NEUROLOGY

Cerebrospinal Syphilis.—In roughly one-half of the cases of the author's series all four reactions were positive; in two-thirds of the cases the Wassermann reaction was positive in the blood, and in about 80 per cent the positive Wassermann test in the blood, the pleocytosis, and the positive globulin reaction occurred in a quite independent way. There can be no doubt that in this group the result of salvarsan treatment in reducing the positive states of the tests is much more marked than in either of the two former groups.

A more difficult point to determine is whether, from the tests, general paralysis and tabes on the one hand can be separated from meningitic, gummatous, or vascular neurosyphilis on the other. From the author's figures it is clear that the tests are not of much assistance in this respect, except in so far as the conclusion is justified that, if the reactions improve rapidly under salvarsan treatment, the condition is likely to be one of cerebrospinal syphilis. In tabes, and still more in general paralysis, the pathological state of which the tests are the expression proves much more refractory even to persistent treatment.

S. A. K. W.

SENSORIMOTOR NEUROLOGY.


In well-established hemiplegia the crossed plantar response is usually bilaterally flexor, though it may be extensor in a few cases. In cases of myelitis and transient intermittent paralysis there may be no plantar reflex obtainable on the paretic side, but the crossed reflex may be extensor, showing that serious change may take place in the pyramidal system and be confirmed by the subsequent development of paralysis. To produce the crossed reflex the stimulus must be strong, whether applied by the methods of Babinski, Oppenheim, or Gordon.

R. G. Gordon.


The author describes a sign which he claims has not been noted previously, and which consists of a small point of tenderness just to the left of the spinal column, corresponding to the 5th dorsal interspace, or about that level. It is always found on the same side as the stomach, and may be an area covering more than one space. The tips of the thumbs or fingers are stroked across the areas in question, and a positive sign is elicited if the patient winces or complains of discomfort. The sign persists for a few days before and after a gastric attack, and in the intervals the tenderness subsides. The origin of gastric crises is shortly discussed, the author suggesting that "the 'central' (cerebral) origin heretofore largely assumed" is unlikely to be the true origin of the attacks. The above statement is unexpected, as since the days of Charcot the spinal origin of lightning pains and other 'crises' has held the field to the exclusion of all others in medical opinion. Treatment by counter-irritation over the hyperalgesic...
area is recommended, with internal administration of a powder containing \( \frac{1}{4} \) gr. each of calomel, brucia, and powdered ipecacuanha. The author evidently writes from a small clinical experience of gastric crises in tabes.

J. LE F. B.


Twenty cases are analyzed. The following symptoms and signs are noted:

Prodromata.—In 18 of the cases seen during the first week of the illness catarrhal symptoms were observed, especially of the nose, like those associated with influenza; there were also pains, headache, frequently vomiting and constipation. These lasted three to seven days. The incubation period was not determined, as no two cases occurred in the same family. In two instances the onset was sudden, with delirium, and in one there was lethargy. Headache was present in all cases, and vomiting in 8. The pyrexia average was seven days. The age incidence was 9 to 78 years; sexes affected, 12 male to 8 female.

Cranial nerves.—In 17 cases diplopia occurred, lasting two to four days. The various nerves were affected as follows: the third nerve in 18 cases, the fifth in 9, sixth in 6, ninth in 7, and the eleventh in 8 cases. The facial was frequently involved, and always more on the right side.

Symptoms.—Apathy or coma was striking, lethargy lasting two weeks to three months. Delirium was observed in 17 cases, catatonia in 16, ataxia of intentional type in 18, choreiform movements in 11; muscular fibrillation was noted in every case to be a striking feature, often local, but occasionally general in distribution. Tremor, coarse and of intentional type, was also constantly observed. Parkinson’s mask was present in all but one of the cases. An eruption was found in 3 cases, in 2 herpetic and in 1 purpuric. Paralysis in the arms and legs was noted in 5 cases, in all of which there was polyneuritis. Euphoria was present in all the cases except those with polyneuritis. All were asthenic. The reflexes showed no constant departure from the normal, and in none was a Babinski’s sign obtained. The cerebrospinal fluid showed no change from the normal in its reactions to the various tests, including the Wassermann reaction. Of the 20 cases, 3 died, 1 patient is still very ill at the time of writing, 15 are better, and 1 is quite well.

