosia) it showed itself in the failure of the patients to recognise and use the ordinary objects of daily service (watch, spoon, glove, etc.) and in a similar failure as far as their pictorial representation was concerned. More elaborate pictures were of course even more confusing. One patient invariably failed to recognise the physician. Yet, again as usual, visual perception was not at fault. The visual qualities of a watch, its shape, colour, metallic nature, polished appearance, etc., were always visually appreciated. In contrast to some other recorded cases of the same condition, the authors’ patients as a rule were able to give sufficiently complete visual descriptions of the features of a given object named to them, such as a tuning fork, and it was remarkable that the patient should absolutely fail to recognise a real tuning fork handed to him when a moment before he had described it fully from memory. On some occasions, again, optical qualities were attributed to objects in description which as a fact they do not possess—a kind of visual confabulation.

In all the cases, in greater or less degree, tactile agnosia (inability to name an object by touch) was observed, and this has been a feature of other recorded cases; it too, was a phenomenon noticed in more striking fashion during examination than when the patient was left to himself. The stereognostic qualities of the test objects were usually recognised. An interesting observation was made in some instances, viz. that the patient recognised the object better by the tactile route when the eyes were shut. Motor apraxia was not noted, whereas alexia was for obvious reasons prominent.

Subsequent examination showed that in all five cases the lesions were approximately in the same region, viz., left cuneus, calcarine area, gyrus fusiformis, gyrus lingualis.

S. A. K. W.


The author publishes the clinical histories and post-mortem findings in six cases of Huntington’s chorea. He regards the insanity in this disease as one of the dangerous type, its most marked features being sudden outbursts of violence, sexual excesses and a tendency to suicide.
Clinically he views the peculiarities of speech, posture and gait as being dependent on the jerkiness of the chorea. A true Rombergism never exists.

His notes on the post-mortem findings in the central nervous system itself are scanty, but he states that, contrary to the usually accepted view, none of his cases showed any evidence of meningoencephalitis. In only one case was there the slightest thickening of the pia-arachnoid. The most marked finding was the extreme and usually general convolutional atrophy. There was no involvement of ependyma or glial reaction. In five out of his six cases there were no signs of cerebral arteriosclerosis.

In all the wasting of the brain had been so gross as to destroy the basal ganglion pattern.

Microscopically, in one case he found evidence throughout the brain and cord of cell degeneration and minute hemorrhages in all areas; in another of cell degeneration and vascular engorgement without hemorrhage. He suggests that the meningeal changes recorded at autopsy in cases of Huntington’s chorea have been due to co-existent pathological conditions, and points out that a disease so markedly hereditary is not likely to be a meningoencephalitis.

P. W.

[14] **Cerebral tumours without papilloedema** (Tumeurs cérébrales sans papille de stase).—**MARTIN and VAN BOGAERT. Jour. de Neurol. et de Psych., 1927, xxvii, 756.

Eight cases are described in which tumours were present in widely different parts of the brain, papilloedema being absent until a late stage. The authors recommend Baillart’s plan of measuring the pressure in the central artery of the retina when increase of the intracranial pressure is suspected.

J. P. M.


A report is given of a non-hair-containing, epidermoid cholesteatoma, unique because of its unusual size. The tumour weighed 69.5 grm. and measured 4 by 5.5 by 8 cm. Its exterior had the lustre of mother-of-pearl and was composed of multiple nodules, each of which was made up of laminated waxy material. These laminae were undoubtedly layers of flattened cornified squamous cells, somewhat resembling those of the epidermis. The tumour originated from the pia mater at the cerebellopontine junction and flattened the cerebellum, pons and cerebral peduncles.

R. M. S.


The authors report a case of infection of the brain and meninges by **Torula histolytica**, a yeast-like organism which reproduces by budding in tissues and in cultures, but which differs from the true yeasts by not fermenting sugars.
The course of the illness was marked by an insidious onset, persistent severe headache, and slowly developed signs suggestive of brain abscess, some form of meningitis or possibly brain tumour. The diagnosis was established by culture of the cerebrospinal fluid.

How the organisms reached the meninges could not be determined; possibly the infection came through the nasopharynx.

R. M. S.


The author analyses a series of fifty cases showing chronic manifestations. He found no evidence of constitutional or hereditary predisposition. Thirty-two of his cases gave a history of having had "influenza," that is, some illness in which fever, diplopia, somnolence, insomnia, delirium and ptosis were usually symptoms.

Motor disturbance predominated greatly over sensory. The only cases showing obvious autonomic disturbance were those with marked sialorrhea. In most of the cases the cerebrospinal fluid showed no change nor was the B.M.R. altered.

Mydriasis was noted in 19 cases, myosis in one, and anisocoria in eight. In ten the pupils reacted " sluggishly" to light, and in one they were fixed. No other eye signs are commented on, nor is any note made of the reaction on convergence.

From a prognostic point of view he considers the cases with the more violent onset to be more liable to develop ultimate Parkinsonism. Hyoscine in doses of 1/75 gr. t.d.s. is the drug of greatest value in treatment.

P. W.


Pardee describes four cases of Parkinsonism which showed oculogyric crises. In all the conjugate deviation of the eyes was to the left and upward. It was usually associated with a cephalogyric torsion movement of the head to the left. The crises in most cases appear two or more years after the acute attack.

In all cases there are associated hyperkinetic phenomena—torsion of head and neck, respiratory episodes, tongue spasms and so on. Other accompanying phenomena may be emotional outbursts, head pain, partial loss of consciousness and vegetative symptoms.

