ABSTRACTS

SENSORIMOTOR NEUROLOGY


Optic nystagmus (train nystagmus) is the name given to the movement of the eyes when a field of more or less heterogeneous objects moves in a given direction before them. It can readily be produced by means of a rotating cylinder with stripes or, preferably, pictures on it (Bárány). When the objects move from left to right before the eyes the nystagmus is to the left, with the quick component to the left. The subject is himself unaware of this movement of the eyes. The usual explanation offered is that the image of an object falls on the periphery of the retina and that the eye is at once moved reflexly to bring it into the macular field, the eyeball moving almost with a jerk in the direction of the object. Dr. Stenvers, however, throws doubt on the reality of this interpretation of the facts, principally because of the occurrence of cases of hemianopia in which, none the less, optic nystagmus with the quick phase to the hemianopic side is found. He cites an interesting series of some twelve cases of the phenomenon, in one of which (No. XI.) a tumour of the right occipitotemporal region was present; optic nystagmus was always obtainable to the right, but never to the left. The conclusion drawn is that it depends on a cerebral reflex, which is abolished by a lesion in the area above the angular gyrus, extending from the occipital pole frontwards. Interference with the optic radiations also disturbs the phenomenon. Several hypothetical considerations are offered to explain the physiology of the eye movement, but no definite conclusion is reached.

S. A. K. W.


Dr. Lutz reports a case of hemianopia in which all the clinical symptoms and signs indicated a lesion of the optic radiations and in which the pupillary inaction of Wernicke was nevertheless found, supposed to be characteristic of a lesion in front of the corpora quadrigemina. It is true the case did not come to autopsy, but on clinical grounds the author has no hesitation in making an exact diagnosis, and in denying, accordingly, the differentiating value of the Wernicke sign. He alludes to the cases of Walker, Dejerine and Jumentié, and others, which show in a definite way, on clinico-anatomical grounds, that the hemipic pupillary reaction of Wernicke, to give it its full name, cannot be used to separate an anterior from a posterior hemianopia.

S. A. K. W.

[54] Several cases of facial paralysis in children observed simultaneously, with remarks on the etiology (Plusieurs paralysies faciales observées simultanément chez des enfants; remarques sur l’étiologie).—P. NOBÉCOURT. Arch. de méd. des enfants, 1925, xxviii, 8.

Six cases were observed in Paris in the course of a few months. An analysis of the clinical features of these cases suggested that they might form successive
links in a chain of evidence bearing on the etiology. All the children were of the ages ten to fourteen. The cases were classified by the author into three groups:

1. A single case of Bell’s palsy of sudden onset without any other abnormality.

2. Two cases: (a) Right facial paralysis with frontal headache and pain behind the right ear; lumbar puncture not successful.
   (b) Right facial paralysis with pain in right ear, redness of external meatus, and small vesicles at the orifice. Increase of lymphocytes in cerebrospinal fluid.

3. Three cases which suggested infective conditions, and in which other disturbances of the nervous system were present.
   (a) Drowsiness, fever, slight paralysis of accommodation. Cerebrospinal fluid showed slight meningeal reaction.
   (b) Vomiting, fever, slight albuminuria, followed by choreiform movements lasting a few days. Cerebrospinal fluid: cells, 225; albumen, 0.85 per cent.
   (c) Fever, headache, vomiting; diplopia.

Professor Nobécourt considers that the cases of the second group are very suggestive of a herpetic origin. The association of herpes and facial paralysis has been discussed by Worms and de Lavergue, and Achard. The third group he regards as abortive cases of encephalitis or Heine-Medin’s disease. Thus of six cases, five appeared to be due to an infective virus, and the three in which the cerebrospinal fluid was examined showed evidence of meningeal reaction. For some years evidence has been brought forward by Dejerine and others that the so-called facial paralysis a frigore is, in fact, due to an infective process; the investigation of these cases appears to support this view.

N. Hobhouse.


The writer mentions three diagnostic features of temporal lobe tumours, on the basis of which he has made a correct diagnosis in several cases, confirmed by operation: (1) in the pointing test the hand on the side opposite to the lesion deviates towards the midline; (2) the patient tends to fall backwards and to the side of the lesion; (3) a unilateral syndrome of the globus pallidus appears on the side opposite to the lesion, consisting in pronounced ‘mimic facial paresis’ (i.e., the so-called Nothnagel’s syndrome).