J. LE F. B.


This short article briefly records the history of the disease and attempts to correlate recently described syndromes with the epidemics described by Jean de Troyes, Ambrose Paré, Sydenham, etc., in the past. Some recent pathological and bacteriological researches are mentioned, particularly those of Bassoe, von Wiesner, McIntosh, Ayer, and Wegeforth. Von Wiesner’s success in transmitting the disease to monkeys from fatal human cases is quoted as placing beyond reasonable doubt the communicability of
encephalitis. Cleland and Campbell's work in Australia with the glycerinated virus, and their success in conveying the disease to five monkeys, and thence through fourteen generations from monkey to monkey, thence from monkey to sheep, horse, and calf, and finally from sheep back to monkey, are all duly summarized. Work by Loewe, Hirschfeld, and Strauss on a filter-passing virus obtained from the nasal washings of fatal cases, and their successful cultivating of small globoid bodies, are quoted as positive evidence of an infective agent, though it is admitted that Flexner and Amoss were unable to confirm these findings.

The questions of carriers, susceptibility, etc., are raised on a broad basis of epidemiology. It is suggested that the infective agents of influenza, poliomyelitis, and encephalitis may spring from a common protozoon stock, though the writer does not attempt to justify his speculation by evidence in its support.

J. LE F. B.


Fifteen personally observed cases of encephalitis, and an analysis of one hundred cases from the American literature, form the basis of this paper. With regard to etiology, the writers suggest that the globoid bodies (Flexner's filter-passers in poliomyelitis), or the Rosenow streptococcus with globoid bodies, might be considered as streptococcal varieties. From one of their own patients, who at the time was suffering from influenzal bronchopneumonia and developing encephalitis with lethargy and diplopia, the writers were able to cultivate a green streptococcus from the blood. This organism agglutinated with the serum of the patient only, and remained unaltered when other sera were used. The patient's serum did not agglutinate the ordinary laboratory strains of diplococci and streptococci. Unfortunately the culture died out before any animal experiments were tried, and could not be replaced.

Among the special symptoms paresis of the third and sixth nerves was frequent. The third nerve was affected in 63 cases out of 115, and the sixth in 39 cases out of the same number.

The trigeminal showed signs in only 9 cases, and its motor division was rarely involved. Facial palsy, often unilateral in distribution, occurred in 15 cases. There was rapid respiration in 3 cases, one of which presented a respiration-rate of 60 per minute for thirty-six hours. In one of the three the patient had attacks of rapid breathing up to 80 or 100 per minute, but here the tachypnoea seemed to be due to myoclonic movements of the diaphragm. Lethargy occurred in 79 of the 115 cases. Out of the 15 personal cases, catalepsy and catatonia were noted in 6 patients and in 20 of the 100 from the literature, while headache formed a prominent symptom in 54. The authors draw attention to the fact that rigidity, or rather increase in muscle tone, is common in the disease, though it ought not to be confused with the more marked rigidity of meningitis where Kernig's and Brudzinski's signs are usually obtainable.
Tremor was noticed in 35 cases, coarse and not of the intention type; muscle twitching was a feature in 2 of the 15 personal cases. Changes in the reflexes were inconstant; 18 cases had a transient Babinski’s sign, and Oppenheim’s reflex was elicited in 5 of the 15 cases seen personally by the authors. Peripheral pain was present in 26 of the 115 patients, but the writers suggest that it is a commoner symptom than appears from these figures, as in their own 15 cases there was a definite neuritic pain complaint in 6. In 6 patients of the writers’ 15 there was increased sweating amounting to night sweats, a fact of interest when compared with the ‘sweating sickness’ of former times. Pyrexia was transient, but present in all except 50 cases. A leucocyte count was made in 86, with an average result of 10,000 per c.m.m., and of these 72 per cent were polymorphonuclears. Blood cultures were negative except in 1 of the 15 personal cases. The cerebrospinal fluid was always clear, but under increased pressure in 7 of the 15. Mononuclears averaged 9 per c.m.m. The globulin test (Nonne Apelt) showed an increase in 4 cases. All the Wassermann tests were negative. The gold chloride curve was mildly luetic in 7 out of 11 which were examined.

The mortality was 81 per cent for the whole series, and 4 out of the 15 personal cases died. Four cases recovered wholly in one and a half to four months. The fatal cases lasted a few weeks only. No special treatment was adopted except repeated lumbar puncture, which seemed definitely to relieve 10 of the 15 cases personally dealt with. The writers give a very complete summary of the literature to date.