Pardee stresses the paroxysmal character of the crises. He concludes that the hyperkinesis of which this is a manifestation is produced by a subcortical, striatal, extrapyramidal mechanism. The conjugate deviation is dependent upon a posterior longitudinal bundle-anterior corpus quadrigeminal complex; the hypertonic crises together with other torsion phenomena bring these two mechanisms together, and point to a striatal-anterior
corpus quadrigeminum-posterior longitudinal bundle mechanism. Etiologically the condition is one of the later manifestations of post-encephalitis lethargica.

P. W.


Ten cases were investigated, and in most of them some slight hypo-excitability of labyrinthine functions was found. This is attributed to lesions of the vestibular nuclei and of the globus pallidus. The authors consider that the cause of oculogyric crises is possibly a transitory hyperæmia in the globus pallidus.

J. P. M.


The phenomena of forced movements readily obtainable in the experimental animal are practically never found in man. Unilateral section of a posterior longitudinal bundle invariably results in circus movements on the sound side and rolling movements to the affected side. The direction of these two forced movements is reversed if the posterior commissure is implicated. Evidence suggests that lesions of the nucleus of the posterior commissure are responsible for the circus movements while lesions of the nucleus interstitialis account for rolling movements. Fibres fine in calibre, descending from the posterior commissure, send collaterals to the third and sixth nuclei, and coarser fibres of the interstitialospinal tract join the fourth. With involvement of these collaterals the phenomenon of skew deviation may be associated. The globus pallidus is in relation to the region of the posterior commissure, but lesions at levels between the two do not appear to alter the direction of the forced movements obtained as is mentioned above. It is probable the connection of globus pallidus and commissural nuclei is double, so that reported cases of conjugate deviation of eyes and head in striatal lesions may thus be explained.

Certain symptoms of postencephalitic Parkinsonism deserve consideration along the same lines. Paralysis of conjugate deviation occurs with some frequency, and paralysis of vertical movement also; the author states that the "anatomical localisation of these lesions is unknown," which is a somewhat misleading comment, and evidently favours the view that supravestibular connections are involved.

A discussion of oculogyric crises follows. In the author's opinion "lesions corresponding to oculogyric crises should be found in the regio subthalamica or locus niger in front of the nuclei of the commissure." In this region the fibre-systems lesions of which cause forced movements in the vertical or horizontal
plane are sufficiently close to each other. Spasmodic movements of eyes in an upward or lateral direction are said to be of supranuclear vestibular origin.

S. A. K. W.


At the beginning of this paper five cases are described which presented more or less vague symptoms suggestive of inflammation of the spinal cord or peripheral nerves; in four of them there was also slight diplopia; all ended in recovery within a few weeks. In none of them was there any prolonged sleep nor is any satisfactory evidence given that the illness in any resulted from the infection of epidemic encephalitis, as the authors guardedly suggest.

In the discussion of the diagnosis of these cases three other cases are described which might equally well have been regarded as resulting from the same infection. One of them subsequently turned out to be a case of disseminated sclerosis, while in another no diagnosis was arrived at, even post-mortem.

J. P. M.


Two cases of infundibular tumour are described.

The first gave rise to an adiposo-genital syndrome with disturbance of glycoregulation; this was soon complicated by a thalamic syndrome and later by a Parkinsonian syndrome. Anatomical study revealed a large tumour of the adamantinoma type which had apparently developed at the expense of the original superior epithelial cells of the infundibulum; from there it had invaded the tuberan floor, filled the cavity of the third ventricle and pushed the thalamic masses aside laterally; it had invaded the lateral ventricles, which it distended greatly, and had pushed aside the white substance of the frontal pole and the central gray nuclei. Histologically, the globus pallidus showed marked lesions; the pituitary gland was intact.

In the second case, the development of a syndrome of intracranial hypertension led rapidly to a thalamo-infundibular syndrome. The infundibular symptoms were polyuria, disorder of glycoregulation and crises of narcolepsy with onirism, fabulation and retrograde amnesia. The thalamic symptoms were pains, hyperaesthesia to cold and trophic changes in the extremities. The extrapyramidal syndrome was represented by slowness, retropulsion and tremor. X-ray examination confirmed the existence of a tumour above the pituitary gland.

The author concludes that the thalamic syndrome must be referred to destruction of the internal nucleus of the thalamus; the true Parkinsonian
syndrome, to a lesion of the globus pallidus. Some extrapyramidal disturbance must be attributed to functional troubles in the frontal pole as the result of dilatation of the lateral ventricles from neoplastic extension.

During the infundibular phase glycoregulation is, as a rule, disturbed, and may be diminished. The meaning of this is not established. The endocrine disturbances are almost always characterised by an adiposogenital dystrophy which may later evolve into a marked cachexia.

In the first case the histopathological integrity of the pituitary gland is of biological interest in relation to the disturbance of glycoregulation, the disorder of fat metabolism and the regulation of sexual function.

R. M. S.

[23] An analysis of abnormal posture of the head in Parkinsonism of various origins.—W. M. KRAUS and N. E. SILVERMAN. Arch. of Neurol. and Psychiat., 1928, xix, 301.

