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paralysis. As in a previous paper of the same writer dealing with the same subject (see this JOURNAL May, 1923, p. 54), it is to be regretted that the title of the communication conveys no information as to its interesting contents.

Summarized, the thesis is that Landry's paralysis is a special biological expression of differing morbid processes, of an unfavourable nature, in which behind the clinical symptoms lies a biological preliminary in the form of toxic action on a nervous system which reacts anaphylactically (allergically) by toxic over-sensitisation.

S. A. K. W.

[114] Is the albumin-content of the cerebrospinal fluid different at different levels? (Bestehen Unterschiede in Eiweissgehalt des Liquor cerebrospinalis in verschiedenen Hohen?).—JACOBI. Münch. med. Woch., 1923, lxx, 670.

It has been shown almost beyond doubt that the cell content of the cerebrospinal fluid is different at different levels, and that the cerebrospinal fluid as a whole is not to be judged in this respect by the result of a single examination. The question then arises whether similar variations occur in the albumin content. That such is the case has been suggested by the findings of Walter and others who carried out albumin estimations on consecutive samples drawn off at the same puncture. Eskuchen, Matzdorff and Schönfeld, however, have not been able to discover such differences.

The author of this paper attacked the question armed with a new method of examination: by means of the 'interferometer,' which determines optically the strengths of solutions, he examined, in each of his cases, five consecutive specimens of fluid taken at the same puncture. In this way he studied thirty fluids. His findings are in keeping with those of Walter, and tend to show that the albumin-content does vary slightly at different levels, and is usually highest in the first sample of fluid drawn off.

J. P. M.

SENSORIMOTOR NEUROLOGY.


An old man of eighty-two suffered from a stroke, without loss of consciousness, which was followed on the fourth day by the appearance of pronounced intention-tremor of the right arm, the leg to a less extent, and of the upper part of the trunk, but excluding the head. At an autopsy two years later hemorrhagic softening of the right nucleus dentatus of the cerebellum was found, with secondary degeneration of the superior cerebellar peduncle. The nucleus fastigii was intact, but the nuclei emboliformis and globosus were implicated. The right middle peduncle was also degenerated, and the contralateral inferior olive and olivo-cerebellar tract in the medulla.

Leiri thinks the intention-tremor of cerebellar disease is due to an attempt on the part of the cortex to correct exaggerated, hypermetric movement the result of the cerebellar lesion. The varying degree of representation in the
cortex of limb and head movements, in different members of the animal species explains the variation in the appearance of tremor during volitional innervation, as also its tendency sometimes to disappear.

S. A. K. W.


Curschmann briefly describes the case of a man of forty-one years of age, who for about five years had been suffering from progressive unsteadiness of gait, giddiness and disturbance of sight. The patient's grandfather had been a healthy man all his life and lived to the age of eighty-eight; the patient's father, however, had developed an unsteadiness of gait at the age of fifty-two, and his sight had become poor; the patient's uncle, also, had suffered from 'staggering' and weakness of sight from about the age of fifty, and a son of this uncle has already begun to be unsteady, though just over twenty years of age.

The patient had a brother and two sisters, all of whom were healthy.

Examination revealed the following objective signs: well-advanced optic atrophy, marked nystagmus, brisk tendon jerks, weakness of the abdominal and absence of the cremasteric reflexes, normal plantar responses; there was no loss of power in any limb and no sensory disturbance. The control of the arms did not appear to have suffered in any way in either this patient or his relatives, so that several of them were still able to carry on sedentary occupations requiring fine hand movements.

In the absence of signs of posterior or lateral column involvement, Curschmann regards the symptoms as purely cerebellar in origin, and classes the case as one of the hereditary cerebellar ataxy described by Marie.

J. P. M.

[117] Chorea cruciata; differential diagnosis between striatal and cerebellar chronic chorea (La chorea cruciata; diagnostic différentiel des chorées chroniques d'origine striée et d'origine cérébelleuse).—L'HERMITTE and BOURGUINA. L'Encéphale, 1923, xviii, 228.