J. Le F. B.


The patient was a woman of 50, who as the result of an acute illness of some weeks’ duration presented the symptom-complex of generalized rigidity of all limbs, with some contracture in flexion of the extremities, extreme slowness of voluntary movements, immobile facies, spasmodic laughter, katatonia, and general apathy. Tests for syphilis were negative in blood and fluid, nor was there any evidence of disease of the corticospinal tracts. A diagnosis of encephalitis involving principally the lenticular nuclei was made during life, and was corroborated at autopsy, when these ganglia were found to be the seat of marked congestion and small-cell infiltration of the vessels, with neuronophagia and disappearance of the nerve-cells. In his comments on the clinical significance of his case, the author has some trouble in explaining the absence of any involuntary movements of the nature of tremor, and suggests that this absence was due to the acute nature of the illness. It might, however, have been pointed out that, in any condition belonging to the paralysis-agitans group, the clinical picture may vary through all intermediate stages from severe rigidity without tremor to pronounced tremor with little or no rigidity.

S. A. K. W.
Acute infectious enteritis with a polyneuritic syndrome.—

An account is given of an epidemic which occurred at the State Hospital for Mental Diseases, Howard, Rhode Island, in the summer of 1917. As a preliminary to their account the writers recall work by Orr and Rows, and by Homen and Laitinen, on infection of the spinal cord via the lymph-spaces following the injection of peripheral-nerve trunks. They point out that one of two lesions may result, either an acute inflammatory reaction with secondary degeneration, or an acute degenerative process, the 'primary' type of lesion.

The epidemic in question was confined to the female wards of the hospital, and resulted in the infection of 47 patients, as well as 4 employees. The onset of symptoms was characterized by acute gastro-intestinal symptoms, nausea, vomiting, diarrhoea with mucus and blood in the stools, and pyrexia of 100° to 104° F. Headache and backache were also complained of. The neuritic symptoms were those of acute peripheral neuritis with pain on pressure over the nerve-trunks, or even upon light touch of the surface in some cases. Both upper and lower limbs showed ataxia, loss of tendon-jerks, and in some of the cases a flaccid paralysis. The epidemic was traced to infected milk from which Staphylococcus aureus was grown in excess of all the other organisms found. The blood, feces, and urine of the patients were also found to give positive growths of the same organism. The infection was looked upon as haematogenous via the intestinal tract, which showed the changes associated with acute gastro-enteritis in the four post-mortems made. The peripheral nerves at autopsy showed an acute haemorrhagic polyneuritis. No poliomyelitis was detected.


The patient was a soldier wounded in 1916 by a fragment of shell which had produced a large lesion of the cranial vault on the left side over the posterior part of the frontal lobe. When examined some months later, the chief clinical symptoms which he showed were katatonia of the limbs, absence of spontaneity and initiative, and speech disturbances of the nature of agrammatism. The author describes the latter at great length, pointing out that there was no defect on the receptive side, and that the agrammatism involved expressive speech only. Further, there was no evidence that it was a mere phase in a recovering aphasia, for it had existed unchanged from the beginning of the illness. The author discusses various theories which have been advanced to explain agrammatism, and concludes that his case favours the localization of the lesion, underlying it in the frontal lobes; he emphasizes the fact that in some of the recorded cases, where agrammatism has been associated with motor aphasia, the pathological evidence did not point to involvement of the temporal lobe, though Pick associates agrammatism with temporal lesions.
The reviewer may point out that Maas, in a paper on the same subject (see Abstract No. 15) criticizes, not very convincingly, perhaps, the description given by Forster of his case as agrammatism, and considers that it belongs rather to the pseudo-agrammatism group.

S. A. K. W.


The difficult subject of aphasia is not made any easier by difference of opinion as to the exact definition to be given to agrammatism, a clinical feature of a large number of cases in the aphasia group. After quoting the views of a number of writers, the author follows Pick and Kleist in considering that the essence of agrammatism is inability to form sentences correctly, and that defects in conjugation, declination, and so on should be regarded as a pseudo-agrammatism. He quotes several cases which appear to show that agrammatism in the strict sense is the result of lesions of the temporal lobe, and narrates in considerable detail two cases of his own.

