SENSORIMOTOR NEUROLOGY.


The author remarks that insufficient trial has been given to arsenical medication, intravenous and intrathecal, in the early stages of G. P. I., most of the reports hitherto published having emanated from asylums. In order to test the efficacy of such treatment before extensive destruction of the brain substance has occurred, it is necessary to diagnose the disease in what he calls the pre-clinical stage.

He records the case of an acquaintance of his own, a man of academic distinction and considerable intellectual powers, in whom he noticed a slight inequality of the pupils. A history was obtained of syphilitic infection twenty-two years previously. The subject reported himself as being in normal health, but examination revealed fixed and unequal pupils. No other abnormalities were detected either in the mental or physical status. Laboratory tests showed that the Wassermann reaction was weakly positive in the blood, positive in the cerebrospinal fluid. The cerebrospinal fluid also contained 22 cells per cubic millimetre, and gave positive globulin tests, with a definitely paretic colloidal-gold curve.

This case (which the author claims is unique in modern literature) shows that even years before the earliest mental symptoms, examination of the cerebrospinal fluid may reveal signs characteristic of G. P. I.

In the early stages of tabes, on the other hand, the examination of the cerebrospinal fluid is not so constantly of diagnostic value. A case is quoted in which the clinical signs of the disease were present with negative Wassermann reactions in blood and fluid: the latter, however, showed 'a lymphocytosis', without increase of globulin. Finally, two cases are described as 'tabes in the initial stage,' with negative results from examination of blood and cerebrospinal fluid. The clinical evidence in support of the diagnosis fails to convince the reader of its accuracy in these two cases. The author's southern temperament enables him to write easily of a 'rapid and complete cure' of tabes if diagnosed in the early stages.

C. P. SYMONDS.


This is an excellent statistical study of 1000 cases of general paralysis, with special reference to some of the many factors which have been thought to have an influence on the course of the disease. By avoiding the use of figures it is possible to give a concise account of the author's main conclusions.

Age.—The age of the patient at the time of infection has no influence on the form of syphilis which may develop in later life. The older the patient at infection, the shorter the latent period. This shortening is relatively greater than the diminished prospect of life in the higher age
groups. The age at infection or onset has no influence on the duration of the disease. The expansive form is commonest when infection occurs before 30, the melancholic form is commonest in late infection, the incidence of the simple demented form is equal for all ages. The latent period varies considerably within each age group. This suggests that some of the following factors may be active:—

**Sex.**—The latent period is slightly shorter in women than in men both for tabes and paresis, and both run a slower course in women.

**Alcohol.**—In alcoholics the latent period is longer than in abstainers, and the duration is considerably increased. (Average duration in alcoholics 30 months, in abstainers 10 months.)

**Treatment.**—The cases of uncomplicated general paralysis in which accurate information could be gained on this important point numbered 398. The latent period in those who had no treatment was 14·6 years; inadequate treatment, 18·8 years; good treatment, 12·9 years. From this it would seem that treatment hastens the onset of paresis. But on analysing the age groups it is clear that this conclusion is fallacious. Most of the cases of inadequate or no treatment are found in the younger groups, where the latent period, as we have seen above, is long, whilst those who acquire syphilis later in life undergo more thorough treatment. Hence it comes about that if statistics are compiled without reference to the age of the patients at the time of infection, it appears as if treatment shortens the latent period. In reality early treatment has no effect thereon. In 20 cases where salvarsan had been used in the early treatment the latent period was unusually short, but no conclusion should be drawn from this, because the cases with longer latency have not yet had time to appear. The duration of the disease is shorter in those who have had good early treatment. Once the disease is established, treatment by fever therapy and by antisyphilitic remedies prolongs the disease, and remissions are more frequent than in untreated cases.

**Occupation.**—This has no influence on the latent period. The duration is slightly shorter in brain workers than in manual workers.

