
Bielschowsky’s proposed classification of the inherited neural degenerations is as follows, incomplete though its author allows it to be in the present state of knowledge:—

I. Pure Dysplasias.
   A. Cortical malformations; micropolygyria, pachygyria, agyria.
   B. Malformation of corpus striatum; état marbré.
   C. Malformation of more caudal parts of neuraxis; micromyelia, syringomyelia and its analogues.

II. Dysplasias with a Blastoma Element. Tuberose sclerosis (related to glioma and Recklinghausen’s disease).

III. Abiotrophies.
   A. Abiotrophies with a blastoma element; pseudosclerosis (and certain forms of diffuse sclerosis?).
   B. Abiotrophies with local total necrosis of the parenchyma:—
      2. Necrosis of globus pallidus: a solitary case recorded by Fischer.
   C. Abiotrophies with selective necrobiosis of ganglion cells:
         b. Ditto with special involvement of certain systems: juvenile (chronic) amaurotic idiocy with cerebellar atrophy.
      2. Selective degeneration of certain cell-systems:
         a. Of nucleus caudatus and lentiformis: chronic chorea.
         b. Of cerebellar systems: cerebellar heredo-ataxia in its many forms.
         c. Of corticospinal system: spastic spinal paralysis, spinal amyotrophy, amyotrophic lateral sclerosis.

Wilson.

SENSORIMOTOR NEUROLOGY.


The case was that of a man of 57, who suffered at the age of 17 from a severe illness, apparently of a toxic or infective nature, as a result of which he showed slight but definite signs of defective cerebellar function. Very gradually these signs became intensified, and when he came under observation his condition was one of advanced cerebellar disturbance. In addition, there was a definite rigidity of certain muscular groups, and the
more or less permanent involuntary adoption of flexor, pronator, and adductor attitudes. Atypical nystagmus was noted. The plantar reflexes were flexor in type.

The cerebellum was much reduced in size, not quite symmetrically, while the cerebrum showed no recognizable departure from normal. The lesions in the former were strictly confined to the cerebellar cortex, which was so severely and generally degenerated as to be practically suppressed. The Purkinje cells were grossly affected and had almost entirely disappeared; molecular and granular layers were equally defective. By comparison, quite insignificant secondary atrophies were found in the central nuclei and in the olivary bodies and olivo-cerebellar systems. The superior cerebellar peduncles and red nuclei were for all practical purposes normal. Archambault attributes the cortical parenchymatous degeneration in part at least to disease of the pial vessels entering the cortex, which were thickened and reduced in lumen, but he states that others might not so explain the changes. Seemingly only a quite moderate degree of glial reaction was noted.

Archambault gives a useful discussion of the correlation between the clinical and the pathological findings. It is curious, however, that no explanation is attempted of the tremors forming a prominent symptom in the case, especially in view of the apparent integrity of the cerebellotegmental systems. Further, the patient’s attitude and certain other features were reminiscent of paralysis agitans, and it is regrettable that so exhaustive an examination does not appear to have included a microscopical investigation of the basal ganglia and regio subthalamica. A full précis is given of previously recorded cases, with the exception, curiously, of that published by Holmes (Brain, 1907, xxx, 466), which is as a fact one of the most striking of them all.

Wilson.


A soldier was wounded by a shrapnel bullet entering the head some four centimetres behind the left mastoid. At a subsequent date he presented highly characteristic left cerebellar symptoms, and, in addition, there were continuous clonic tremor or involuntary contractions of the soft palate, left side of the pharynx, and, to a less extent, of the left vocal cord. In the absence of a pathological examination, Pfeifer is content to record the documentary value of his case, and to assign the clonic contractions to that group of involuntary movements known to develop on experimental separation of the cerebellum from its pontomedullary connections. As there was no volitional paralysis of the muscles in movement, the phenomenon is a purely cerebellar one, comparable to nystagmus (Pfeifer does not mention the term ‘palatal nystagmus’ sometimes employed) and to tremor of mid-brain type.

Wilson.

KLIEN gives a minute anatomical investigation of a case of continuous rhythmic contractions of all the muscles involved in swallowing, from palate to diaphragm, coupled with synchronous clonic twitches of the orbicularis oculi, following an apoplexy, and continuing unchanged for eleven months, until death. Post mortem there was found an old lesion in the right cerebellar hemisphere in the immediate vicinity of the dentate nucleus, spreading outwards to destroy the white substance of the lobus semilunaris superior; a considerable portion of the dentate in its ventral aspect was involved in the lesion. From it degenerations were readily traceable to the cerebellar cortex of the same and of the contralateral side, to the red nucleus, and to both olives.

Among the various anatomical conclusions, for the details of which the original must be consulted, reference may be made to the evidence adduced by Klien in favour of the view that the olivocerebellar connections are to a considerable extent from cerebellum to olive, i.e., cerebellofugal, and not only cerebellopetal as has been so often maintained.