The first was that of a man of 51 who presented the condition in characteristic form. At the post-mortem an enormous lesion was found involving almost the whole of the speech area of the left hemisphere and, from its position, undoubtedly cutting off to a large extent all communication between that area and the corresponding parts of the right hemisphere. The second case was that of a man of 36 who similarly showed agrammatism, particularly that form of it which is known as the ‘telegram style’ of speech. In this second case the lesion was found to be a large cyst involving the greater part of the left temporal lobe and also the lower half of the central gyri. In both of these cases a phase of practically complete aphasia had been followed by considerable return of speech, both on the motor and on the receptive side, leaving the agrammatism as above mentioned. Very briefly, the author’s chief conclusion is that there is no ‘centre’ a lesion of which produces agrammatism, but that this condition is the result of an attempt on the part of the right hemisphere to assume speech functions as a whole when it is in reality less well equipped for so doing than the left.

S. A. K. W.


The authors distinguish extradural, extramedullary, and intramedullary tumours, of which the intradural extramedullary group is the most important from the therapeutic point of view. They divide these into tumours whose density is greater, and tumours whose density is equal to or less, than that of the cord. Extramedullary tumours of the former class commence by causing root signs at the site of the tumour, and as they enlarge may show signs of all stages of compression of the cord up to
complete transection. The level remains practically unchanged throughout the course of the disease. A tumour of the latter class causes little compression, but may interfere with vascular and lymph supply and cause oedema of the cord. The root signs will be less intense, and the compression signs slow in development and relatively slight. The signs of involvement of motor and sensory tracts will be most marked in the region of the tumour, and just above and below it, spreading downwards with diminished intensity, whereas in hard tumours the signs of sensory-tract involvement are greater in the lower spinal segments. In soft tumours there is considerable parallelism between sensory and motor signs, whereas in hard tumours the motor signs are earlier and more marked. In soft tumours the level of involvement may move upwards through two or three segments, xanthochromia is more common, and remarkable exacerbations and remissions in the course of the disease may occur.

_Differential Diagnosis between Extramedullary Soft Tumours and Intramedullary Tumours._—(1) Pain is present from the beginning in all soft tumours, but only in those intramedullary tumours in which the tumour reaches the posterior roots or surface of the cord. Pain in intramedullary tumours is, therefore, usually a late phenomenon, or may not occur at all if posterior roots or the meninges are not affected. (2) In the vast majority of cases the symptoms develop more rapidly in extramedullary soft tumours (3) Tract sensory signs and symptoms are more intense and more widespread in intramedullary tumours. (4) Trophic changes are greater in intramedullary tumours. (5) Intramedullary tumours show less rectal and vesical signs. (6) Xanthochromia is rare in intramedullary tumours; it appears only when the tumour reaches the surface of the cord. (7) Deep spinal-column tenderness indicates rather an extramedullary tumour.

_Signs Pointing to the Site of the Tumour._—(1) _Valuable signs:_ (a) Root signs, consisting of neuralgic pains with symptoms pointing to a distal distribution of an affected nerve; (b) Zones of hyperesthesia immediately above the seat of the tumour and more marked at the homolateral site; (c) Level abolition of skin and tendon reflexes; (d) Deep spinal tenderness; (e) Alteration in vibratory sense; (f) No sweating below the level after injection; (g) Ocular symptoms, such as lateral nystagmus and difference in the pupillary size as well as the palpebral fissure, which may all be present in high cervical tumours. (2) _Less reliable:_ (a) Homolateral paralysis; (b) Heterolateral disturbance of pain and temperature sense, reaching its highest level only after considerable cord compression, late in the disease; (c) This sensory loss, when complete, is usually three segments below the actual level of the tumour.

The authors' conclusions are: (1) Progressive spinal-cord diseases giving level signs and symptoms should be carefully observed and studied. (2) When no distinct level can be established, the effect of lumbar puncture should be carefully watched. (3) Xanthochromia of the spinal fluid in level spinal-cord progressive affections usually means spinal-cord tumours. (4) One should operate only when a level has been established. Probing above and below the suspected site, while useful in many cases, often fails.
(5) Operations, frequently by their decompressive effects, help even in intramedullary tumours. (6) The possibility of a soft tumour in atypical level cord lesions must be borne in mind, as these are most frequently overlooked.