In 1927 Kraus introduced the term “dissolution of erectness” to describe the posture in Parkinsonism, which is usually a combination of mask-like facies, forward flexion of the head and trunk, flexion at the hips and knees, adduction of the arms and flexion of the elbows and fingers. This classic dissolution of erectness does not, however, always occur in all details, and in the present paper the authors have analysed the various atypical positions of the head which may be encountered. For this purpose lateral views of patients with Parkinsonism were carefully traced from photographs, and two lines were drawn, one from the external meatus to the outer canthus, another from the external meatus through the trunk and crossing a horizontal line corresponding to the floor. The angle made by the meatus-canthus line with the meatus-floor line is called the head-trunk angle. The angle made by the meatus-floor line with the horizontal floor-line is called the trunk-base angle. The normal head-trunk angle is 110 degrees; the normal trunk-base angle is 90 degrees.

In a group of seventeen cases of Parkinsonism the trunk-base angle was always less than 90 degrees, irrespective of the position of the head. The head-trunk angle varied from the normal in either direction; to 154 degrees in extension, and to 71 degrees in flexion.

In addition to the dorsal and ventral flexion of the head, there is, as a rule, lateral flexion and rotation. Thus the flexion forward of the head, such as occurs in pure dissolution of erectness, is obscured by these deviations in all three planes. An increase of the head-trunk angle has not been found in the degenerative types of cases. It is common in the encephalitic type.

R. M. S.

This is the record of a remarkable case. The first symptoms were frequent epileptiform attacks, and soon after the onset of these the patient began to increase rapidly in weight. Eighteen months later he was found to have hemianopia (he had only one eye), and a commencing atrophy of the optic disc. He had become sexually impotent. Sugar tolerance was greatly increased. In the next few months he developed intense pain in the left half of the body, and hyperalgesia, and became blind. Two years after the onset a Parkinsonian syndrome came on and the patient began to suffer from cachexia. Finally he had paralysis in flexion.

At autopsy a large suprahypophyseal tumour was found which seemed to have grown from the infundibulum and spread thence along the floor of the tuber cinereum, filling the third ventricles, pushing the thalami laterally; it had invaded the lateral ventricles and caused great enlargement of them, damaging inter alia the globus pallidus. The tumour resembled the hypophyseal adamantinomata but was of mixed structure. The pituitary was histologically intact.

J. P. M.


The writer reports a case of this syndrome occurring in a young married woman of 34. The first symptoms which appeared five days after a miscarriage were a peculiar feeling down the whole right side of the body, and inability to speak for two hours. Ten days later she noticed that the right leg dragged in walking. This lasted for a year, during which time the whole right side felt "as though asleep." There was no hemiataxy or hemiathetosis, but spontaneous paroxysmal pains occurred on the right side.

There was some impairment of stereognosis in the right hand, and slight loss of postural sense. Deep pain and vibration sense were considerably diminished in the right hand. Light touch was felt as a painful stimulus all over the right side. There was no sphincter disturbance. She had a high blood pressure but no signs of renal disease. The blood Wassermann reaction was negative.

The author comments on the rarity of the syndrome as early as 34, and explains this on the ground that vascular changes in the thalamus are its commonest cause. He suggests an embolus following on uterine thrombosis as the cause in his case. The associated signs of involvement of surrounding structures which are almost always found, and are probably due to oedema, were absent in this case. Spontaneous pain he considers due to pressure caused by gliosis of the infarcted part on incoming thalamic fibres. P. W.
Postural variations in nystagmus (Quelques mots au sujet du nystagmus de position).—Buyx and Hennebert. *Jour. de Neur. et de Psych.*, 1928, xxviii, 147.

In 1913 Bárány described two cases in which nystagmus was induced or varied according to the position of the patient’s head in space. The first was that of a woman with disseminated sclerosis in whom a horizontal nystagmus towards the left was present as long as she lay with the right side of her head on the pillow. The second was that of a man who if he lay with his head turned to the right showed rotatory nystagmus on deviating the eyes to the right and vertical nystagmus on deviating them to the left. Bárány showed that the occurrence of the nystagmus depended on the posture of the head, and was not affected by the posture of the neck (relation of head to body).

Since then numerous similar observations have been recorded. Buyx and Hennebert at a discussion in the *Groupement Belge otoneuro-ophtalmologique* emphasise that there are many sources of error to be guarded against, of which the chief is that nystagmus may be caused by displacement of the head relative to the trunk, or by the movement involved in bringing the head into the new position for examination. A case in which nystagmus resulted from changing the position of the head relative to the trunk has been recorded by de Kleyn and Versteegh. In some other recorded cases nystagmus manifested itself both after changing the position of the head in space and after changing it in relation to the trunk. It has been shown that in animals rotation of the head on the trunk may interrupt the circulation in one vertebral artery and thus in occasional cases it may induce circulatory disturbances in the labyrinth on the same side and consequently nystagmus. Variations of the positions of the head relative to the body must therefore be avoided in examining for real postural nystagmus. Secondly the movement of changing the position of the head must be performed very slowly.

The nystagmus seen in these peculiar cases is usually rotatory on deviation of the eyes to the lower side and vertical on deviation towards the upper; it usually appears after a short latent period and persists only a few seconds or minutes; its amplitude is small and its occurrence with successive trials is in many cases inconstant. There has been a tendency to connect the origin of this postural induction of nystagmus with the labyrinthine postural reflexes on the eyes described by Magnus and de Kleyn. This view was combatted by Professor Quix, who pointed out that these latter reflexes were tonic or lasting reactions. Quix attributes the postural induction of nystagmus in certain cases to a functional disturbance of the semicircular canals which is accentuated in certain positions by the normal action of the otoliths on the macule of the saccules. Quix has described an apparatus in which patients can be examined with the head in any angular position in space; it consists of a large circular frame mounted at each end of its horizontal axis on a stout tripod, and capable of being rotated round this axis and fixed in any position;
within this frame on a diameter at right angles to the axis of rotation, a much narrower oval frame, like the top of an operating table, is fixed, and this is capable of rotation round its long axis and can be fixed in any position. The patient is fastened on this smaller frame in such a way that he feels perfectly secure. The pressures of the otoliths have been calculated and their pressure for the head in any given angular position can be expressed as a percentage of their maximum pressure.