Neurologists generally will be interested in this temporal ‘triad,’ for temporal tumours are often difficult to diagnose, especially those of the right side. The symptom (2) above, however, is frequently found in the case of tumours that have nothing to do with the temporal lobe; Nothnagel’s symptom is certainly not pathognomonic of pallidal lesions; and the pointing test deviation can be found in conditions whose connection with the temporal lobe is problematical. The simultaneous occurrence of all three in one case is doubtless of importance; cerebral tumours, however, are the least satis-
factory morbid states from which to draw conclusions as to localization of function.

S. A. K. W.


The author is sceptical of any interpretation of head attitudes in cases of cerebral tumour based on disorders of innervation mechanisms, holding rather that the determining factors of these postures are essentially mechanical. In tumours above the tentorium he finds that the head is commonly retracted, to allow cerebrospinal fluid to pass more readily from the third ventricle; in subtentorial tumours the head is usually bent forward, to enlarge the communication between the fourth ventricle and the cisterna, to make the latter bigger, and to prevent pressure on the medulla. Disturbance of innervation can be compensated for by cerebral activity, but these attitudes cannot so be corrected.

These views are interesting but are open to criticism.

S. A. K. W.


A young woman of nineteen had a sudden apoplectiform attack, with vertigo, vomiting, and loss of consciousness. Clinical examination proved negative. Three years later (age twenty-two) the same sequence of events recurred, with right hemiplegia and aphasia on this occasion. Moderate recovery ensued. Seven years later (age twenty-nine) a fresh apoplectic attack resulted in the death of the patient. A cavernoma (haemangioma) was found in the region of the left basal ganglia. During life none of the signs of cerebral tumour was ever discovered.

The article contains a useful discursus on apoplexy in young people, with numerous references to the literature.

S. A. K. W.


Much valuable clinical and pathological evidence is here collected which goes to substantiate the already well-known fact, that on the cortex the centre for thumb and fingers lies next to that for the corner of the mouth, lips, and cheek. In the author's case (clinical) it is of particular importance to note that apart from moderate subjective paræsthesiæ in these areas the objective diminution of sensibility implicated almost solely the sense of pain. Interesting data are supplied showing that corresponding motor and sensory centres exist on opposite sides of the central fissure, and that the above-mentioned thumb and lip area is in the distribution of a fine twig of the middle cerebral artery, the arteria interopercularis parietalis.

S. A. K. W.

The clinical and pathological features of this unusual case are of interest in the study of the effects of lesions in the corpora striata. In a man with right-sided hemiplegia of three years' standing choreiform movements suddenly commenced on the left or unparalyzed side. On section the brain showed a large area of thrombotic softening involving the greater part of the left putamen, globus pallidus, caudate nucleus and the knee of the internal capsule. On the right side a smaller and more recent area of softening involved chiefly the putamen and to a less extent the globus pallidus and internal capsule.

Tremor (Quelques remarques sur le tremblement).—Ladislas Haskovec. L'Encéphale, 1925, xx, 110.

TREMOR is found in organic and functional nervous conditions, in a hereditary form, in infections and intoxications. It is seen, too, in normal individuals on occasion, after muscular exhaustion, sexual excesses, wounds, catheterization, chill, emotional overaction.

In states of hysteria, neurasthenia, psychasthenia and traumatic neurosis, tremor may be pronounced, amounting, indeed, to a so-called névrose tremblante. On the other hand, many cases of tremor are called 'functional' when their basis is, as a fact, one of an organic nature (inclusive of toxic cases).

Tremor results "from lesions of the neural mechanisms controlling normal muscle tonus and regulating normal volitional movement." The phenomena are complex, and much more investigation is requisite to explain their appearance in differing states. The author takes tremor to be a reflex, or a manifestation of the automatism of "subcortical centres, cerebral and cerebellar centres, and also medullary centres," and considers it is not of myogenic origin.

This paper usefully reminds the neurologist of the complexity of the subject, but makes no specific contribution of a constructive kind.


Ten cases are given in some detail, occurring in the course of some eighteen months, and signalized by (1) severe vertigo; (2) vasomotor disturbances such as pale and cold extremities, and hyperidrosis; (3) low blood pressure. Some patients exhibited polyuria; other occasional symptoms were vomiting, faints with loss of consciousness, and (in three cases) apparent exaltation of the sexual functions.

Arguing that his cases belong to the epidemic encephalitis group, the writer proceeds to speculate that the pathogenesis of the symptoms is to be attributed to implication of the regio subthalamica and hypothalamica.

In this communication are described several cases of postencephalitic motor disorder characterized by spasm of the ocular muscles, resulting in, for example, an upward deviation of the eyes in a 'cramp' lasting for perhaps a quarter of an hour, but in some instances continuing for as long as half the day. With such ocular deviation the head may be extended and the palpebral apertures may be opened to their widest, nor will blinking then result when appropriate stimuli are tried.