R. G. GORDON.

[17] Sacralization of the fifth lumbar vertebra, and the troubles which result from it (La sacralisation de la cinquième lombaire, et les accidents qui en resultant).—G. NOVÉ-JOSSE RAND and A. RENDU. *Presse méd.*, 1920, xxviii, 514.

It is well known that the 7th cervical, the 12th dorsal, and the 5th lumbar vertebrae possess attributes of the vertebral region immediately below them, which may be present in such marked degree that the particular vertebra partakes entirely of the character of the region below. When this happens to the 5th lumbar vertebra, it is spoken of as its sacralization. The change may only occur in one half of the vertebra, the other portion remaining normal. Patients with this condition have complained of severe pains of sciatic or lumbago type. Radiography represents clearly the nature of the condition. It has been found in just under 2 per cent in a series of 400 radiographs taken at random, and in nearly 3 per cent of a series of 800 cases where lumbar pain was complained of. This morphological variation of the 5th lumbar vertebra therefore deserves recognition by the clinician. In a well-marked case the transverse processes are much increased in size and tend to approach the shape of the wings of the sacrum. Sometimes they articulate with these latter, and at others are firmly united to them.

The chief clinical manifestation of the condition is pain, but this may be an inconstant symptom. Indeed, the malformation may remain latent indefinitely. As a rule, however, pain is first noticed between the ages of twenty and thirty years—which points to some relationship between the symptoms and the termination of the ossification of the pelvis. The pain may be dull and continuous, or occur only in neuralgic attacks. It is usually in the lumbar region, and may radiate extensively from this—especially towards the sciatic region. The painful region is tender on deep pressure. Some local deformity of the spine may be found; as a rule this is a tendency to lordosis.

As for the cause of the pain, soft structures may be pressed upon, or nerve-trunks may be interfered with or put on the stretch. This applies more especially to the 5th lumbar root, in the distribution of which hyperaesthesia, muscle wasting, and altered deep reflexes may be found.

An interesting association occurs between this condition and spina bifida—the latter being present in about 10 to 15 per cent of the cases, and pointing possibly to a common pathology, i.e., a developmental abnormality.

The treatment has been palliative (drugs, etc.) or radical (removal of the abnormal transverse processes). In four cases operative interference produced some improvement, but as a rule the clinical condition is not sufficiently severe to justify operation.

W. JOHNSON.
[18] **Hemiplegia in pregnancy** (L'hémiplégie pendant la grossesse).—

Puerperal hemiplegia, so-called, includes a rather heterogeneous collection of pathological states from which two main groups emerge: (1) Hemiplegia following the confinement, and (2) Hemiplegia during the pregnancy itself. The author bases his review of the latter condition on an analysis of some 46 cases, personal and from the literature. Apart from the ordinary causes of hemiplegia (syphilis, cardiac disease, etc.), to which of course the pregnant woman as well as any other may be liable, certain varieties of hemiplegia are apt to be associated with pregnancy. In 17 of the 46 cases the patient, although often enough quite young, suffered from a cerebral hæmorrhage. The physiological hypertrophy of the left ventricle, and increased richness of the blood, considered by some as capable of explaining the cerebral hæmorrhage, are in reality insufficient, and an additional factor seems to be necessary. It is true that during the actual labour a cerebral hæmorrhage of mechanical origin may occasionally occur, but the great majority of cases are attributable to the presence of renal disease—the albuminuria of pregnancy. Even in these cases, however, the possible effect of localized cerebral œdema and of the toxic elements in the uremic state ought not to be ignored. Next to these albuminuric cases, embolism from a pre-existing cardiac condition seems to be the second most common cause of hemiplegia in pregnancy, whilst the third most common variety is meningeal hæmorrhage. A number of cases of this kind are on record, but the explanation is unfortunately not clear. Meningeal hæmorrhage may occur in the absence of any albuminuria or cardiac disease, or any evident visceral or toxi-infective condition. One ingenious theory is that increased activity of the suprarenal glands in pregnancy, as revealed by a condition of hypercholesterinæmia, leads to an increase in blood-pressure from time to time and so to meningeal hæmorrhage. This variety of hemiplegia is, as a rule, progressive, and can be diagnosed with accuracy by means of lumbar puncture.

It will be seen that the hemiplegias of pregnancy are not the direct, but the indirect, result of that state. Pregnancy exercises on their appearance a mechanical, toxic, or dysserasic influence.