On the physiological side the author’s view, admittedly hypothetical to a large extent, may be expressed as follows. The cerebellum exercises a tonic influence over the functioning of Deiters’ nucleus; when the former is diseased, impulses going from the latter to the eye-muscle nuclei are so altered that nystagmus results; analogously, the motor nuclei of pons and medulla are under the influence of stimuli from the nucleus motorius tegmenti (Edinger), of which Deiters’ nucleus is but a part; if the latter functions imperfectly owing to cerebellar defect, ‘nystagmus’ in the former motor mechanisms will result; among these is the mechanism for swallowing.

WILSON.

[65] Clinico-anatomical contributions to our knowledge of aphasia, agnosia, and apraxia (Klinische und anatomische Beiträge zur Kenntnis der aphäischen, agnostischen, und apraktischen Symptome).—VON STAUFFENBERG. Zeits. f. d. g. Neurol. u. Psychiat., 1919, xxxix, 71.

Of the eight cases reported with a wealth of detail in this very long paper, reference may be specially made to two in which apraxia was a prominent symptom.

The first was that of a man, age 56, whose symptoms consisted, inter alia, of pronounced left-sided motor apraxia and tactile agnosia, and of bilateral ideational apraxia. In addition, the phenomenon of tonic innervation as described by Wilson and Walshe was present in the left arm and hand. Pathologically important lesions were the following: destruction of the corpus callosum in its middle and posterior thirds, and spread of the lesion in the right hemisphere to the under-surface of the parietal and
supramarginal cortex; by these the left-sided apraxia, tactile agnosia, and
tonic innervation can be understood. The more general psychical symptoms
shown by the patient, inclusive of the ideational type of apraxia, are
assigned by the author to the diffuse vascular condition and to the reduction
as a whole of the white matter of the left hemisphere.

The other case was that of a man of 45, with right homonymous hemi-
anopia, right hemiplegia (flaccid), right hemianæsthesia; his psychomotor
and psychosensory symptoms consisted in slight sensory aphasia, perseve-
ration, echolalia, and, on the left side, a severe degree of apraxia more or
less of a mixed type. The pathological basis for the symptoms was a large
area of softening round the left supramarginal gyrus, spreading deeply
into the brain; no vascular changes otherwise were noticeable. The
author discusses the possible explanation of the apraxia, and attributes it,
in part at least, to 'Fernwirkung', i.e., to the distant effect of the lesion on
linked neuronic systems, mainly those of the frontal and corpus callosum
areas. It is probable, further, that a degree of functional separation of
the left sensorium from the motorium occurred, a condition emphasized by
Liepmann and others as being of significance for apraxia.

Wilson.

[66] A case of encephalitis periaxialis diffusa of Schilder [Ein Fall
von Encephalitis periaxialis diffusa (Schilder)].—Von Stauffenberg.
Zeits. f. d. g. Neurol. u. Psychiat., 1918, xxxix, 56.

Encephalitis periaxialis diffusa is a rare disease, of which apparently
only some seven instances have been recorded. It presents certain peculiar-
ities distinguishing it in toto from any other nervous disease, with the
exception, in some respects, of disseminated sclerosis.

V. Stauffenberg's patient was a woman medical student, age 21, whose
illness led to a fatal issue in eight months. It began with ocular symptoms
in the form of amblyopia, restriction of the field, and left papillitis, followed
by temporal pallor. These ocular phenomena rather cleared up after three
months, to be followed by a somewhat silly euphoria. A diagnosis of
disseminated sclerosis was made. Later the right eye became almost blind,
reflexes were exaggerated, visual agnosia appeared, first the left and then
the right limbs became anesthetic and spastic, and there was a double
Babinski sign. Eventually coma supervened on top of a progressive
psychical deterioration. The cerebrospinal fluid was under pressure, and
showed a great pleocytosis. During the illness moderate rise of temperature
occurred again and again. A subsequent diagnosis of some form of
encephalitis analogous to acute disseminated sclerosis was suggested.

Pathological examination revealed in characteristic form the peculiar
changes of encephalitis periaxialis diffusa. Great areas of the subcortical
white matter were absolutely colourless with Pal's method, indicating
complete stripping of the myelin sheaths of the fibres of projection and
association. With slight exception the cortex itself stained normally,
rendering the contrast between it and the white matter the more
striking. The zone of arcuate fibres indicating junction of grey and white
matter stood out prominently. A similar lesion was found in the optic
chiasma. The diseased areas resembled plaques of disseminated sclerosis in the conservation of the axons as seen by Bielschowsky’s method. The sheaths of the vessels in these areas were filled with cells; glial cells were abundant throughout, especially of the spider type.

