

loses efficacy, galvanism has a more marked action, and at a variable time after death remains alone efficacious; mechanical stimuli acquire a relatively greater action than electrical stimuli, and are finally the only stimuli capable of eliciting contraction; the last electro-muscular reaction which the author obtained was 9 hours after decapitation, being a local, sluggish, prolonged contraction to the direct application of the galvanic electrodes, apparently a result of chemical action.

As a sign of death, the author concurs in Roserthal's opinion that electrical exploration is the best, most certain, and most rapid test of that event; the approximate time after death may even be pronounced on the above data. Thus, for instance, if in a limb faradism elicits response from the flexor muscles, no response from the extensor muscles, it may be affirmed that death has happened between 4 and 5 hours previously. If at the same time on the face we more easily elicit contraction by galvanism than by faradism, and if the contraction is sluggish, we are further assured that death has happened about 4 hours previously.

The electrical test fails only where it is necessary to know *rapidly* whether death has taken place, but the only case in which this necessity can arise is sudden death during pregnancy, where there is a possibility of saving a life by the Cæsarian section. In this single instance, other tests need be relied on, namely, the contrast between the appearance before and after sudden death, and the comparison of the maternal and foetal pulse.

In sum, the electrical test affords the best sign of death, may inform us of the period of its occurrence, and is a powerful means of reawakening the circulatory and respiratory functions.

A. WALLER.

Strümpell on the Pathology of the Spinal Cord.—In this paper (*Arch. f. Psych.* x. p. 676, and xi. p. 27), the author discusses the subjects of spastic spinal paralysis and combined systematic disease.

Spastic Spinal Paralysis.—The first case reported is that of a man, aged 25, who had contracted syphilis four years previously. Death happened within nine months of the appearance of the first symptoms. During the greater part of the time the symptoms were those characteristic of spastic spinal paralysis: there was gradually advancing paresis and paralysis of the inferior extremities, excess of the tendon reflexes, rigidity of the muscles, the usual spastic phenomena, and an absence of all sensory symptoms. It was not

however a typical case of the disease, for at an early period transient irritability of the bladder was noticed, and two or three weeks before death paralysis of the bladder and bed sore appeared, sensibility became affected, the muscles of the limbs atrophied, and the tonic contraction of the extensors was succeeded by contraction of the flexors.

The post-mortem revealed the presence of a diffuse chronic myelitis of the upper dorsal part of the cord. Westphal, it will be remembered, has shown that this part of the cord is very frequently affected in chronic myelitis. Though the disease was diffuse, it was observed that certain parts were more prone to be attacked than others, and that a striking symmetry obtained between the lesions on the two sides. Thus in the posterior columns, the columns of Goll were always diseased, and if the lesion spread to the adjoining columns of Burdach it was always the posterior parts that were most affected. In the lateral columns the posterior regions were more affected than the anterior, and the lateral limiting layers were almost intact. The anterior columns showed few signs of disease beyond a thin zone of marginal degeneration on each side. The changes in the grey substance were most marked in the anterior cornua; the most noteworthy appearances being the paucity of the ganglion-cells, the number of large spindle cells, the presence of cells with fatty granules, and, most important of all, the widely dilated vessels which gave quite a cavernous look to the tissues. In other places the adventitia of the vessels was much thickened. The myelitis had a further peculiarity; it did not consist of one large focus, but had a floccular or disseminated character, and presented a strong resemblance to the disseminated cerebro-spinal sclerosis. Indeed cases have been described in which the distinction was so puzzling that the diagnosis of chronic myelitis or disseminated sclerosis rested on the presence or absence of secondary degeneration.

In the latter disease secondary degeneration is not met with above and below the focus of myelitis; tracts of ascending and descending degeneration were respectively observed. There was ascending degeneration of Goll's columns and of the direct cerebellar tracts. The degenerated cerebellar tracts were traced upwards to the medulla. They lay nearer the olivary body than Flechsig figures them. There was descending degeneration of the lateral pyramidal tracts, of the direct cerebellar tracts, and for a short distance of a thin band of fibres on the outer side of each Goll's tract (*bandelettes externes*). The degeneration of the cerebellar tracts was found

as low as the upper part of the lumbar region. Clarke's columns were, throughout their length, considerably poorer in cells than usual. The connection between these cells and the cerebellar tracts seems much closer than that between the cells of the anterior cornua and the pyramidal tracts, for while disease of Clarke's cells very frequently accompanies degeneration of the cerebellar tracts, we often have disease of the pyramidal tracts without alteration of the cornual cells. How shall we explain this descending degeneration of the cerebellar tracts? Three hypotheses seem open to us. The morbid process may have spread from the pyramidal strands to the contiguous cerebellar tracts, or the case may have been one of systematic disease of the cerebellar tracts complicated with dorsal myelitis, or lastly, it may be that focal lesions of the cord in certain circumstances are associated with descending as well as ascending degeneration of the cerebellar tracts. The last view is adopted by Strümpell.