J. P. M.


To the commoner symptoms of insulin shock the authors add certain hitherto unrecorded signs of nervous system involvement. Their patient had been having 90-100 units of insulin a day before entering hospital.

While in hospital he manifested, in addition to the usual symptoms of insulin shock, speech disturbance, some degree of amnesia, and generalised tonic-clonic convulsions in one of which he bit his tongue. In mild attacks his vision was blurred; in a severe attack the pupils became dilated and inactive to light. After one severe attack with convulsion, he showed a right hemiparesis and a diminution of the abdominal reflexes. They adduce evidence which goes to show that hypoglycaemia is not the only factor producing shock symptoms, for these may come on in the absence of hypoglycaemia and be relieved without glucose therapy.

They also suggest that shock symptoms are more prone to occur in persons with un-stable sympathetic systems, and that the reduction in the inorganic phosphates and potassium of the blood caused by insulin may be an additional factor in producing such symptoms. They draw a tentative parallel between the disturbance of intermediary metabolism known to occur in tetany, and this condition.

P. W.


In the case reported by the authors the outstanding clinical features were the manifestations of increased intracranial tension, including almost all of the cardinal signs and symptoms of cerebral neoplasm, such as attacks of giddiness, intense headache, projectile vomiting and papilloedema. These signs and symptoms, with the gradual unfolding of the clinical picture, interrupted by slight remissions, and the absence of febrile reaction, justified a diagnosis of tumor cerebri. In the absence of well-defined focal signs the bilaterality of symptoms suggested a median line location, possibly between the hemispheres. Ventriculographic studies and the injection of dye revealed the presence of bilateral, symmetrical dilatation of the lateral ventricles and demonstrated conclusively the lack of intercommunication between the third and lateral
ventricles. These observations definitely localised the lesion and placed it in the third ventricle, but the true nature of the disease was not recognised during the life of the patient.

Microscopic investigation indicated the presence of an inflammatory process limited mainly to the ependymal lining of the entire ventricular system of the brain. The mild inflammatory and degenerative changes seen in the adjacent brain tissue were little more than a reaction to a contiguous inflammatory process. In the widespread ependymitis there were present all gradations, from the mildly acute purulent in the lateral ventricles to the chronic granular ependymitis in the aqueduct and the fourth ventricle. The third ventricle was occupied by a pseudocyst formed of thickened ependyma.

The presence of several transitional stages in the inflammatory process in one and the same case supports the belief that many of the so-called chronic forms of ependymitis, including granular ependymitis, especially when they are associated with an inflammatory process elsewhere in the central nervous system, as in general paralysis, are the results of a slowly progressive form of inflammation of the lining of the ventricular system.

R. M. S.


The authors have studied the nature of the responses that normal persons give when the tests used in the study of aphasia are applied to them. The control material consisted of two main groups: persons with superior and average intelligence without organic nervous disorder, and persons with organic brain disease of different types. The subjects in both these groups were without speech disorders. In selecting the control material, an attempt was made to obtain young persons without nervous disease, in order to perceive what reactions healthy, non-aphasic persons would give. In addition, however, patients with organic brain disease but without disturbances of speech were selected in order to approach more closely the condition of the aphasic patient who has brain disease of a highly localised nature. The tests employed were those devised by Henry Head.

It was found that the normal person often gives responses which are exactly the same as those obtained in cases of aphasia. In some tests the errors were so frequent and coincided so closely with those in aphasic cases that it was deemed wise to discard these tests. Although it is impossible to separate in any way the function of speech from intelligence, the results with Head's tests indicate that they investigate intelligence to a far greater degree than they do speech, and the conclusions drawn from them must stress, automatically, the loss in the former sphere rather than in the latter. The authors conclude that any other series of tests would have given identical results,
but they consider the tests used by Head to be as good as, if not better than, any others that have been devised for the testing of aphasia.

R. M. S.


The author records five cases of pellagra. He discusses briefly the various theories as to the mode of origin of the disease, and adopts the vitamin deficiency hypothesis.

He points out that the diagnosis cannot certainly be made without the triad of alimentary, cutaneous and nervous symptoms. The rash is the most important diagnostic sign, and he stresses its symmetry, its sharp delimitation, the branny desquamation and its predilection for certain sites. The subsequent pigmentation tends to be chronic and patchy.

That the cerebrospinal fluid is normal in spite of the gross cellular changes in the cord is against an infective hypothesis. Amnesia, mental retardation and tremor are among the early mental symptoms; the later are usually melancholia or confusional insanity with suicidal tendencies.

In most of his cases he was able to find evidence of faulty diet, and better dietaries, including eggs, fresh meat and milk and green vegetables caused immediate improvement. The common occurrence of the disease in institutions and among the poorer classes is correlated with dietetic faults in both cases.

P. W.


A case of actinomycosis of the right lung and right posterior chest wall is described, in which disturbances of function of the thoracic sympathetic chain occurred: they were probably irritative. The area in which sympathetic changes were manifested included the right side of the face and neck, the entire right upper extremity and the right side of the chest down to the twelfth rib. The midline and the line of the twelfth rib formed sharp boundaries.