S. A. K. W.


In infantile hemiplegia the weakness is sometimes confined to the ankle movements. If the knee movements are found to be defective in a given case, then the ankle is, as well, invariably involved. Finally, if the hip movements are weak, then both knee and ankle movements are found to share in the disability.

Clinically the degree of paralysis is to be gauged by: (1) The amount of contracture; (2) The active power still remaining in the weakened muscles.

1. The amount of contracture must be investigated carefully in the
case of each joint. It is found to diminish as the proximal portion of the limb is approached. (a) In the examination of the foot, the subject is made to lie on his back on a table, with the legs from the knees downwards hanging over the edge. In this position the feet roughly form a right angle with the legs. On now raising the foot on the affected side until the whole leg is in a straight line, a position of equinus—to a greater or less degree—becomes assumed by the foot, the amount of the equinus being dependent on the degree of the contracture present in the calf muscles. (b) Similarly, when examining the degree of contracture of the knee-joint, the subject is made to lie on his back, but this time the legs lie horizontally on the table. The child is then aided into a sitting position, and the knee of the hemiplegic side is at once seen to rise from the table and assume a semiflexed position. This is due to contracture of the hamstring muscles, and is comparable to Kernig’s sign. (c) In order to investigate the state of the hip flexors, the subject is placed in the prone position and the heels are drawn up towards the buttocks. In this position, owing to the contracture of the iliopsoas and the tensor fasciae femoris, the hip on the affected side fails to extend to the full amount required, with the result that this side of the pelvis becomes raised from the bed. These phenomena are well shown in the diagrams illustrating the article.

2. As regards the power present in the weakened muscles, it is the slowness of the voluntary movements which is characteristic of hemiplegia—this slowness being due to agonists having to overcome the contracture of antagonists. The carrying out of the simplest volitional movements serves for the demonstration of this condition.

The gait in infantile hemiplegia depends on the extent of the paralysis. If the contracture is limited to the muscles which move the ankle, the child walks on the ball of the foot, keeping the knee flexed until almost the end of his step. Where there is knee contracture as well, the knee remains flexed continually whilst walking; and in the third variety, where the foot, knee, and hip all exhibit some contracture, the affected leg can only function somewhat like a stilt. Owing to this stilt-like movement of the hemiplegic leg, the trunk is made to perform a ‘bowing’ movement during the process of walking (mouvement de salutation).

TREATMENT.—The analysis of the degree of contracture and of the amount of residual movement in the weakened limb forms a basis for therapeutic measures. The muscles involved should first be treated by passive movements directed towards diminishing their contracture. These must be followed by simple re-education exercises designed to develop the power and rapidity of their contraction. Special measures must be taken for dealing with the equinus condition—these varying with the degree of deformity which may be present.

W. JOHNSON.


The author’s system is based on a formula of Todd—“I know of nothing so profitable for the paralyzed members as a regular system of exercise,
active when the patient is able to perform them, and passive if he be incapacitated”. Methodical massage and motor re-education improve the nutrition of muscle and fibrous tissue, and in addition re-establish co-ordination of movement. The statistics of the Salpêtrière gathered by Possard over the last five years show that of the cases of hemiplegia so treated, 3 per cent were completely cured, 17.7 per cent were greatly benefited, 64.7 per cent showed notable improvement, while in 17 per cent the result was doubtful. As a preliminary, careful digital exploration of the muscles and estimation of their tone are made before massage is started. The persistent absence of any ‘muscle shock’ on attempted movement is regarded as prognostically unfavourable. Examination reveals the muscles most affected by the paralysis, but it is not possible, in the writer’s opinion, to make a useful topographical table therefrom, as the same muscles are not affected alike in a series of hemiplegics. It is noticed that the muscular atrophy of hemiplegia depends more upon the contracture of antagonists than upon the severity of the paralysis. Quoting Brissaud—“Muscular atrophy in hemiplegia is always accompanied by contracture, which is followed by flaccid hemiplegia”—the writer states that in all his cases of residual hemiplegia atrophy was present.