The pathological findings in Sydenham's chorea, as in choreas consequent to infective disease, e.g., post-encephalitic, are so diffuse as to be of little use for the purposes of pathological physiology. In this respect less exception can be taken to cases of chronic chorea, with or without mental accompaniments. These are the expression of a lesion of double polarity—cortico-frontal and striatal (putamen-caudate). According to Marie and Lhermitte, the frontal lesion is responsible for the mental changes and the striatal for the chorea. The author rejects absolutely the hypothesis of a cerebellar origin (superior cerebellar peduncle) for the choreiform movements of Huntington's chorea, but accepts such an origin for certain other choreas. It is noteworthy that lesions of the dentate do not appear to be followed by choreiform movement, and that in the case of the superior cerebellar peduncle the lesion is commonly in the vicinity of its crossing (Wernekinke's commissure). Further, the authors consider such chorea is never derived from lesions of the red nucleus, but are
doubtful whether in certain instances chorea may not have a thalamic source. (Incidentally, the reviewer draws attention to the doubtful nature of any hypothesis which would assign symptoms to disease of a nerve-tract but not to the grey matter of its origin or termination.) Further, the authors do not mention the difficulty arising from the association of lesions of the superior cerebellar peduncle with tremor.

Among the symptoms accompanying chronic chorea, and assigned by the authors to disease of the corpus striatum, are dysarthria, spontaneous akinesis, and loss of automatic movement. Their case is that of an old lady of eighty-six, with severe choreiform movement of the left arm and hand, and of the right foot. The face was the seat of grimaces, especially on the left side. The movements had come on suddenly, and were accompanied by obvious cerebellar symptoms—adiadochokinesis, dysmetria, hypotonia, etc. The suggestion is that the lesion was situated at or near Wernekink’s commissure. Quà chorea, the movements are distinguished from those of striatal origin only by the association of cerebellar signs with them, according to the authors. Chorea cruciata is ingeniously suggested on the analogy of hemiplegia cruciata.


The case here described appeared clinically to be one of eighth nerve tumour, but proved at autopsy to be a cystic glioma, growing from the anterior part of the left cerebellar hemisphere and extending into the ponto-cerebellar angle and over the front of the pons and medulla. Although the trigeminal nerve was lapped round the tumour, the only cranial nerves showing clinical evidence of involvement appear to have been the sixth, seventh, and eighth. The case presented during life the phenomenon of ‘pendular knee-jerk’ described by the author, and was one of the first in which he had observed this sign. He describes it as a ‘passivité’ of the antagonistic muscles of the thigh, and uses this term in preference to ‘hypotonia,’ which is also used to describe a condition of hyperextensibility of muscles, a phenomenon not present in the ‘passivité’ of cerebellar origin. The author’s work on this subject, as also that of Holmes and Stewart, to which he draws attention, is well known.

J. G. Greenfield.


The case is that of a young man of twenty-eight, with the usual symptoms of intracranial tumour. Homonymous right hemianopia, right hemianesthesia, alexia and agraphia suggested a localization in the left parieto-occipital region, duly confirmed by autopsy.

The patient exhibited well-marked motor apraxia, and the type of defect shown leads the author to regard so-called ideational and motor apraxias as clinically indistinguishable.
It seems certain that lesions of the supramarginal gyrus and vicinity are of themselves capable of giving rise to apraxia, usually bilateral; unilateral apraxia (left side) is commonly the result of callosal lesions. The author's discussion is of interest, but his conclusion that supramarginal apraxia is a 'sensory apraxia' in reality, due to disorder of the sensory components in the images of movements, is open to objection.

S. A. K. W.

[120] The etiology of a tic developing fifteen months after an atypical lethargic encephalitis (Discussion sur l'étiologie d'un tic survenu quinze mois après une encéphalite légère atypique).—De Saussure. Arch. Suisses de Neur. et de Psychiat., 1923, xii, 298.