The sympathetic changes were indicated by localised hyperidrosis, appearance of goose-flesh, pilo-erection and unilateral nipple erection, an exaggerated response to the drawing of a pin across the right side of the front of the chest, a lowering of the temperature, with pallor of the right hand as compared with the left, and bizarre responses of the right upper extremity to extensive changes in temperature. In a general way, the reactions of the right arm and hand were the opposite of those of the left.

No difference between the right and the left upper extremities were observed in blood pressures, growth or condition of the hair or nails, reactivity of muscles or nerves to electrical stimulation, nor in their reactions to epinephrine.

Permission for an autopsy could not be obtained. R. M. S.

Working at the psychiatric clinic of the university of Minsk, Lowenberg had the opportunity of studying the microscopic appearances in four cases of rabies, and in general was able to confirm the observation of Schukri and Spatz that the inflammatory changes in rabies are of a type and localisation quite similar to those found in cases of lethargic encephalitis.

The brunt of the attack seems to be borne by the substantia nigra and the substantia centralis grisea and, to a lesser extent, by the pontine nuclei. In all of these the reactive phenomena are strikingly localised about the parenchyma, the nerve cells being largely destroyed and accompanied by neuroglial proliferation and neuronophagia. In the cortex, basal ganglia, white matter and cerebellum the lesions are of a predominantly degenerative character.

This picture was especially characteristic of the author’s first two cases, whereas cases 3 and 4 showed some deviations. In case 3 the picture was that of a meningitis, there being leucocytic infiltration, marked oedema and occasional haemorrhages. In case 4 the appearances were those of an acute purulent meningitis, but without abscess formation. These four cases demonstrate that there is a relation between the length of the period of incubation and the character of the lesion, for the shorter this period, the more acute and intense is the process. Thus, in the first two cases, with incubation periods of over six months, there was no meningitic involvement and the degenerative process was more distinct. In case 3, with an incubation period of a little more than a month, the inflammatory reaction was more pronounced, while in case 4, in which the incubation period was only nineteen days, an intense inflammatory process was present.

A further point of interest, which appears to have escaped the author, is the relation of the site of the infected wound to the incubation period. In cases 1 and 2 the bites or scratches were inflicted on the hands, in case 3 on the hands and chest, and in case 4 on the face. The short incubation period in cases 3 and 4 is exactly what one would expect in a disease having all the characters of a lymphogenous infection of the nervous system.

R. M. S.


Dr. F. Negro claims to be able to demonstrate the occurrence of the ‘cog-wheel’ phenomenon in various syndromes distinct from those of Parkinsonism, in which it was described originally by C. Negro in 1902. In these its degree is less, yet it is distinctly recognisable. His argument is to the effect that the phenomenon is the outcome of sarcoplasmic rigidity or hypertonicity; assuming
that the sarcoplasm is innervated by the parasympathetic, he states that the supposition would be that sarcoplasmic rigidity follows from the stimulating action of thyroid secretion on the parasympathetic; were this the case, however, it should be more pronounced in vagotonic than in sympathicotonic states. But the reverse is the case. The conclusion that sarcoplasmic innervation must be from the sympathetic and not the parasympathetic seems the natural one to make. The author, however, brings forward evidence suggesting that the thyroid secretion has a double effect, on the two divisions of the system.

J. V.

[34] Haemangioma of the spinal cord.—C. W. Rand. Arch. of Neurol. and Psychiat., 1927, xviii, 755.

The author reviews the literature of twenty-one cases of haemangioma of the spinal cord, and describes two new cases.

The majority of haemangiomas have been found on the dorsal aspect of the cord and it is supposed that they originate from the pial veins. They may be seen in the cervical region, although the lower dorsal and lumbar regions are the locations of choice. The vessels may enlarge to enormous proportions and may present a nest of greatly dilated, closely packed, interwoven veins which compress the underlying cord at times almost to a point of flatness. On occasion, the varicosities may extend into the cord itself, and the nutrition of this structure becomes so impaired that areas of softening result.

Spontaneous rupture is prone to occur and is cited as the most common cause of sudden paraplegia. The rupture may be free into the spinal canal, or it may be intramedullary. Thrombosis has been reported in a number of instances and is perhaps more common that spontaneous haemorrhage. It may affect the vessels within or without the cord proper. In certain instances the blood supply to the surrounding vertebrae may be greatly increased, and the veins of the adjacent soft tissues dilated almost to angiomatous proportions.

A clue to the diagnosis is sometimes afforded by the presence of a nævus or multiple nævi in the dermatome supplied by the cord segment from which the pial haemangioma arises, and when present it is deeply pigmented.

Surgical intervention offers little hope of improvement in these cases.

R. M. S.


A case of malignant chordoma at the third lumbar vertebra is reported. This is the second case of its kind out of fifty-eight published case reports.

Tumours of the notochord are difficult to differentiate from myxoid chondroma and colloid carcinoma of the intestinal canal. Usually chordomas are soft and gelatious in consistency, and clinically are often mistaken for abscesses, but the tumour described in this paper was of fibrous consistency.
and of a mixed character. In its outer layer it resembled a sarcoma, but the physaliphorous cells and the staining quality of the matrix characterised the tumour as a chordoma.