Further details in the treatment are the employment of superficial and deep massage, with light longitudinal and circular pressure at first, followed by digital vibrations; such treatment is adopted as early as twenty days after the attack. The later massage consists of percussion, progressive kneading, and varieties of tapping, all movements to be made gently, and limited to the affected portions of muscle. In massage of the nerve-trunks, combined longitudinal and surface pressure with one hand and vibrations with the other are recommended. Where some muscles are spastic and others hypotonic the latter alone are treated, while the former are relaxed as far as possible during the treatment. Massage is gradually succeeded by passive and active movements. It is remarked that in general the patients do not like to move the affected limbs, but they usually desire to walk at the earliest opportunity; hence the almost invariable sequel that the affected lower limb recovers before the upper member. The author does not add, as a qualification to this statement, that the more recently evolved complex co-ordination of the upper limb in man certainly explains much of the disparity in recovery of function between the upper and lower limbs of the hemiplegia. When the patient is able to perform a few active movements he is then started upon a course of muscle re-education. The tendency to associated flexion movements in the arm is countered by education with the elbow extended while the movements at the shoulder are being encouraged. When the shoulder movements are established, elbow flexion is allowed, and the hand is next educated in grasping large objects, such as a tumbler, etc. The act of walking is re-educated by active leg extension until the affected foot reaches the ground. The patient is invariably made to place his affected leg first in taking a step; this is in contra-distinction to Erben’s method, where the strong leg is advanced first. The writer believes that the advancing of the affected leg first will
correct the dragging defect so commonly present in the gait of a hemiplegic. Flexion movements of the affected leg are taught with the patient sitting. The glutei and external rotators are exercised in a co-ordinated walk as follows: (1) Flexion of thigh on pelvis; (2) Extension of leg; (3) Exaggerated rotation of foot outwards; (4) Displacement of affected limb.

The good results of the whole treatment are explained on the assumption that re-education creates supplementary centres which replace those destroyed. The simple appliances used are a double inclined platform, a rolling chariot, benches of various heights, and traction appliances with weights and pulleys.

Adjuvants are heliotherapy, thermotherapy, and balneotherapy; but electric treatments are deprecated on account of the tendency they have to increase the contractures.

J. Le F. R.

[21] Condenser tests in the diagnosis and prognosis of nerve injuries.


These observations are based upon experiences with some 2000 cases of injury to nerves of the upper and lower limbs seen while the author was acting as neurologist to the Woolwich Military District from 1916 to 1919. It is pointed out that the faradic coil is not an accurate instrument for testing muscle reactions, since the factors upon which the response depends—voltage, duration of each electrical impulse, rate at which the impulse is delivered—are variable; further, the majority of cases of nerve injury fail to show any response to the faradic coils in clinical use. Galvanism, as ordinarily used for testing, is similarly inaccurate, for voltage, milliampère readings, resistance, etc., require standardizing. In the condenser set the voltage is fixed and the duration of each impulse known; provided that the same precautions are invariably taken to minimize skin resistance, and pads of the same size always used, we have a fairly accurate means of testing muscle reactions. The condenser used was the modification of the Lewis Jones set suggested by Purves Stewart. The condensers are charged from a direct current, and, by means of a rheostat, can be fixed at 100 volts. The scale consists of twelve stops varying in capacity from 0.016 to 4.0 microfarads; for convenience, a muscle is spoken of as reacting on, for example, No. 6 stop, meaning a contraction occurs with a capacity of 0.10 microfarads at 100 volts. Before testing, the limb is immersed in warm water for five minutes, and two wooden-handled electrodes, fitted with circular pads 1 in. in diameter, are used in the test, one being placed on the motor point and the other elsewhere on the muscle. Working from above downwards, the various capacities are tried until the lowest stop on which the muscle shows any appreciable reaction is reached. Thus, when it is stated that a certain muscle reacts on No. 8 stop, we mean that the muscle will not react on any stop lower than No. 8.

As all normal muscles do not react on No. 1 stop (0.016 mf. at 100 volts), extensive observations were necessary to determine the normal standards; these are detailed. In general, it may be said that the larger and more superficially situated muscles of the arm react on No. 1 stop,
the deeper muscles of the forearm react on No. 2, while the intrinsic muscles of the hand react on No. 3. No definite conclusions can be drawn from condenser tests made within one month of injury.

With regard to injuries to mixed nerves, although motor paralysis and complete sensory loss in the area of cutaneous supply may be found at an examination made more than a month after injury, the nerve may still recover its function apart from operation. It is in these cases that condenser tests are of great value. If, in lesions of the musculospiral, median, anterior crural, sciatic, external or internal popliteal nerves, the majority of the muscles react on No. 7 stop (0.25 mf.) or below, an incomplete lesion may be diagnosed and recovery expected without operation. The same may be said of an ulnar-nerve lesion in which the intrinsic muscles of the hand react on or below No. 8 stop.