It is mainly the remarkable limitation of the morbid process to large subcortical areas, somewhat greyish-yellow or greyish-green on fresh section, sharply differentiated from the cortex, that distinguishes the disease from disseminated sclerosis, to which, nevertheless, it is probably akin. V. Stauffenberg gives a résumé of the already recorded cases.

**Wilson.**

[67] **Traumatic thalamic syndrome** (Syndrome thalamique traumatique).


The patient, a soldier, age 28, was rendered unconscious by a piece of shell which penetrated the face just below the left orbital ridge. He came under observation two months later. He complained of headache, and his speech was slowed. On examination, he presented a slight right hemiparesis. This condition was masked by the presence of choreo-athetotic movements in the right arm, which increased during excitement and were quite beyond control. He attempted to keep the arm quiet by grasping the right thumb firmly in the left hand. On voluntary movement of the right arm the limb became grossly atactic. The right leg showed definite evidence of loss of co-ordinative power.

On the right side the tendon reflexes were brisk. No clonus. Normal plantar reflex. Babinski’s second sign (combined flexion of pelvis and thigh) present. Abdominal and cremasteric reflexes normal. There was deficient perception of all forms of superficial sensation (touch, pain, heat, cold) on the whole of the right side with the exception of the abdominal and inguinal regions. Vibratory sense was absent in the right arm and leg; there was also complete astereognosis. Sense of position was unaffected. Special senses were undisturbed. The chief subjective sensory disturbance consisted of severe pain in the right hip and shoulder. This tended to occur in severe paroxysms, especially after any period of immobility.

The position of the piece of metal causing the injury is shown in a diagram. It lay a little above and behind the sella turcica and slightly to the left of the mid-line (i.e., in the position of the left basal ganglia).

Symptoms such as these have been shown previously by Dejerine and others to be due to a lesion of the posterior and inferior portion of the external nucleus of the thalamus. Usually the patient with the thalamic syndrome presents an exaggerated response to painful and thermal stimuli, but apparently no such condition was observed in this case.

W. Johnson.

[68] **Syndrome of Babinski-Nageotte; the cerebellar and vestibular disturbances, the sensory disturbances** (Syndrome de Babinski-Nageotte; les troubles cérébelleux et vestibulaires, les troubles sensitifs).—DESCAMPS and QUERCY. *Revue Neurol.*, 1919, xxvi, 187.

After a brief survey of the literature of this syndrome (that of the posterior inferior cerebellar artery), the authors report a new case, with
exhaustive details. The patient, whose illness began suddenly with vertigo, stumbling, and vomiting, three and a half months before the first detailed examination, at once attracted attention owing to his constant right lateropulsion. At rest there was no abnormal sign; but, on standing, the patient showed a tendency to fall to the right, and on walking displayed a curvature of the trunk with the concavity to the right, and a constant deviation from the line of march, also to the right. There was a constant, slight, conjugate deviation of the eyes to the right on forward fixation, corrected automatically by a compensatory rotation of the head in the opposite direction. Appropriate tests revealed signs of a lesion of the vestibular nerve on the right side. Signs of cerebellar disease were present, all on the right side of the body.

In addition, there appeared a light oculosympathetic paresis on the right, which was followed by anaesthesia of the right cornea, and later by hypo-esthesia of the mucous membranes supplied by the right trigeminal; the sensibility of the skin being affected later, and then mainly in the distribution of the ophthalmic division. Signs of implication of the vagal nucleus were limited to a slight asymmetry of the soft palate.

Examination of bodily sensation revealed no abnormality until the end of several weeks, when the patient began to complain that his left side felt cold, and hypo-esthesia to heat and cold on this side was established. There were no signs of involvement of the pyramidal fibres, or of those conveying deep sensation.

In a brief discussion of their case, the authors point out that the dissociation of the crossed hypo-esthesia must be due to a lesion limited in its sensory effects to the spinothalamic fibres as they lie behind the olive. The syndrome, in fact, is that of a posterolateral lesion of the medulla, and the frequency of temperature anaesthesia in bulbar syndromes may possibly be explained by the fact that, of patients with bulbar lesions, it is those with the syndrome of the posterior inferior cerebellar artery who most commonly survive.

A diagram is given to illustrate the anatomy, and there are several references to the literature.

C. P. Symonds.


Various defects have been noted in frontal lesions, such as mental changes, ataxia, apraxia, dysarthria, and so on; but it can still be said without exaggeration, that so far there exists no true symptomatology of the frontal lobes. It seems to the authors, however, that deep lesions of these lobes give rise to a special clinical syndrome, characterized by defects of orientation in space, in cases with no signs of disease of the brain or vestibular apparatus. In some cases the trouble is so gross that the patient complains of his inability to find his way about; in others careful tests are necessary to unmask it. This is done by rotating or otherwise altering
the position of the blindfolded patient, and then causing him to point to objects whose situation he had previously noted. Controls were made with healthy men, and with men wounded in other parts of the brain; they showed no impairment of this function, whereas every patient with an extensive wound in the frontal lobe made mistakes, and the defect seemed to be proportional to the extent of the lesion. It is not necessary for both lobes to be affected, and the side of the lesion is immaterial, as is the direction of rotation in making the test.