R. M. S.


No one has done more than Lhermitte to throw light upon the various types of senile paraplegia. Over twenty years ago, in collaboration with Lejoinne, he isolated a clinicopathological group in which muscle changes alone occupy the picture. Under the title of "myosclérose rétractile" (or "myopathic primitive sénile") Lhermitte refers to a type of slowly progressive paraplegia without any corresponding affection of the central or peripheral nervous systems. Despite its frequency in institutions for the aged, but little attention has been paid to this condition. The onset is usually seen in extreme old age; women are affected more often than men. Pains, cramps and other dysaesthesiae localised to the calves usually constitute the first symptom. Later appears difficulty in walking and some months afterwards the patient became bedridden. Thereafter the course is more rapid; muscles waste and shorten, and the lower limbs assume an attitude of flexion. At first the legs can be passively extended, but in time this becomes impossible even under anaesthesia. The first picture is that of extreme paraplegia in flexion; the muscles are profoundly wasted and feel hard on palpation; the tendons stand out like violin strings. Both active and passive movements are impossible, and attempts at manipulation are very painful. There is no marked change in the electrical reactions, but mechanical excitability is increased and myoidema is constant. The upper extremities and the neck and trunk usually escape. Cremasteric and abdominal reflexes are absent; the knee jerks are often sluggish and it may be difficult to elicit an ankle jerk. There is never a Babinski response. Lhermitte discusses the differential diagnosis from arteriosclerotic rigidity, with which indeed it is frequently associated. Jakob's disease, or senile *Vorsteifung*, is regarded by Lhermitte as merely a variety of arteriosclerotic rigidity. Histological changes in senile myosclerosis are profound; they comprise atrophy, pigmentation and plasmoidal regression of the muscle-fibres with fatty, pigmentary and other types of degeneration; colossal increase of the inter- and intra-fascicular connective tissue, with replacement of muscle fibres by fatty or fibrous elements; some of the fibres are hypertrophied. The lymphatic spaces are distended, and there are accumulations of small round cells. The genesis of the muscle changes is thought to depend not solely upon an inadequacy of the peripheral circulation but to embrace also a stagnation in the lymphatic
flow from the muscles, together with a disorder of general nutrition. These factors are exaggerated by the forced immobilization of the limbs.

The paper is finely illustrated.

M. C.

[37] **Vasomotor and pilomotor manifestations.—**TEMPLE FAY. *Arch. of Neurol. and Psychiat.*, 1928, xix, 31.

The author calls attention to the localising value of vasomotor manifestations in lesions of the spinal cord.

The patient is placed in diffuse daylight of moderate intensity, the possibility of shadows being avoided, the covering is quickly removed, and the entire body scanned carefully. This is best done at a distance of from three to six feet from the patient, and careful attention should be paid to the presence of areas of hyperaemia, which are suggested by a broad band of flushing about the chest and abdomen, demarcated above by the normal flush of the body. The direction of this band of vasomotor manifestation is in striking accord with the segmental zones of Head. Several variations of the phenomena may exist, and all have been found to be of equal localising significance, indicating an abnormal instability in this area. The skin area above and below the line of demarcation may vary in texture and in the degree of light reflection, if the patient is observed from an angle such as from the head or from the foot of the bed. The actual line of demarcation may assume a feather-edged appearance and may be no larger than the diameter of a piece of twine, extending in a serpentine fashion at right angles to the axis of the body until the axillary line is reached, and then coursing upward at an angle of about 30 degrees toward the spinous processes. The zone of demarcation in cases in which the history indicates a long-standing involvement of the cord may simulate the difference between a slight sunburn and normal skin, so that the area below the level of the lesion appears to be slightly pigmented, and this effect may extend over a large area of skin below the line of demarcation, giving the appearance that the patient had been over-exposed to the weather.

The examiner will be struck by the rapid evanescence of the vasomotor reaction in the zone under observation. During the initial period of observation a flushing should appear at the level noted, similar to that seen in slight hyperaemia following the application of heat to the skin. If the patient is exposed to a draught or cold, it may be noted that this area blanches rapidly, giving rise to an apparent ischaemia or to a zone distinctly white in comparison with the normal areas above. Confirmation of the level of vasomotor demarcation can be intensified by heat, and the zone will be found to coincide exactly with the first signs of sensory change. Pilomotor manifestations can be obtained by flicking the skin with a wet towel or by the application of a current of cold air. Similar results can be obtained by deep pinching of the skin and the trapezius muscle at the base of the neck; this produces a homolateral reflex
spreading rapidly down the same side to include the lower extremities. This is a test for function of the cord and may give the level of the lesion by failing to appear below the involved segment.

In the author's series of thirteen cases the vasomotor line of demarcation proved to be the most reliable sign, and in each case it determined the exact level of localisation.

R. M. S.


DR. WARTENBERG's long and valuable discussion of problems connected with the function of dorsal and ventral spinal roots does not lend itself to a convenient summary. It is replete with analyses of numerous personal and recorded cases, and covers various separate if cognate questions each one of which is more or less controversial. His investigations have extended over a number of years and concern diverse clinical material. The paper is full of useful references to the literature.

The author is at pains to impress the reader with the uncertainty attending many of the "conclusions" that have hitherto been utilised as data on which to base current conceptions of the function of anterior and posterior roots. Some of his own decisions may be briefly indicated.