This is a curiously interesting clinical document, in which the sequence of events was somewhat as follows: In February, 1920, a young woman of twenty had an attack of influenza, followed in a few days by convulsions, hallucinations, psychomotor agitation, inversion of sleep rhythm. The acute stage soon passed, but for no less than fifteen months the insomnia persisted, and during the same period amenorrhœa was noted. In May, 1921, an emotional shock rather upset the patient, and within a day or two incessant yawning (every two or three minutes) developed, associated with an overwhelming desire for sleep. The yawning continued for fifteen days, and was followed in turn by intense bilateral neuralgia of the face and by bilateral spasm of the lower facial musculature, including the platysma and sometimes the sternomastoid. During the spasm respiration ceased and a brief period of polypnœa ensued. The pain associated with the spasm was exceptionally severe, and persisted for about two months. Later in the year the patient's mental condition rather deteriorated, irritability increased, and coprolalia developed, suggestive of Gilles de la Tourette's disease. She was removed to an asylum in December, 1920, and since has spent three periods there, her involuntary movements continuing in a more restricted fashion, the spasm becoming a tic, and her mental state remaining unchanged. Since February, 1920, the menses have appeared only three times.

The discussion given by the author is instructive though inconclusive, and is illustrated by many useful references to the literature.

S. A. K. W.


A full description is given of a case in which the patient became so completely 'paralysed' that he could make no movements except those of the eyes, swallowing, and respiration, nor could his trunk or limbs be moved passively. This differentiated the condition from catalepsy, which has been described as occurring after encephalitis. The Babinski sign was never positive, nor were there any indications of pyramidal tract involvement. This suggests that extra-pyramidal motor lesions may produce an excessive spasticity which, in the case quoted, is said to have been far greater than that found in even severe cases of Parkinsonian rigidity. In some of the latter new bone-
formation has been found similar to that described by Dejerine-Klumpke and Ceilier in injuries to the spinal cord, where the ossification was preceded by edema and showed no signs of amelioration, nor was any subsequent improvement in motility of the limb noticed. In the case described, however, the limbs regained motility and the ossification diminished, and even in one place disappeared. The pathogenesis of this ossification is obscure, but seems to depend on the failure of certain nerve fibres to function. The present case showed that they were not concerned in the sensory or pyramidal tracts, since these were not affected. A second case is described in which the left arm and leg were similarly affected. The course of this case was remarkable in that the distal joints recovered before the proximal, in contra-distinction to what obtains in pyramidal tract lesions. A third case is described illustrating the difficulties of diagnosis between cerebral embolus and encephalitis. A comparison of the epidemiology of encephalitis and poliomyelitis concludes a valuable contribution.

R. G. Gordon.


A young woman of twenty-one had a slight attack of encephalitis lethargica in February, 1920, which left her somewhat dull and stiff, but otherwise well. In August of the same year, the stiffness became progressively worse, so that by November she lay in a condition of severe Parkinsonian rigidity, with complete absence of spontaneous movement. On November 24, her temperature and pulse began to rise; the next day the pulse was 184 and the temperature 104.4° F.; death occurred on November 26, when the temperature was 106° F. In the absence of any inflammatory lesions in the lungs or viscera the authors attribute this hyperpyrexia to an attack on the medulla oblongata by the virus of encephalitis. The examination of the brain showed depigmentation and disappearance of the cells of the substantia nigra, chromatolysis and disintegration of a certain number of the cells in the globus pallidus, general excess of small cells in the brain tissue and dilatation of vessels, but no perivascular infiltration.

J. G. Greenfield.


In this epidemic of 139 cases one or two peculiarities are worthy of mention. The first outbreak was in a village several miles from Tübingen. There in the week before the 'day of Assumption' an epidemic of 'influenza' fell upon the infants' school; catarrh of the upper air passages was the chief symptom, and it was so severe that the authorities temporarily closed the school. The teacher had a severe attack of 'catarrh,' became paralysed and died; one
of the pupils died later, also with paralysis. Except for these two cases, the epidemic in that particular school ran the course of one of influenza.

In an institution in Tübingen itself fourteen children suffered from feverish 'colds,' and their temperature charts, all very similar, suggested an ordinary slight influenza epidemic. But in three of these cases paralyses appeared.

The experiences in these two schools raise the question of the frequency of abortive attacks of the infection which is responsible for acute anterior poliomyelitis. In the school in Tübingen the non-paralytic cases were much commoner than the paralytic in the proportion of 11:3; Schall states that Brosstrom, in 1919, found the proportion to be 4:6:1, but until we are able to identify cases of the non-paralytic type of the infection such statistics must be very few and not above suspicion.