The syndrome only appears when the lesion is extensive enough to affect the greater part of the fibres leaving or entering the cortex of the frontal convolutions.

W. J. Adie.


The writer draws attention to a series of cases in which signs of injury to the brain following scalp wounds varied from mild concussion to actual contusion, with apparently slight head wounds. The paper deals largely with the motor symptoms, and was written before Head's paper on cerebral sensory functions was known to the writer. Contusion of the brain was detected in 17 cases out of 54. The motor and visual evidence was most sought, so that it is likely a larger percentage would have resulted from sensory and other investigations. General signs and symptoms found were: unconsciousness, headache, giddiness, nausea and vomiting, increase of tendon-jerks, and clonus of an unsustained character. Contusional changes: the standard for a contusion diagnosis ranged from increase locally of tendon-jerks, as compared with the healthy side, to true Jacksonian epilepsy.

The chief emphasis in the paper is that apparently trivial scalp war wounds frequently result in relatively grave cerebral lesions at the same time, though these may pass off later on.

J. le Fleming Burrow.


The author enumerates the distinctions between choked disc and syphilitic optic neuritis or neuroretinitis. In the latter there may be no visual complaint, the lesion being discovered incidentally. The swelling is never more than 2–3 D, in contrast with that in papilledema. The result may be: (1) Complete resolution; (2) Secondary atrophy. The cause is the setting free of some spirochetal toxin in the optic nerve itself, attended by true inflammatory changes in the blood-vessel walls extending to the neuroglia, causing exudation, infiltration, and hemorrhages, and, later, proliferation of the mesoblastic tissues of these structures.

The treatment is urgent, and is that of the cause: it consists in eliminating the poison from the system before the local action has produced an inflammatory infiltration round the nerve fibres, by massive doses of specific medication (salvarsan and mercury), aided in severe cases by diaphoresis and purging, and with dark glasses to protect the eyes from strain.
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The author has seen no ill results upon the eyes from the effect of therapeutic doses of arsenic when in the form of salvarsan. Rabbits inoculated by him and J. A. Johnson with arsphenamin showed no ocular lesion after four months. He advises complete specific treatment in every case in which luetic lesions are found by incidental ophthalmological examination.

M. A. BLANDY.

[72] Familial optic atrophy with tremor and mental changes

A report of two cases, the subjects affected being the two elder children of a family of six.

In Case 1, a male, dimness of vision was first noticed at 18 years and progressed for three months, after which apparently his condition remained stationary up to the date of examination ten years later. Visual acuity was reduced to ability to count fingers with the right eye at 30 cm., with the left at 50 cm. There were colour blindness for red and green, and a central scotoma of $30^\circ - 40^\circ$ in each field, but no narrowing of the periphery. The opthalmoscope revealed post-inflammatory atrophy of both optic discs, without macular changes. Case 2, a sister of the above patient, showed a more or less similar condition, the picture being that of Leber's hereditary optic atrophy. No abnormality was noted in radiograms of the skull. There is no specific reference, apparently, to the sella turcica. Certain mental and cerebellar symptoms were also present in the second case.

The authors, after noting that optic atrophy is not uncommon in the hereditary cerebellar ataxie of Marie, compare and contrast their cases with those of Behr and Takashima. These, twelve in number, presented signs typical of Leber's atrophy, together with those of a mild degree of pyramidal and cerebellar disease, but were all children, in contrast with the two which are the subject of the paper under review.

The authors conclude with the hope that their cases may help to link up those of Behr to the Leber group, and establish a single type of familial nervous disease to include both.

C. P. SYMONDS.


A simultaneous affection of posterior-root ganglia, and of sympathetic fibres and ganglia, is common, although the classical authors are silent on this point. For example, when the lowest cervical or 1st dorsal root ganglia are affected, it is not rare to see paralysis of the sympathetic on the same side. Again, post-herpetic pains resemble in many ways the causalgias of peripheral-nerve lesions, which we know are due to sympathetic involvement. What is true for the trunk and limbs is also true
for the face, but here the anatomical arrangements lead to a disharmony
in the distribution of the sensory and sympathetic effects.