After section of posterior roots (several consecutive roots on one side) all forms of cutaneous sensibility are lost, but a degree of deep sensibility remains. To determine its presence the examiner must use stimuli of sufficient strength and duration. It appears definite enough that this deep sensibility is not as a fact identical with that which can be demonstrated on the normal side; sensations of pressure and of pressure-pain arise more slowly than normal, and tend to remain for an appreciable moment after cessation of the stimulus; they have a high threshold; as a rule, the sensation is less painful than normal, more diffuse, more blunt—in a word, it is but a fraction, so to speak, of normal deep sensibility. Yet stimuli have local signature. It undoubtedly presents in these respects some resemblance to so-called visceral sensibility; a summation of stimuli is requisite, in regard to both number of end-organs stimulated and duration of stimulus. The author is evidently impressed with this resemblance, which embraces both clinical characteristics and conjectural site of origin.

A further conclusion is to the effect that such excitations must reach the cord by anterior roots. Other possible routes are examined at length, but rejected for want of evidence. The author exercises admirable scientific restraint when he says that the path may be by anterior roots, not must. In different parts of the same nervous system, and for different individuals, the amount of sensibility thus conveyed by anterior spinal roots or cranial nerve analogues varies materially.

S. A. K. W.
Abstracts


The authors record the case of an infant which recovered from icterus neonatorum gravis. The infant was the third child of the family. The two elder children had died of the same disease. The delivery of the third infant was normal in every way, but it was deeply jaundiced within twenty-four hours of birth. This jaundice persisted up to five weeks of age, during which time, although the stools and urine contained bile pigment, the child’s weight curve was normal for its age. There was no fever. The Wassermann reaction of the mother and child was negative. Bleeding and coagulation times were normal.

At six months the child could not support its head and showed spasticity of the thigh adductors. At two years old the child was idiotic in appearance, could only utter a few monosyllables, and could only walk on its toes with support. The gait was spastic and the plantar double extensor.

The authors review the suggestions as to the pathogenesis of this condition, and feel that sepsis and maternal toxæmia are not likely to have played any part in their case. They quote the results of autopsy which have been recorded up to the present. Necrosis of the ganglion cells of the basal nuclei and round-cell perivascular infiltration were found in two cases by Hoffmann. Icteric pigmentation and necrosis of the ganglion cells of the basal nuclei, the medulla, the olivary bodies and the gray matter of the anterior and posterior horns of the cord have been found in newborn infants dying of the disease. Pigmentation is not, however, constant. The liver, in some cases, has shown diffuse necrosis and destruction of cells most marked in the centre of the lobules. Cerebral hæmorrhage or injury was found in no case. The two outstanding features in symptomatology are motor disturbances of different kinds and intellectual defect.

The authors follow Hoffmann and Hausmann in believing that cerebral changes only occur where there has been definite necrosis of liver cells. Cholæmia as such cannot produce these changes for they are not found in congenital atresia of the bile ducts, or in all cases of icterus gravis. They support this view by adducing the cerebral symptoms which occur in acute yellow atrophy, and the brain changes found in infants suffering from septic icterus and septic nephritis. In both these conditions necrosis of liver cells does occur.

They therefore consider, that icterus neonatorum gravis must be regarded as a possible cause of some cases of cerebral diplegia.

P. W.


The author recognises two forms of myatonia congenita—the commoner occurring in one member of a family and causing early death; the other being
one-tenth as common, familial, and benign in that the patients survive to puberty and may even recover.

He records a case of the latter type, that of a boy, age seven. The parents were healthy but the mother had had one anencephalic infant, and one stillbirth at three months. The patient was the third child, the two next children were healthy, and the sixth pregnancy resulted in a miscarriage at eight months.

The patient was never able to hold his head up properly and had always walked with a waddle from the age of 14 months. He talked normally and was mentally up to the average. The head was held constantly retracted owing to absence of the sternocleidomastoids and overaction of the posterior nuchal musculature, which was in a state of contracture. Cephalic rotation was almost impossible; the child moved his eyes only to look to the sides, and he had to use his hands to bend his head forward. This contracture readily accounts for his waddling gait, and the constant gape. No microscopic examination was made of the neck muscles.

The other features on which the diagnosis was based were the generalised small musculature, the muscular flaccidity, the absence of all deep reflexes except on reinforcement and the abnormal mobility of all joints. The author brings the neck contracture into line with the talipes equinus and the lamb contractures so often seen in amyotonia congenita, but he considers it to be thus far unique.

P. W.

[41] Toxic infectious disease in the peripheral and central nervous system—so-called neuronitis.—S. Brock and M. Ivimey. Arch. of Neurol. and Psychiat., 1928, xix, 129.

A series of five non-fatal cases is presented, illustrating toxic or infective processes involving the peripheral nerves and the central neuraxis. This motley group, with a varied etiology, showed a mixed symptomatology referable to the peripheral and central nervous system.

The authors suggest a nomenclature in which the term neuritis is retained—peripheral neuritis for processes limited to the distal parts of the nerve trunks; radiculitis, for involvement of the roots, and central neuritis (spinal, medullary, pontine, etc.) for involvement of the neuraxis. When more than one part of the nervous system is implicated, these terms may easily be compounded.

R. M. S.


The case reported appears to be the first clinical instance recorded of spinal haemorrhage in thrombopenic purpura.

The patient was a man of 30, with a pronounced haemorrhagic diathesis (full details given), in whom a flaccid paralysis of both lower extremities and of the muscles of the abdomen, as well as loss of sensibility up to the second
rib, set in suddenly without ascertainable cause. During six months of observation the condition passed from the stage of flaccid paraplegia, through spasticity with flexion contractures, to spasticity in extension.