In three cases convergence was completely in abeyance or much impaired; another case showed horizontal and rotatory nystagmus; another, defective downward deviation. Notwithstanding these symptoms, the author declares that the "absence of ocular palsies" in his cases excludes any nuclear or supranuclear lesion. A distinction is drawn between voluntary eye movements and "looking at a moved object," which is held to be reflex. The conclusion is reached that the ocular spasm of the recorded cases is a special variety of "failure of automatic movements" which is supposed to characterize Parkinsonism.

S. A. K. W.


The patient was a man of twenty-seven, who in 1918 was wounded by a shell fragment which cut through the left common carotid. Apart from various symptoms the result of local involvement, the patient showed a pronounced motor aphasia, a right-sided hemiparesis without the signs of pyramidal disease, and peculiar spasms of the right arm and leg. The authors attribute the latter to involvement of the 'striate apparatus,' implication of which they think is indicated by the following points: (1) the spasms were increased by approximation of the origin and insertion of the muscles concerned, "just as is characteristic of striatal contraction-conditions"; (2) alterations in the contraction-attitudes of the spastic limbs took place when the position of the head in space was changed passively, similar to those found in cases of hemiplegia; (3) the common carotid branches supply the basal ganglia.

As regards the first two of these points, it will be seen that the authors assign to striatal affection phenomena the connection of which with the basal ganglia is far, indeed, from being proved. They state in so many words that the 'neck-reflexes' seen in hemiplegic conditions are not the result of the pyramidal disease; "the concomitant implication of the striatal apparatus is the specific cause." In the reviewer's opinion this is speculation *in excelsis,* and it is contradicted by numerous clinico-anatomical facts.

One of the interesting features of the case was the frequent adoption of what the authors term the "pointing posture" of hand and forefinger (right). They give an illustration of the same attitude in a case of right-sided Jack-
sonian epilepsy, during the attack; there was at first a tonic contraction of the right arm, in this attitude, followed by gross clonic twitchings in the limb. Other symptoms and signs indicated a lesion (traumatic) of the lower end of the precentral and the posterior ends of the lower two frontal gyri, a localization which was confirmed by operation. In spite of all this, the authors hold that only the clonic part of the epileptiform attack is cortical and that the brief and transient spasmodic contraction of the hand in the "pointing attitude" must be due to an excitation of the striatal apparatus; they suppose "either a cortical excitation passes over to the striatal mechanism," or that "the increased excitability of [the latter] is the result of its isolation from cortical influence."

The authors do not appear to have considered the possibility of the spasmodic movements of their case having a cortical origin; these are declared to present distinct resemblances to athetosis (as is at once apparent from the photographs), which may result from cortical disease; they take the comparative absence of pyramidal signs to militate against the symptoms of their case being pyramidal, but they do not apparently mention the point that where such involuntary movements are prominent a severe paralysis of the pyramidal system is not to be expected.

S. A. K. W.


The syndrome first described by Babinski and Nageotte was attributed by them to obliteration of the terminal part of the vertebral artery with encroachment upon the basilar trunk. Later observers, following Wallenberg, have described a similar clinical syndrome from obliteration of the posterior inferior cerebellar. The authors distinguish two syndromes: one is that defined by Babinski and Nageotte; the other they call the lateral medullary syndrome, which corresponds clinically with Wallenberg’s syndrome, but is really due to obstruction of a branch of the basilar which they call the artery of the lateral recess of the medulla. Their attention was attracted to the subject by the following case. A patient was observed who presented a rightsided hemiparesis and associated sensory loss, with leftsided weakness of palate and pharynx, and cerebellar symptoms, but intact vocal cord. Oculosympathetic signs were absent. The diagnosis made was a softening of the lateral part of the medulla from obstruction either of the vertebral or the posterior inferior cerebellar. At autopsy minute examination of these two vessels proved that they were intact. The basilar trunk near its commencement was thickened and its lumen narrowed, especially at the origin of its lateral branches. Horizontal sections of the medulla showed an area of softening exactly in the situation anticipated. (The extent of the lesion is illustrated by diagrams and a photograph.)

As the result of dissections and experimental injection the authors conclude that the arterial supply of the medulla is arranged as follows:—

1. The paramedian vessels arise from the basilar, vertebral and anterior
spinal arteries; their territory includes both pyramids and extends backwards in quadrilateral form as far as the floor of the fourth ventricle.