At present the classifications of herpes zoster of the face lack clearness;
the manifestations may be grouped as follows:

1. **Trigeminal Zona.**—(a) Complete. Vesicles in the distribution of
the three roots, with participation of sympathetic ganglia causing anaesthesia
of the cornea, alteration in the size of the pupil, and exophthalmos.
(b) Partial. Eruption limited to one or two branches. (c) Associated.
Trigeminal zona with motor paralysis of the ocular muscles or of the muscles
of mastication.

2. **Facial or Otitic Zona.**—(a) Complete. Vesicles on the ear and
external auditory meatus (geniculate ganglion); sensory and taste loss on
tongue (nerve of Wrisberg—chorda tympani); facial paralysis; diminution
of hearing, subjective auditory disturbance. (b) Partial. Vesicles on
ear; slight auditory troubles.

3. **Trigeminal and Facial Zona.**—Facial paralysis. Vesicles on ear
and on one or more of the trigeminal root areas.

The cerebrospinal fluid penetrates the aqueduct in the petrous bone
and bathes the 7th nerve, and it also reaches the Gasserian ganglion.
Inflammatory processes in the ganglia may cause an increase in cells and
albumin in the fluid. Cases showing this increase are more likely to suffer
from post-herpetic neuralgia.

W. J. Adie.

[74] Syndrome of the jugular (or posterior lacerated) foramen
(Syndrome du trou déchiré postérieur—paralysie des nerfs glosso-
pharyngien, pneumogastrique, spinal).—M. Vernet. *Revue Neurol.,
1918, xxv* (2), 117.

The author describes twenty-two cases of the syndrome, some of which have
followed the wounds of war (in which the projectile has travelled diagonally
across the base of the skull), but more frequently the cause has been an
inflammatory or compressing lesion. The syndrome is due to paralysis
of the 9th, 10th, and 11th cranial nerves, and it is pointed out that a ready
position where these three nerves can be involved by the same lesion is
the jugular foramen.

The symptoms may be arranged as follows:

1. Due to paralysis of the glossopharyngeal: difficulty in swallowing
(especially solids), owing to paralysis of the superior constrictor of the
pharynx on the affected side. On examining reflex pharyngeal movement,
‘curtain movement’ of the posterior pharyngeal wall towards the normal
side can be seen. Also some loss of taste over the posterior half of the
tongue on the side of the lesion will be found.

2. Due to paralysis of the vagus: loss of sensibility of the soft palate,
pharynx, and larynx on the affected side, also increased secretion of saliva,
disordered respiration (pseudo-asthmatic type), and troublesome paroxysmal
cough.

3. Due to paralysis of the spinal accessory: hoarseness, owing to
paralysis of the homolateral vocal cord. Also paralysis of the same side
of the palate, with perhaps nasal regurgitation. Some acceleration or irregularity of the pulse. Paralysis of the sternomastoid and trapezius on the affected side.

4. Due to other causes: (a) lesion of sympathetic fibres, which produces myosis and enophthalmos on the side of the lesion; (b) swelling high up in the neck owing to certain lymphatic glands becoming palpable.

Important points in differentiating the syndrome from disease in the medulla oblongata are: the absence of involvement of the 5th nerve, vertigo, disturbance of equilibrium, and paralysis of the 12th nerve, all of which are probable features of a medullary lesion. Traumatic cases and those due to inflammatory lesions (particularly the syphilitic variety), when treated, tend to improve. Examples of the incomplete syndrome are also given, and the paper is well illustrated with diagrams.

It will be seen that the author’s account of the distribution of the 9th, 10th, and 11th nerves does not follow the description of the English text-books.

W. Johnson.

[75] A sixteenth case of total anatomical section of the spinal cord
(Sur un seizième cas de section anatomique totale vraie de la moelle épinière. Étude spéciale du réflexe cutané plantaire).—

In a monograph entitled "An Anatomical and Clinical Study of Fifteen Cases of Complete Division of the Spinal Cord," published in 1917, the authors described in detail the symptomatology of these cases at the early stage. The duration of the illness was from several days to several weeks. Complete paralysis and loss of sensibility below the level of the lesion was associated with normal muscle tonus at the beginning of the illness, absence of tendon reflexes, and preservation of the plantar cutaneous reflex in flexion; cremasteric reflexes were often present, less frequently the abdominal reflexes; in three-quarters of the cases the so-called defensive reflexes were absent, but in 50 per cent widespread reactions could be evoked by stimulating the soles of the feet; there were retention of urine and inversion of the thermic distribution in the lower limbs.

Special attention was paid to the plantar reflex, and in fourteen of the fifteen cases a response, which consisted of flexion (downward movement) of all the toes, could be evoked immediately or within a few days after the injury. Extension (upward movement) of the toes was not observed in a single case.

According to Guillain and Barré this flexor reaction is not identical with the physiological response in healthy individuals. The response is delayed, and the movement is leisurely, progressive, and regular; it is sometimes feeble, though often quite extensive. The flexed attitude of the toes is maintained for an appreciable length of time, and the return to the original position is carried out slowly. In one patient only was the response crossed.