If no response is obtained below No. 8 in muscles normally reacting on No. 1 or 2 stops, the case should be treated on the usual lines and re-examined after an interval of six weeks. If no improvement is then found in the condenser reactions, operation should be advised. At operation the nerve is usually seen to be compressed by scar tissue or partially divided. If actual retrogression is found after the six weeks' interval, exploration should be advised without hesitation. If the muscles supplied by the injured nerve fail to react on any stop below No. 10, the majority reacting on Nos. 10 to 12, the nerve is suffering from severe compression, partial anatomical division, or, in some cases, complete division, and is most unlikely to recover apart from operation.

The author has met with comparatively few cases, examined at a period exceeding two months from the date of injury, in which subsequent operation has revealed complete division of the nerve, to show a response on any stop below No. 12 in any muscle supplied by the injured nerve. In the case of lower-limb muscles, no response can usually be obtained even on No. 12 when the nerve is completely divided. When all muscles supplied by the injured nerve fail to react on the highest stop, therefore, complete division, either physiological or anatomical, is usually present. If not anatomically divided, the nerve is found to be so intimately involved in scar tissue as to render resection and end-to-end suture necessary.

As in severe injuries involving the lower-limb nerves the muscles often fail to react even on the highest capacity (No. 12) at 100 volts, it has been suggested that it would be desirable to use a higher voltage than 100 for testing leg muscles. Although indicated for the more accurate estimation of reactions, for practical purposes the author's experience has been that if the muscles fail to react on No. 12 at 100 volts, operation will certainly be required. Whenever he has seen such a case, operation has invariably been advised, and in all cases revealed a severe injury which apparently could only recover as the result of surgical intervention.

A practical difficulty sometimes arises when muscles are much atrophied; on No. 8 or No. 9 stop being reached, the condenser discharge produces marked contraction in muscles in the neighbourhood of those in which stimulation is being attempted. Although unsatisfactory from the point of view of exact testing, the practical disadvantage of the occurrence
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is not as great as might appear, for it may be safely assumed that the severity of the nerve lesion is such as to require operation. In all cases met with, the operation advised was never found to be unnecessary. With regard to injuries to mixed nerves with only partial cutaneous sensory loss, the muscles innervated by the affected nerve seldom fail to react on or below No. 10 stop. If reacting on No. 8 or below, recovery almost invariably ensues without operation. Cases in which the majority of muscles react on No. 10 should be examined every four weeks; if no improvement be apparent after the third examination, exploration should be advised. Some cases appear to remain stationary; in such instances operation usually reveals moderate involvement in scar tissue or a partial division of the nerve, and surgical treatment is followed by rapid improvement.

As regeneration proceeds, there is usually a progressive diminution in the capacity of the condensers required to evoke a contraction in the muscles supplied by the damaged nerve. When the condenser reactions improve progressively month by month, it may be safely assumed that recovery is taking place in spite of the continued absence of voluntary movement. When the reactions approach No. 8 stop from above, voluntary movement may be expected shortly to appear. If, after operation, no change in the condenser reactions takes place month after month, it is probable that the operation has been unsuccessful and that no recovery will occur.

It is not suggested that one should rely solely on quantitative condenser reactions; other important factors often have to be considered, such as the condition of the paralyzed muscles, the absence or otherwise of systematic treatment, sensory changes, etc. The purpose of the communication is to illustrate the value of condenser tests in assisting one to arrive at an opinion as to the extent of a nerve lesion, the nature of the treatment to be recommended, and the ultimate prognosis.

Author's Abstract.

TREATMENT.


The writer is here concerned with that form of vertigo which results from vasomotor or toxic (sympathetic or endocrine) disturbance of the vestibular apparatus, and for which, on examination, no organic cause can be found. Such vertigo is fundamentally a labyrinthine phenomenon, and its cause is irritative in character. The feeling of giddiness is usually as fleeting as the cause which produces it. Vasomotor stasis of the vestibular branch of the internal auditory artery, leading to a congestion of the semicircular canals, produces the pure form of vertigo, i.e., a sensation of giddiness which is unaccompanied by deafness or tinnitus. These latter do occur concurrently, however, if the vasomotor disturbance involves in addition the other (cochlear) branch of the internal auditory artery.