J. P. M.


Redlich reports a case of so-called Erb's syphilitic spinal paralysis, but calls in question the specific nature of the disease. Though both clinically and histologically the findings in the case reported (a male, aged sixty-one) correspond closely with the description given by Erb, in 1892, Redlich considers that the directly syphilitic nature of the lesion is unproven and prefers to attribute it to an ordinary arteriosclerosis affecting the cord as a whole. The patient, it is true, had acquired syphilis at the age of eighteen, but from that time until the onset of typical symptoms about six years before his death he had led an apparently healthy life. Possibly the arteriosclerosis may have been induced by a slowly acting syphilitic toxin; yet we are very much in the dark about the working of such a toxin, and there may be many other toxins capable of producing similar effects. In any event, the morbid process, whatever its nature, affects many spinal tracts and is neither a focal degeneration nor a system disease.

Taking all the evidence into consideration, Redlich comes to the conclusion that Erb's syphilitic spinal paralysis is not a so-called clinical entity but a group of symptoms which reveal a particular stage of a chronic and diffuse spinal degeneration.

ALFRED CARVER.


"A spinal cord tumour is a foreign body in an extensible bony canal compressing the adjacent structures which that canal contains in the order of their compressibility." The authors describe how the growth gradually presses on and displaces the cord, first affecting its blood and lymph supply, and this alone may induce many clinical symptoms. They point out how localized compression may dam up cerebrospinal fluid above the tumour, and this may cause pressure which will mislead observers into diagnosing the tumour as being situated two or three segments higher than it really is.
They lay stress on the fact that no one symptom is specific of any tumour, and especially of cervical tumours; consideration should rather be given to the composite picture. Paralysis may be flaccid or spastic; rigidities, spams and weakness of cervical muscles should always be observed with the possibility of spinal tumour in mind. Root pains may occur, and paralysis of the diaphragm is often important. Organic reflex trouble is an early symptom, and this, together with symptoms referable to the cervical area, should be regarded as significant. Lumbar puncture is stated to be dangerous in these cases, and should only be resorted to with great care. Eight illustrative cases are given.

R. G. Gordon.


The plexus of the last four cranial nerves and the cervical sympathetic below the parotid gland renders these nerves liable to simultaneous injury by bullet wounds, tumours, glands, or infections. A review of the literature is given in relation to the several functions of these nerves, and the ambiguity as to the limits of respective function of the ninth and tenth is noted. In the two cases fully described no data could be adduced to solve this question, since both nerves were affected. The author is, however, satisfied that the vagus is not a purely sensory nerve, as Vernet suggests. The observation which is important is that the tone of the facial muscles was impaired though voluntary control was intact. This, in relation to certain cases of facial paralysis in which voluntary control is lost but tone is little affected, suggests a dual innervation of the facial musculature. The author thinks that the tonic status of the muscles depends on the cervical sympathetic, while voluntary movement depends on the seventh nerve.

R. G. Gordon.


Sacralization of the fifth lumbar vertebra is one of the less common causes of pain in the lumbo-sacral region. In these cases the transverse apophyses of the vertebra are so enlarged as to resemble the sacral ilia. They may, or may not, be structurally united to the sacrum, but in any case the intervertebral space through which the fifth lumbar nerve emerges is considerably reduced. Clinical symptoms, when present, seem to originate coincidentally with the termination of ossification in the enlarged process, which may occur as early as five and a half years or as late as forty-eight years. The chief sign is pain in the back, unilateral or bilateral, according as one or both sides of the vertebra are affected; changes of posture occur, and the lumbar lordosis is lost. Rarely there is an accompanying degenerative change in the spinal cord. A case is quoted in which this occurred apparently as a sequela of the radicular involvement.