During the short time that the patients lived, the form of the response did not materially alter. The reaction often disappeared just before death.
Their sixteenth patient was wounded by a piece of shell at the level of the 9th rib. At the time of the first examination, six hours after the injury, there was a right hemopneumothorax and loss of sensibility and complete paralysis without hypotonia below the level corresponding to the lesion. The tendon reflexes in the lower limbs were absent, but stimulation of the sole of the foot on either side evoked distinct flexion of the toes. Movements of defence could not be evoked either by pinching the skin of the dorsum of the foot or by hyperflexion of the toes. The cremasteric reflex on both sides was present. The abdominal reflexes could not be obtained, but there was marked meteorism. There was retention of urine. The patient died a few hours after the injury. At autopsy the cord was found to be completely divided at the level of the 7th thoracic vertebra.

The authors conclude that the earliest plantar response to be obtained after transection of the spinal cord is flexion of the toes.

G. Riddoch.

[76] Autonomy of the spinal cord after complete division (Autonomie de la moelle consécutive à la section complète de l’axe spinal).

In 1890 Bastian arrived at the conclusion that transection of the spinal cord in the lower cervical and upper dorsal regions resulted in permanent abolition of the abdominal and cremasteric reflexes and of the tendon-jerks in the lower limbs, the only somatic reflex reaction to persist being a slow movement of the toes in response to pricking the sole of the foot. As early as 1898 Marinesco expressed doubts as to the validity of Bastian’s theory, and he now maintains that for its support certain conditions are essential:
1. The patient must live long enough to allow of the possibility of recovery of reflex activity in the isolated portion of the cord: that is to say, for a period of at least four weeks. (2) The elements of the reflex arcs, namely, afferent nerves, posterior root-cells, motor nerves, and muscles, must be intact, as proved in the case of the latter by electrical examination. (3) Where a pathological examination is made after death, it is necessary to obtain precise information on the condition of the root-cells of the reflex arcs of the patellar- and Achilles-tendon-jerks, the motor nerves to the quadriceps cruris and the muscles of the calf, and the fibres of the extensor muscles of the knee, especially those of the vastus internus.

A careful analysis from this standard is made of all the cases recorded by Bastian and his adherents, and the result shows that these observers prove only that, for the period immediately following transection of the spinal cord, reflex activity in the affected parts is almost completely suppressed and, when the reflex arcs are gravely injured, is permanently abolished.

Bastian’s theory has been finally refuted by the mass of evidence accumulated during the recent war from the study of traumatic injuries of the spinal cord. A large number of cases have been recorded in which the totality of the lesion had been verified at operation or autopsy. Marinesco adds to the literature two other instances of complete division of the spinal cord.
His observations, together with the findings of Claude and Lhermitte, Roussy and Lhermitte, and Riddoch, afford incontestable proof that the spinal cord, when completely isolated from headward centres by transection in the upper thoracic and lower cervical regions, is able to recover its reflex activity after a certain period has elapsed.

His general conclusions are summarized as follows: (1) Spinal man shows autonomous reflex activity, and the spinal cord is the seat of elementary reflexes. (2) This mode of activity is inferior and does not correspond, as in the decerebrate cat, to a well-defined automatism. (3) Withdrawal of the stimulated lower limb, with extension of the opposite limb, is the vestige of a co-ordinated mechanism for walking. In man more than in any other animal the ability to walk is acquired through education of superior centres, conservation of which is necessary for the presence of postural activity in the muscles of the lower limbs, above all in the extensors. Transection of the spinal cord results in disharmony between the afferents of the reflex arcs and the efferents of the extensors, though after the initial depression of function has passed off no such disharmony exists in the case of the flexors. (4) Recovery of function in the isolated portion of the spinal cord is not due to regeneration of severed fibres, though regeneration does occur. Fibres of the posterior roots, as well as those of the white substance of the cord (Marinesco and Minea, Rossi, Cajal), take part in a regenerative process; but it is purely anatomical, and does not serve any useful purpose. Moreover, the fibres of the descending tracts show no sign of regeneration. (5) Postural tonus in the extensor muscles is permanently abolished, and primary extension reflexes cannot be excited by stimulation of the proximal part of the lower limb.

G. Riddoch.

[77] A special lesion of the anterior and posterior roots in crushing of the cord from spinal fracture (Sur une lésion spéciale des racines antérieures et postérieures dans la section par écrasement de la moelle consécutive aux fractures du rachis).—J. Lhermitte. Revue Neurol., 1919, xxvi, 185.