**Fatigue and exposure.**—In soldiers who served in the great war the latent period was shortened on an average by two and a half years. It might be said that the diagnosis would be made earlier in the field than under peace conditions; but this would not account for such a big difference, a period which equals the total duration of the disease in many cases. A more important consideration is that the figures must necessarily refer to cases with a short latent period, for those with a longer incubation have not yet developed the disease. At present it is not possible to state with certainty that the onset of general paralysis was hastened by military service. The duration is the same in soldiers as in others.

**Trauma.**—This has no influence on latency or duration.

Other factors which from these figures seem to have no effect on the course of the disease are: habitus, anomalies of internal secretion, the presence of stigmata of degeneration, and intercurrent diseases.

W. J. Adie.
NEUROLOGY


This interesting paper describes thirty-two cases of conjugal or cohabitation syphilis of the nervous system, observed during a period of eight years. They were derived from twelve sources. In one instance a man, his two mistresses, and his innocent sister all developed neurological signs of syphilis. It is interesting to note that in some cases a different form of nervous disease appeared in each of two or three patients of a group infected from the same source or from one another, one developing tabes, another paresis, and a third syphilitic meningitis. So many cases of conjugal tabes and conjugal paresis have been reported that we are glad of a corrective to the idea that this is the rule rather than the exception. In fact, such cases, by their rarity, seem to be evidence against, rather than for, the existence of a neurotropic strain of the Spirocheta pallida.

J. G. GREENFIELD.

[121] Studies in heredo-ataxia.—EMANUEL BERGMAN. Upsala Läk. Forhandl., 1921, xxvi, Häft 5–6, contrib. no. 3.

In this clinical paper two families of Friedreich’s disease and one of so-called hereditary cerebellar ataxia are described. In the first there are six cases of Friedreich’s disease among fifteen brothers and sisters; the second has two branches, with five cases among ten brothers and sisters in the one, and one out of six in the other. The third family is one with four cases of hereditary cerebellar ataxia in four generations.

The author is compelled to admit the difficulty of recognizing the latter disease as a well-marked clinical and pathological entity, and is inclined to look on both as different expressions of one and the same condition, viz., ‘hereditary ataxia’. He is unable to help us by definite contribution to the problems of etiology connected with these two affections, but concludes tentatively that in hereditary cerebellar ataxia inheritance is dominant, and in Friedreich’s disease recessive.

In the former family the disease began at four years of age in the representative of the youngest generation, in her father at forty, and in her grandfather at fifty-six. The symptoms were gross ataxia, little spasticity, occasional tremor, dysarthria, pallor of discs, no nystagmus, brisk knee-jerks, Achilles-jerk absent on one side and reduced on the other, double extensor response. Bergman observes with truth that the only way to fight these diseases actively is by trying to prevent them, and recommends more intensive study of the variations of the diseases in different families, with a consciousness of the end in view; perhaps the time will come “when by means of eugenics we can seek to prevent as far as possible the origin and inheritance of these diseases”.

S. A. K. W.

[122] Family spastic paraplegia (La paralégie spasmodique familiale).—P. VAN GEHUCHTEN. Revue neurol., 1920, xxxvi, 901.

The writer opens with a brief review of the literature and the attempts made at classification of the various combinations of spastic paraplegia of
familial type with other symptoms. He concludes that we may consider as a separate type a group of cases in which the symptoms are confined to those of a paraplegia usually limited to the lower limbs, in which pathological changes are found in the spinal cord only and are chiefly manifest in the pyramidal tracts. He proceeds to describe in detail a familial group of these cases.

The case first investigated was that of a soldier, age 35, with a five years' history of slowly-developing weakness and spasticity of the lower limbs, without sensory changes or sphincter involvement. Clinical examination revealed the signs of a bilateral lesion of the pyramidal tracts. It was subsequently discovered that, of the patient's four brothers and sisters, three presented signs and symptoms of a similar character, these in each case having appeared at about the age of 30. The mother of these patients, 64 years of age at the time of examination, proved to be completely paralysed, the symptoms in her case having begun at the age of 35. Of her three brothers and sisters, two had been affected presumably with the same complaint, and one of the sisters had a son also attacked by the same malady at the age of 30.