The pathological diagnosis is discussed at some length and finally regarded to be one of hæmatomyelia. The findings in the spinal fluid were curiously indefinite. As for the blood picture, its features were: small number of platelets (less than 35,000); non-retraction of the coagulum; prolonged periods of bleeding; almost normal coagulation-time; positive stasis test; extravasations of blood on contusion—all typical of essential or benign thrombopenia.

J. V.


The authors determined the incidence of Chvostek’s sign in a series of 55 infants ranging in age from 12 hours to 21 days. All the infants had a positive Chvostek’s sign. They considered the sign positive when a contraction was obtained at the lip, alae nasi or forehead.

Blood calcium estimations were normal in all cases, and other signs of tetany such as Schultz’e’s, Lust’s and Trousseau’s were not elicited.

The authors stress the fact that the sign cannot be elicited in a restless or crying infant.

They conclude that as Chvostek’s sign can be demonstrated in newborn infants with no evidence of tetany, it is not to be looked on as pathognomonic of this condition in early infancy.

P. W.


The author records the case of a female infant, age three-and-a-half months, showing unilateral phrenic palsy. The labour had been difficult, a breech presentation in a primipara, and the infant sustained a fracture of the left humerus and a right-sided Erb paralysis.

On examination he found the respirations rapid and irregular, and the middle of the sternum retracting with each inspiration. The right half of the chest was flattened and moved less well than the left. There was dullness at the right base. The abdominal respiratory movements were completely absent on the right side, and an X-ray examination showed a paradoxical ascent of the right half of the diaphragm in inspiration.

The child was watched for two and a half years during which time it had three attacks of right-sided bronchopneumonia.

The author reviews the literature on phrenic paralysis. Few cases due to injury are on record owing to the well protected position of the nerve in the neck,
Obstetric injury to the phrenic nerve was first recorded in 1921 by Kofferath. It has never been seen without concurrent injury of the brachial plexus, and recovery has occurred in the other two recorded cases.

The symptoms are all referable to unilateral diaphragmatic paralysis, but are more severe in the newborn as a state of atelectasis exists on the affected side.

P. W.

[45] **A case of hemihypertrophy (Un cas d’hemi-hypertrophie).—**VERMEYLEN. *Jour. de Neur. et de Psych.*, 1928, xxviii, 103.

The recorded cases of hemihypertrophy number 53. To these Vermeylen adds the case of a young girl of 12 in whom the left side was considerably larger than the right. This hemihypertrophy involved face, ear, tongue, neck, breast and limbs. The hemicircumference of the trunk on the left side was six centimetres more than on the right, the circumference of the left calf and that of the left thigh were each four centimetres greater than the corresponding measurements on the right. The left leg was six centimetres longer than the right. The hands and feet and genital organs showed no asymmetry. The girl was mentally defective, her intellectual development being that of a child of seven.

The most probable explanations of hemihypertrophy are (1) that it results from a tendency to twinning in the ovum (Gesell), and (2) that it is a consequence of an abnormality in the development of the sympathetic system on one side (Leustrup).

J. P. M.


The author describes his investigations in a series of 35 cases of migraine. Cases which showed other pathological changes were excluded.

A very complete examination was made, and in 32 out of his 35 cases he demonstrated a bilirubin retention varying from 1 to 5 units as estimated by the van der Bergh reaction. The urobilinogen in the urine varied, but was found constantly raised in those cases with a high bilirubin retention.

Most of his cases gave a familial history and in most the headaches had begun in the first or second decade. All showed headache of the hemicrania type.

The van der Bergh reactions were lowest in the purely cephalic type, higher in those showing intestinal disturbance, and highest in the cases of abdominal migraine. In the latter cases, the symptomatology is identical with that of cephalic migraine in the early part of the attack; intense epigastric pain and vomiting of bile characterise them in their later stages.
All his cases showed a low systolic blood pressure, 90-110, and constipation.

He considers that a failure on the part of the liver to synthesise and detoxicate the amino bodies formed during digestion leads to a series of anaphylactic changes in the cerebrum and other viscera analogous to those occurring in Quincke’s oedema. Treatment should be directed to overcoming the constipation, and protecting the liver by avoiding animal protein and giving a lacto-vegetarian diet.

P. W.

PROGNOSIS AND TREATMENT.


The author reports a case where signs of chronic arsenical poisoning followed on taking a dose not far short of the fatal one. Intense purging and collapse followed on the ingestion of the dose. Five days later arsenical dermatitis appeared, twelve days later paraesthesiae in the hands and feet and seventeen days later weakness in the arms and legs. Seven months later there was still definite objective sensory loss in the hands and feet, tenderness of the calves on pressure, the knee jerks were absent and there was wasting of the small muscles of the hands and of both thighs.

Arsenic was found in the urine. The patient was given 16 injections intravenously of 0·75 gr. sodium thiosulphate on alternate days. Arsenic had disappeared from the urine within three weeks, and at the conclusion of treatment he was practically normal.

The rationale of the treatment is that the sodium thiosulphate forms a soluble arsenenate which is rapidly excreted, thereby displacing the relatively insoluble arsenious acid compounds.

P. W.


Removal of the superior cervical ganglion and periarterial sympathectomy do not appear to be of much value in the treatment of atypical neuralgia, mostly in the trigeminal distribution. The author presents the data from ten selected cases, in five of which the sensory root operation had first been performed. In only one case did the patient obtain any relief, and a doubt remained as to whether the relief was permanent, as the patient could not subsequently be traced.

R. M. S.