Permanent abolition of deep and cutaneous reflexes in the paralyzed parts, after the phase of spinal shock has passed, may be due to several causes: (1) More or less complete destruction of the lower segment of the cord; (2) Lesions of peripheral nerves; and (3) Lesions of posterior roots. The author has observed another modification of the radicular system in two cases of complete division of the spinal cord in the dorsal region. The first patient was injured by the roof of a dug-out falling on him. He lived for two months, and the only reflex response that was obtained in the lower limbs was flexion of the small toes on the right side and extension on the left. The second patient fell from a height of three metres and was immediately paralyzed from a level corresponding to the fourth thoracic segment. Flexion of the toes could be evoked by plantar stimulation, but all other reflexes were abolished in the trunk and lower limbs. He died three days after the injury.

At autopsy complete separation of the cord was found at the level of
the lesion. The extraspinal portion of the posterior roots was swollen. Histologically the swollen parts were composed of alveolar tissue, masses of myelin in process of disintegration, nerve fibres, and vacuoles. In the dorsal region Clarke’s column was displaced backwards and the central canal of the cord was distorted. Gross changes of a similar nature were also present in the anterior roots and anterior horn cells, and Lhermitte believes they are due to stretching of the spinal axis and roots.

G. Riddoch.

[78] Two cases of syringomyelia or syringobulbia with nystagmus (Zwei Fälle von Syringomyelie bzw. Syringobulbie mit Nystagmus).

The author quotes with approval the conclusions of Leidler (Zeits. f. Ohrenheilk., 1918, Bd. 76), that :

1. Nystagmus in syringobulbia is produced by a lateral fissure in the medulla oblongata, which, usually unilateral, extends from the region of the central canal towards the descending root of the 5th nerve, and involves the roots of the 9th, the nuclei of the 10th, sometimes the nucleus of the 12th, the internuclear roots of the 8th, and rarely parts of the 7th. The fissure never extends beyond the level of the exit of the 7th nerve.

2. Nystagmus occurs when the fissure damages even a small part of the descending root of the 8th, or the fibres which pass from it to the posterior longitudinal fasciculus.

3. The vestibular reactions in these cases are normal.

4. In the rabbit, after section of the arcuate fibres to the caudal part of Deiters’ nucleus, the nystagmus is rotatory; to higher parts, horizontal.

5. The findings in the rabbit and in man agree completely.

Levy-Suhl describes two cases with nystagmus and normal vestibular reactions.

W. J. Adie.


Following the experimental observations that in artificial septicemias in animals the withdrawal of spinal fluid invariably led to the development of a fatal meningitis, whereas in control animals, not punctured, meningitis did not occur, the following clinical research was undertaken:—

In 93 patients where lumbar puncture was performed to confirm a diagnosis of suspected meningitis, 38 gave a positive turbid fluid at the first puncture. In 55 cases a clear fluid was obtained, and in 6 of these a blood-culture taken at the same time gave a positive growth, 3 meningococcus and 3 pneumococcus. Of the 6 patients 5 subsequently developed a clinical meningitis, the sixth case (of pneumococcal septicemia) went on to complete recovery. The 5 cases which showed clinical meningitis, as evidenced by subsequent punctures and post-mortem findings, seemed to develop meningeal symptoms soon after lumbar puncture, unless a previous intravenous injection of serum had been given.
The authors emphasize that lumbar puncture performed during a septicemia is fraught with the danger of localizing the infection in the central nervous system. They summarize their work by stating that infection of the meninges occurs frequently after the release of normal spinal fluid by lumbar puncture during a septicemia. They urge that a blood-culture be taken before lumbar puncture for diagnosis, so that the blood may be proved sterile, and danger of meningeal infection, by way of the choroid plexus, or locally by needle trauma, is at least minimized. They also stipulate that minimal amounts of cerebrospinal fluid be withdrawn for laboratory tests, and that small-bore needles be used, so that the local trauma is small, and subsequent leakage of spinal fluid into the surrounding tissues will be avoided as far as possible. A full review of the literature is quoted.

J. le Fleming Burrow.


From the examination of some 246 cases of spastic hemiplegia or paraplegia ('spastic syndrome'), Bing has observed, inter alia, the following points:—

1. In pure cerebral cases the extensor plantar response is but rarely accompanied by simultaneous contraction in proximal muscle-groups of the limb; in spinal cases, on the contrary, such contractions are extremely common: thus in 79 cerebral cases only 10 showed a contraction proximally (tendon fasicie latè, adductors, quadriceps), but of 56 spinal cases no less than 45 did.

2. Between these is a cerebrospinal group (disseminated sclerosis, amyotrophic lateral sclerosis, etc.) in which some 30 per cent of cases gave proximal contractions with the Babinski sign.

3. The phenomenon of involuntary flexor contraction (flight-reflex, shortening-reflex) is especially apt to accompany the extensor response if the lesion is any kind of compression of the cord.