The author concludes with some remarks upon the diagnosis of the disease, the pathological findings reported in cases which have been verified post mortem, and the obscure question of etiology. References are given to papers by Lonain (Paris, 1898) and Rhein (New York, 1916), which are said to contain full bibliographies.

C. P. Symonds.


A clinical description of two cases (brothers) in a family in which apparently some nine male members were affected in five generations. The disease began in each without extraneous factor, at the same age, and with the same symptoms; it followed an identical chronic progressive course, and had appeared in apparently identical form in the elder generations also. The paper is accompanied by a very useful précis of some seventy-nine cases from the literature since 1890, derived from a review of nineteen original communications, but it omits all reference to valuable papers by Ernest Jones and other English writers.

S. A. K. W.


A woman, age 62, in excellent health, suddenly lost the use of the right arm and leg, with unimpaired consciousness and no speech defect. Power returned gradually, and in six months she was performing her household duties. Three months later, again quite suddenly, she lost power in both legs, and in a few weeks the arms were also paralysed.

On admission.—Complete quadriplegia, hypertonia extreme in legs, slight in arms, muscles of shoulder girdle and intrinsic hand muscles wasted,
all deep reflexes increased equally, abdominals absent, plantars extensor, no fibrillation, sphincters intact, no subjective or objective sensory troubles, Wassermann negative in blood and fluid, Pandy + + + + , cells not increased.

**Clinical diagnosis.**—Probably atypical amyotrophic lateral sclerosis.

**Post mortem.**—Endotheloma size of a cherry on inner surface of dura 6 cm. below medulla on anterior aspect of cord, cord quite flat, no other lesion in nervous system.

**Special points.**—Complete absence of pain throughout course of disease, sudden onset, and early remission.

W. J. Adie.


The authors recount the structures likely to suffer from compression in cases of distention of the third ventricle, and among the resulting symptoms draw particular attention to those which are associated with lesions of the tuber cinereum and infundibulum. In a paper by Claude and Lhermitte in 1917, a case was described in which a tumour strictly limited to the cavity of the third ventricle gave rise to what the authors named the ‘infundibular syndrome’. This consisted of attacks of narcolepsy, cardiac irregularities, polyuria, and polydipsia. The hypophysis in this case was normal, and its possible rôle in the production of these symptoms was therefore excluded.

In the case here described the hypophysis showed marked changes, while the infundibular syndrome was absent. The patient, a man, age 29, came under observation in May, 1920, and died six days later following a right-sided operation for decompression. The symptoms, which were of about one year’s duration, consisted in the first place of weariness and lassitude, together with changes in the temperament. To these were shortly added complete sexual impotence. In the course of five months the patient put on ten kilo. in weight. For five months he had suffered from severe headaches, accompanied by vomiting, and transient mistiness of vision. On examination, he was drowsy and suffering from severe headache. Slight nystagmus on lateral deviation, and bilateral papilledema, were the only other signs of nervous disease. In spite of the gain in weight already mentioned, there was no marked adiposity, nor were the genital organs atrophied. Lumbar puncture was twice performed. Following the second occasion the patient became comatose, with unequal fixed pupils, and some rigidity of the left limbs. Cheyne-Stokes’ respiration developed, and the patient died twenty-four hours after a right-sided decompression operation.

At the post-mortem a closed cyst the size of a pigeon’s egg was found within the distended third ventricle. The lateral ventricles and foramina of Monro were much dilated, while the aqueduct of Sylvius was reduced.
to a linear slit. On either side the walls of the cyst were attached to those of the third ventricle, whilst at its anterior end it was prolonged in the form of a diverticulum which ended in a solid nodule of growth occupying the pituitary fossa. The pituitary body was much damaged, the posterior lobe having been practically obliterated by pressure, while the structure of the anterior lobe, though still recognizable, contained few normal cells. Microscopical examination showed the growth to be of an epithelial nature, consisting of columnar cells in the periphery of the nodule, with colloid degeneration in the centre. The walls of the cyst consisted of a layer of delicate connective tissue, containing in its meshes cells similar to those already described. At the points of the attachment on either side to the walls of the ventricle, the surface of the optic thalamus was covered with a layer of epithelial cells of similar type, beneath which there was proliferation of glial elements, while the superficially situated nerve-cells showed degenerative changes.