2. The short circumferential arteries supply the lateral parts of the medulla. Here a distinction must be made between the upper and lower halves of the medulla. In the upper half there is on each side a wedge-shaped area with its base situated externally between the pyramid and the restiform body and its apex reaching as far as the wall of the fourth ventricle, which is supplied by a branch of the basilar, taking origin from that trunk just after its formation, before it gives off the anterior inferior cerebellar. This vessel, which the authors call the artery of the lateral medullary recess, runs laterally and down to reach the bulbopontine groove at the upper border of the olive. The remainder of the upper medulla, i.e., the dorsal segment on either side, is supplied by ascending branches from the posterior inferior cerebellar. In the lower half of the medulla the arrangement is simpler, the central parts, including the pyramids, being supplied by the paramedian vessels and the remainder by the posterior inferior cerebellar.

Analysis of previous case reports with pathological notes shows that in each case there has been obstruction of the vertebral and (or) basilar: in some cases the posterior inferior cerebellar has also been involved, but in certain cases it has been intact.

A lesion of one of the paramedian vessels may cause hypoglossal paralysis with contralateral hemiplegia. Obstruction of the posterior inferior cerebellar produces a clinical syndrome which varies with the site of the lesion, but is apt to include disturbance of deep sensibility (restiform body), vertigo (Deiter's nucleus) and oculosympathetic signs.

This paper, which is lucid, brief and well illustrated, deserves consultation in the original.

C. P. S.


Neurologists are generally of opinion that in every case of progressive bulbar palsy there is to be found post-mortem an associated degeneration of the pyramidal tracts.

In the case recorded, that of a man of seventy-three, the signs of glosso-labio-pharyngeal paralysis were observed during life, together with brisk arm jerks, normal abdominals, flexor plantars, feeble knee jerks and absent ankle jerks.

No disturbance of sensation could be found. The spinal fluids gave a negative Wassermann reaction and colloidal benzoin curve, and normal cell and protein content.

Histological examination revealed atrophy of the bulbar nuclei and a systematic degeneration of Goll's column from the sacral region up. No pyramidal degeneration was found.

C. P. S.

Cases of pure brachial monoplegia of spinal origin are rare, but the instance here briefly reported seems particularly clear-cut. It followed a gunshot wound of the right side of the neck in 1916. The patient, examined subsequently, exhibited neither sensory change nor pathological reflexes in the right leg; the right arm was typically monoplegic, with no loss of sensibility and with increased deep reflexes; sensation was normal on the left.

An interesting discussion on localization in the upper spinal pyramidal tract leads to the conclusion that the arm fibres are dorsal and the leg fibres ventral in the tract. It is shown that in pure arm cases loss of sensibility does not occur anywhere, and this is taken to prove that it is more remote from the spinothalamic path than the leg fibres. Again, if Flatau's 'law' that in the cord the fibres of shorter length are closer to the central grey matter, while longer fibres are more peripheral, is correct, then it supports the authors' contention, for the dorsal part of the pyramidal path is nearer the grey matter (dorsal cornu) than the ventral.

S. A. K. W.


Dr. Goldflam describes an interesting and unusual case apparently of recurrent subarachnoid haemorrhage of spinal type, leading eventually to a cystic formation at the level of the cauda equina, found at operation. He refers to the rarity of the condition, citing a few other cases, the details of which are usefully given for purposes of comparison.

He takes the view that the "vasomotor components of migrainous attacks" are responsible for many cases of spontaneous cerebral subarachnoid haemorrhages; in others, the condition is a neurogenic diapedesis of the red blood corpuscles. Spontaneous spinal subarachnoid haemorrhages also occur; their origin is obscure. The fact that they recur suggests a neurogenic basis.

S. A. K. W.


The author's case is that of a young woman of twenty-four, who had observed for some six years that the left arm and leg were gradually becoming weaker and seemed to be getting shorter. More recently the same changes were noticed in the right limbs. No cause for the condition could be ascertained. On examination various muscles in the limbs were seen to be atrophic; the flexor groups more particularly, however, were found to be, as it were, shrunken and hardened, and they resisted passive stretching; it being impossible, for instance, to extend the arms completely at the elbow. The power of the affected muscles was considerably reduced. Muscle reflexes
were weak; electrical reactions were diminished, without R.D. No myasthenic or myotonic phenomena could be discovered. Clinical tests of all sorts revealed no other changes. A small piece of muscle from the calf, excised for examination, showed no sign of degeneration of muscle fibres, but only a kind of curliness, like wavy hairs.