4. Gordon's paradoxical reflex (toe-extension on deep pressure over gastrocnemius group) was found 27 times in 188 Babinski-positive cases; it is rarer in cerebral than in spinal cases, and appears to be indicative of slight degrees of spinal compression, in Bing's experience.

5. A crossed plantar response in extension was noted only 6 times, exclusively in unilateral cerebral cases.

6. In transverse lesions of the cord, extension of the reflexogenus zone for the Babinski phenomenon to the instep, ankle, etc., is in general a sign of the severity of the lesion: this does not, however, appear to be the case for cerebral lesions.

Wilson.


Five cases are described of peripheral neuritis beginning with distal atrophy and weakness, and slowly spreading towards the proximal segments of the
limbs. In four of the cases the symptoms started in the hands. The disease is slowly progressive. The clinical picture is typical of peripheral neuritis, viz., subjective pains with acroparesthesiae, wasting, weakness, slight sensory disturbances, and changes in electrical reactions. The characteristic feature is the tenderness and increase in size of the nerves, on which localized enlargements can sometimes be felt.

The etiology is unknown; the disease is not hereditary or familial, and syphilis can be excluded. Similar cases have been described by Long and Hoffmann in which, apart from the peripheral neuritis, no other evidence of disease of the nervous system was found.

J. L. Birley.


*Lateral Flexion of the Trunk.*—If a patient with sciatica stands with hands on hips, and attempts to bend the trunk, first to the right and then to the left, without flexing the knees, the movement is limited, most often towards the affected side, rarely towards the normal side. The limitation is well marked in cases with a crossed scoliosis, i.e., with the concavity towards the unaffected side.

*Dorsiflexion of the Foot.*—With the patient on his back with limbs extended, the foot is suddenly dorsiflexed. This elongates the nerve, especially the posterior tibial branch, and the patient reacts by flexing the limb at the knee and hip. He complains of pain in the calf, or along the course of the nerve. This test is useful in those cases where the pain is greatest in the internal or external terminal branches of the nerve.

*Internal Torsion of the Foot.*—When the foot is suddenly rotated inwards, pain is felt on the external aspect of the leg, and the limb may be flexed. This sign is not obtained so often as the preceding one, no doubt because the internal popliteal nerve is more sensitive than the external popliteal.

The tests have been made on many cases during three years, and have been found useful where the diagnosis was otherwise doubtful.

W. J. Adie.


This paper contains a neurological record of the methods used in caring for peripheral nerve cases in No. 1 American Red Cross Military Hospital, and a report on the results following nerve suture. The material included 857 histories of peripheral nerve injuries, and the records of 205 reparative nerve operations. The following conclusions are drawn:—

1. The musculospiral is the nerve most frequently injured in war, the ulnar nearly as often; the sciatic is next; and the external popliteal, which is fourth in order of frequency, is involved more than twice as often as the internal popliteal.
2. Following operation, the musculospiral and sciatic nerves make the best recoveries, the results in the case of the sciatic being equally as good as those of the musculospiral.

3. The condition of an injured nerve, when examined by sight and touch at the time of operation, is invariably worse than the previous clinical findings would lead one to expect.

4. When, at the time of operation, all the methods to determine whether simple liberation or excision and suture is the best procedure having been utilized, doubt still exists, excision and suture should be performed.

5. Repair of an injured nerve as early as possible should be the aim of every surgeon.

6. Patients convalescing from nerve reparation should be encouraged to use the extremity affected, for volitional effort plays a part in the return of function.

7. The more respect the surgeon shows nerve tissue when repairing an injury, the better will be his results. The nerve should be stripped and handled as little as possible, and the ends should be so approximated as to place in apposition corresponding fasciculi of the cut nerve.

The writers do not attach much importance to Tinel's sign as evidence of nerve regeneration.

R. M. S.


This paper is a plea in favour of the view, first put forward by the writer in 1911, that the type of birth palsy, known as the 'Erb-Duchenne type', is due rather to a posterior subluxation of the shoulder-joint, with a secondary lesion of the nerves in the axilla following upon inflammatory reaction, than to any trauma to the 5th and 6th cervical nerve-roots during parturition. The writer points out that the commonly accepted original views of Erb and Duchenne rest upon papers published in 1872 and 1874, and that these were based on electrical reactions of the affected muscles. The writer suggests that a series of cases electrically examined would probably dispose of the old views in favour of a shoulder-joint lesion, at least in the vast majority of cases. Much experimental detail worked out upon the bodies of still-born infants is quoted in favour of the author's views, and a paragraph on treatment soon after birth closes the paper.

J. le Fleming Burrow.

TREATMENT.


The method of corpus-callosum puncture, introduced by Anton in 1908, is simple, practicable, and worth trying in suitable cases. A spot about