Further examination of the brain and spinal cord showed no changes which the authors considered to be of significance. They comment upon certain points of peculiar interest in the case:

On lumbar puncture the cerebrospinal fluid appeared to be under no excessive pressure. This was due to the blocking by compression of the aqueduct of Sylvius. The dilatation of the right pupil and left hemiplegic rigidity are put down to compression of the right side of the mid-brain.

The early symptoms were those of disturbed pituitary function only. This is in accordance with the fact that the grey matter around the third ventricle was compressed but not invaded by the neoplastic formation.

The order of development of the symptoms and the mode of their production is best explained by the assumption that the tumour developed in the first instance from a remnant of the pharyngeohypophyseal diverticulum. They remain somewhat uncertain, however, as to this point, admitting the possibility of its origin in the ependymal cells of the ventricular wall.

C. P. Symonds.


This paper is a review of observations of epilepsy resulting from cranial wounds during the war. The author may perhaps antagonize most English neurologists, who follow the teaching of Hughlings Jackson, by talking about scars resulting in constant irritation of the brain and so producing fits.

In the first part he classifies cranial wounds into perforating and non-perforating, and except for noting the fact that while perforating wounds are more likely to cause fits than non-perforating ones, and that all cranial wounds may be followed by epilepsy, he comes to no definite conclusions. He distinguishes between the transitory fits due to commotion and infection and those which persist for long periods. He gives certain statistical results of inquiry into etiology which are quite inconclusive.
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In the second part he considers the causes of the period of latency between the wound and the first fit. These, he considers, depend on the duration of the process of cicatrization of the cerebral wound and on the site of the wound; but as a general rule, if no fits have occurred eighteen months after a wound, none are likely to occur.

In the third part he describes with Gallic thoroughness the varieties of prodromata, auras, crises, and post-paroxysmal manifestations. He points out with justice the extreme difficulties under which an epileptic labours in the labour market, and lays stress on the difficulties of just assessment for pensions purposes.

In the fourth part he discusses treatment. Under medical measures are discussed bromides, which he thinks should be given in the form of the purified sodium salt; borates, which are of doubtful value and seem to be more useful in commotional cases than in others; and urea derivatives such as veronal, luminal, and dial. Combinations of these may also be given, but on the whole the author favours luminal. In dealing with surgical procedure, he advocates this when there are signs of an obvious foreign body or collection of pus or blood. He very properly reminds his hearers that epilepsy is caused by scarring of the cortex, and therefore any procedure which destroys more of the cortical tissue is harmful. He lays it down that exploratory surgery of the cranium is absolutely unjustifiable, and opening the cranium, and still more the opening of the meninges, should only be done when there is a very definite indication for doing so. It is a pity that all surgeons do not take this to heart; it is evident that those who still advocate surgical interference in epilepsy except in very special cases do not have the courage or wisdom to follow up their cases after operation for two or three years, or they would soon change their opinions.

It is very striking that, from beginning to end of a paper on fits resulting from the war, no mention is made of hysterical epilepsy. Anyone who has had much experience of these cases, and has applied the therapeutie test, knows how many of these cases are wholly or partially functional and amenable to treatment by some form of psychotherapy.

R. G. GORDON.


By transient hemiplegia the author means those cases which are due to a temporary deficiency in the blood-supply of certain areas of the brain that is of insufficient duration to cause permanent damage. He points out that the theory of local arteriospasm often advanced to explain such attacks does not and cannot rest on any proofs, and suggests that physical changes in the blood are just as likely to be responsible as local changes in the arteries. The viscosity of a fluid is one of the factors influencing the rate of its flow through a tube. The viscosity of the blood alters with increase of concentration of cells, colloids, or CO₂, but in health this is not sufficient to produce symptoms. Extreme dehydration of the tissues resulting from
excessive sweating, diarrhoea, etc., will raise the viscosity 25 to 30 per cent, and in conditions of actual or relative hypotension or general vascular disease this may slow down the cerebral circulation sufficiently to produce symptoms.