Myosclerotic changes occur in (1) ischemic paralysis; (2) myositis of various origins (rheumatic, infective, parasitic, etc.); (3) the early stage of myositis ossificans; (4) sclerodermia. Discussion has also taken place as to the occurrence of this myosclerotic state in muscular dystrophies, and numerous observers have reported cases apparently of such a kind (Jendrassik, Hahn, Gowers, and others). The author points out that in his case some of the muscles were shortened without being atrophic, and concludes that the sclerosis is not secondary to antagonistic contracture, but is primary and due to a special muscular disease. For this essential, primary, muscle shortening he proposes the term given in the title. He distinguishes between myosclerosis and contracture, considering that the histology of the case he reports proves that the shortening is due to a change in the muscle fibres and not to any interstitial connective tissue growth. Reference is made to cases described as myositis fibrosa progrediens and to the "atrophic and retractile myosclerosis of old age" (Dupré and others).

S. A. K. W.

[69] Progressive symmetrical muscular atrophy limited to homologous muscles (triceps, brachials and quadriceps femoris) (Atrophie musculaire symétrique progressive limitée à des muscles homologues).

—Knud H. Krabbe. Rev. neurol., 1925, xli, 481.

A man at the age of thirty-six came under the author's observation for weakness and wasting of triceps and quadriceps on both sides. The affection had begun insidiously ten years before in the lower limbs and had progressed gradually. It was preceded by painful spasms in the lower limbs. The family history was negative.

The affected muscles were weak and wasted. The appropriate tendon jerks were absent. No sensory disturbance was discovered. The Wassermann reaction was negative (whether in blood or spinal fluid not stated).

Five years later the affected muscles were completely paralyzed and failed to react to galvanism or faradism. No other part of the body was affected. The only other evidence of disease was a systolic blood pressure of 200. The author discusses the probable nature of the disease and concludes in favour of a myopathy. He has been unable to find any similar cases in the literature. (A case of myopathy limited to the quadriceps has recently been recorded by E. Bramwell, Proc. Roy. Soc. Med., 1922, xvi, No. 2, Section of Neurology.)

C. P. S.


As was to be expected, the application of the physiological work of Magnus on experimental animals to clinical cases in man has begun on a large scale.
The author divides his paper into two sections. (1) Tonic neck reflexes are produced by altering the position of the head relatively to the trunk. The best clinical subject for these is the hemiplegic. Associated movements (Mitbewegungen) of the paralyzed extremities are effected by the tonic neck reflexes. "Where neck reflexes are, there also are associated movements." The reflexes cannot be demonstrated in normal adults. In some instances the result of the reflexes is to bring about a notable if transient increase of power which may be of service to the patient. As a rule, the reflexes concern mainly the extensors, or mainly the flexors, with minor variations. Inclination of the head to one side is of less effect as a reflex-producing agency than rotation of the head; the least constant in its effect is head flexion and extension. The author considers that in man implication of both pyramidal and extrapyramidal systems is necessary for the development of the neck reflexes.

(2) Tonic labyrinth reflexes can be demonstrated in the adult. The scheme employed by the author is one in which the reflexes are elicited by reference to the relation of the axis of the body to the horizontal. There is one position in space, in this respect, which exercises a maximum effect in the production of extensor tonus, and one, similarly, if less pronounced, which produces a maximum of flexor tonus. These positions, apparently, do not quite correspond in man to what is found in the experimental animal. The duration of tonic labyrinth reflexes is some five to fifteen seconds. They are best seen in hemiplegics, and for their appearance involvement of both pyramidal and extrapyramidal systems, at some point above the medulla, is in the author's opinion essential. The centre is neither in the cerebral hemispheres nor in the cerebellum.

S. A. K. W.


The author, being struck by the fact that in Gordon's original cases the phenomenon that bears his name was found only when the pyramidal lesion was cerebral in site, and not spinal, investigated fifty spinal and fifty cerebral cases of pyramidal disease. In none of the former was the sign demonstrable, while it was present in eight of the latter. The further deduction is made from this study that Gordon's 'paradoxical' reflex is mainly if not entirely to be expected in cases in which the cerebral lesion is acute or subacute, or in which there is a general lowering of cerebral function, e.g., in cases of Jacksonian epilepsy, repeated apoplectic attacks, disorders due to high blood pressure, slight confusional states, giddy attacks, and the like. It may be found where all other ordinary pyramidal signs fail. There is no parallelism between its occurrence and loss of the abdominal reflexes. It is not obtained in chronic cerebral cases.

The conclusion is that it has a value of its own, though its explanation is not easy, and it should not be regarded either as an indication of pyramidal disease or as a forerunner of such definite pyramidal signs as the Babinski reflex.

S. A. K. W.