R. G. Gordon.

Symptoms referred to the distribution of this nerve are not commonly described in relation to sciatica, though crural neuralgia is common in toxemia, appendicitis, etc. Certain authors have insisted on the frequency of radicular lesions in sciatica, and have pointed out that such radiculitis often affects the lumbar segments giving origin to the crural nerve. In such cases there is pain in its distribution, wasting of the quadriceps extensor, and sometimes interference with the patellar reflex. Golombeck found the latter abolished in 16 per cent. of cases of sciatica. The author was able to find symptoms referable to the anterior crural nerve in thirty-nine cases out of fifty of true sciatic neuritis. These take the form of pain in the distribution of the crural saphenous nerves with tender points in the groin, inner side of knee, internal malleolus and big toe, of muscular wasting of the quadriceps and sometimes abolition of the knee-jerk. After some discussion as to the exact location of the morbid process in sciatica, the author gives his view, that there is an inflammatory process which attacks that part of the nerve which lies between the meninges and the plexus.

R. G. Gordon.


The author accepts the principle of dissociated sensibility and differentiates an affective and a critical function in sensation subserved by different nerve fibres. When the affective function returns first in regeneration the dissociation is protopathic in type, while when the critical function returns first the dissociation is epicritic. One case of each type is described, and from these he concludes that:

1. The occurrence of protopathic and critical dissociation after peripheral nerve injuries points to the existence of two fundamental independent anatomical systems of nerve fibres for the mediation of the basic critical and affective forms of sensibility.

2. The critical and affective systems have intimate functional relations, each system functioning in a general way as the antagonist of the other.

3. Within certain limits of stimulation each system may partially inhibit the other so that elements from each may fuse, or enter consciousness simultaneously, forming the basis of our ordinary composite sensations, such as pricking, heat, cold, etc.

4. The fundamental critical system is further differentiated into independent sets or sub-systems of fibres for the mediation of the specific elements, warmth, cold, etc.

5. The affective system does not become differentiated otherwise than as it functions side by side with the critical system, supplying the affective element in our ordinary composite sensations.

6. 'Protopathic' heat and cold as primary forms of sensibility have no existence in fact.
7. Spatial discrimination as tested by the simultaneous application of the compass points is not a primary form of sensibility.

8. The so-called 'epieritic ranges' for heat (warmth) and cold, considered as primary independent forms of sensibility, have no existence in fact. They are merely an arbitrary division of the general range, which for each of the critical elements, warmth and cold, is a natural continuum.

9. The radial nerve mediates deep critical and affective forms for portions of the fingers and dorsum of the hand.

10. Normal and newly regenerating primary sensory neurones of the affective system, when injured or overstimulated, continue to hyperfunction for some time after the stimulus has been withdrawn.

11. Newly regenerating primary affective neurones exhibit a marked tendency to function according to the 'all or nothing' principle.

12. Over-reaction is, in part, the result of release of the affective mechanisms, with their inherent 'all or nothing' tendency, from the inhibitory influence of the critical mechanisms.

13. Neither system, as low in the scale as the batrachians, seems to be prior to the other in point of time or development.

14. In deep sensibility the critical and affective elements are mediated by separate anatomical systems which have functional interrelations like those found in the superficial critical and affective systems.

15. The 'co-operation of antagonism,' under which the critical and affective systems function, furnishes not only the basis of cognitive methods of adjustment to environmental changes and of ordered sensation and psychic development, but is the basic point of ordered functioning in neural mechanisms, from the simplest reflex to the most complex mental reaction.

R. G. GORDON.

[130] Late distal myopathy (Les myopathies distales tardives).—NAVILLE, CHRISTIN and FROMMEL. L'Encephale, 1923, xviii, 182.

In a useful review the authors add five personal cases to those already recorded, and believe that distal myopathy in the female has characters of its own. Thus they stress its appearance at a later age than in the male, five out of eight cases having developed after the age of forty, its slow evolution, its incidence on the peripheral extensors of the limb. In the case of male distal myopathy, the age of onset is notably earlier, ten out of thirteen cases having developed before the age of thirty; the small muscles of the distal segments are affected somewhat rapidly, and the symptoms generally seem a compound of a myopathy and a myelopathy. The hereditary element in distal cases is conspicuous by its absence, both in male and female examples; the latter, according to the authors, show as a rule purer instances of true distal myopathy than the other sex, in which the course of the disease does not always follow a 'march' from periphery centralwards. There is a good bibliography.

S. A. K. W.