He concludes that in the care of patients of advanced years or those known to have vascular disease, and in those who have had cerebral vascular crises, dehydration of the tissues or too great restriction of intake of fluids should be avoided. The introduction of anticoagulant salts and of fluid may be indicated in certain cases of transient hemiplegia or aphasia, in order to prevent actual thrombosis in vessels when slowing of the circulation is the cause of the symptoms.

R. G. Gordon.


The author gives an interesting account of changes in the pressure of the cerebrospinal fluid at different stages in four cases of this disease. A considerable rise in pressure from sixteen to twenty-two weeks after the disappearance of acute symptoms coincided with an increase in the severity of the residual symptoms. Once the rise occurred, the pressures ranged between 200 and 400 mm.—averaging about 300 mm. as long as the cases remained under observation. Hartmann concludes that the late symptoms—headache, torpor, general weakness, slow cerebration, pains in the limbs, restlessness, and so on—are caused in part at least by meningitis serosa, and suggests that as repeated punctures produce no permanent lowering of the pressures, some method of permanent ventricular drainage might be tried.

W. J. Adie.


The author pleads for a return to the consideration of subjective symptoms. He admits that the patient’s interpretation of these is quite valueless, but points out that the medical profession have not been guiltless of erroneous interpretation. If subjective symptoms are neglected, the objective findings are apt to lose all proportion one to another, so that fads and prejudices arise. The diagnosis of the psychoneuroses must depend entirely on subjective symptoms; but the physician must not neglect objective signs in these cases by being too impressed by the mental subjective symptoms. The author points out the value of the subjective symptom of numbness and other paresthesias as the first sign of sclerotic changes in the central nervous system in such conditions as pernicious anaemia, thalamic lesions, and arteriosclerosis. The reliability of the patient must, of course, be estimated in evaluating subjective symptoms; but it is unnecessary to mention the importance of muscular hyperalgesia in diseases of the viscera, nor must headache and vertigo ever be neglected in diagnosis.
The paper is a wholesome reminder that the patient is a part of the disease, in an age which is too prone to depend exclusively on the laboratory.

R. G. GORDON.

**TREATMENT.**


The author points out the difficulty in deciding whether to operate or not in cases of injury of the cervical spine. He quotes Elsberg, who condemns operation in cases which show complete loss of sensation, motor power, and reflex activity below the lesion, but who advocates it in cases when all activity is not lost at first, but with progressive loss within the first few days after injury, especially if compression of the cord by fractured vertebrae or extravasated blood can be demonstrated. The danger of operation on the cervical cord is affection of the vagus and phrenic nerves by the edema induced, with resultant respiratory and cardiac paralysis. Three out of the author’s four cases recovered without operation, whereas Elsberg’s figures for those not operated upon was 70 per cent of deaths. If the patient does survive, the functional recovery is surprisingly good compared with cases of injury in other parts of the cord. The most frequent residuals are paresis in the extremities, and atrophies in the small muscles of the hand, with stiffness in one or both lower limbs. There is little loss of sensation, and bladder and rectal control is usually re-established. The statistics of surgical interference are not encouraging even in partial lesions, but Elsberg believes that this should improve with greater experience and better technique; the author, on the other hand, thinks that great conservation should be observed, though in the later stages disabling root pains may demand interference. The cases of four patients are described in full; one of them died, but the other three made relatively good recoveries, all without operation.

R. G. GORDON.


Starting from the hypothesis that in every case of neurosyphilis the nervous system is invaded in the secondary stage, the author takes up the position that every patient with secondary syphilis should be regarded as harbouring spirochaetes in his intrathecal tissues. He considers that at this stage it is dangerous to push antisyphilitic treatment too energetically, as, if one is successful in eradicating the spirochaetes from the general body tissues, the blood will be deprived of antibodies and the spirochaetes in the intrathecal tissues will then have free rein to multiply and attack the nervous system. This theory rests on the hypothesis that antibody reaches the intrathecal tissues from the blood-stream, but no facts or experiments are adduced to prove this. As a corollary we get the unexpected statement