The slight implication of sensibility is remarkable, considering the extent of the myelitis. As the lateral limiting layers were intact, the author suggests that perhaps they are the conductors of the sensory impressions.

The next case was, as regards its symptomatology, a typical case of spastic spinal disease. The most remarkable abnormality was the excessive dilatation of the central canal of the cord. The dilatation commenced between the origin of the first and second cervical nerves and extended throughout the cord. It was greatest in the dorsal and upper cervical regions. In some places the cavity measured 8 mm. from side to side. In the upper part of the dorsal cord the central canal appeared double on transverse section, owing to a small diverticulum from the canal at a lower level. The dilatation was not the result of softening or shrinking in the surrounding tissue, but was a primary change due to abnormality of development.

Besides the hydromyelus there was degeneration of the pyramidal strands of the lateral columns. The appearances were exactly similar to those found in secondary descending degeneration, but as there was no evidence of direct interference with the tracts, and as the lateral limiting layers, which pressure radiating from the central canal would naturally affect most, were almost intact, as too the degeneration was not confined to the pyramidal tracts, but implicated also the cerebellar strands, Strümpell inclines to think that the case was one of hydromyelus accompanied by primary systematic disease of the pyramidal and cerebellar

tracts. A narrow belt of degeneration, probably the result of an extension of the disease from the central canal, surrounded the grey matter in the anterior and lateral columns. In the posterior columns there was no sign of systematic disease; on the contrary, the affection had an exquisite disseminated character.

Taking a general view of the case, the author remarks that there was a hereditary predisposition to nervous disease, which showed itself in the earlier stages of development in the anomalous condition of the central canal. Probably the faulty laying down of the strands of the cord, some systems being more affected than others, predisposed to the early appearance of the systematic affection.

Spastic spinal paralysis occurs in widely different diseases. We have considered at length two conditions in which it was seen, and to these Strümpell adds, giving cases, injuries of the spine (myelitis from compression), tumours of the cord, multiple cerebro-spinal sclerosis, chronic hydrocephalus, and those affections of the nervous system which are sometimes found as the sequelæ of acute illnesses, and especially of typhoid fever. It is impossible as yet to specify the anatomical lesion that underlies the disease, though, as Erb stated, primary systematic degeneration of the lateral columns often induces it.

In pursuing this investigation we must be careful to distinguish between the paralysis and the spastic phenomena. We may have the latter without a trace of the former. We must also remember the complexity of the group of spastic symptoms. This includes, besides the phenomena dependent on excess of the tendon reflexes, various kinds of muscular contraction and contracture, e. g., the contracture due to direct motor irritation (flexion of the leg with convulsive dorsal flexion of the foot, *Beugecontractur*), and the contracture due to reflex influences (extension of the leg with strong plantar flexion of the foot, *Streckcontractur*); and to these must be added localised convulsive movements that are produced by a variety of causes.

Combined Systematic Diseases of the Cord.—The first case presented the group of symptoms found in amyotrophic lateral sclerosis: paresis of the extremities, trophic changes, and spastic symptoms. There was simple atrophy of the muscles of the forearm and hand, but in the triceps and the muscles of the lower extremities, there was, in addition, a striking degree of fatty infiltration of the muscles: the reaction of degeneration was not observed. The most constant of the spastic phenomena was the

strong dorsal flexion of the foot and toes, with which was often combined flexure of the thigh and leg, making up what the Germans call a *Beugecontractur*. The tendon reflexes were excessive. Convulsive flexing movements were observed in the legs from time to time, and generally affected them alternately. The passive contractures observed in paralysed limbs must be distinguished from the spastic contractures just mentioned. The former are permanent, and the deformity of the limbs gradually grows worse. The latter vary in intensity at different times, partly owing to the varying intensity of the central irritation, partly to the varying irritability of the nerves and muscles. In the former there is club-foot; in the latter, dorsal flexion of the foot. Strümpell thinks that the spastic contracture is due to primary disease of the pyramidal lateral columns, the fibres being still in connection with their trophic centres, and hence retaining their irritability. Applying the same reasoning, secondary degeneration of the lateral columns cannot account for the contracture of hemiplegia, for the fibres are separated from their trophic centres, and speedily lose their irritability.

A peculiar feature of the case was the association of movements in the lower limbs. There was not only an abnormal irradiation of the voluntary impulse to other muscles on the same side, but the patient could never flex his thigh either voluntarily or as the result of a reflex excitation, without inducing very intense dorsal flexion of both feet. There was complete incontinence of urine, and ultimately purulent cysto-pyelitis. Sensibility to cutaneous stimuli was perfect. A feeling of cold and formication was experienced in the lower limbs, but never to any great degree. Towards the end, pains of some severity were felt, but they were quite different from lightning pains.

The case was diagnosed as amyotrophic lateral sclerosis, though there were evidently important points of difference between it and Charcot's cases of this disease. For example, in Charcot's cases the disease generally commenced in the upper extremities, and travelled downwards; in this case an exactly opposite course was pursued. Further, in Charcot's cases there was usually atrophy, without fatty infiltration, of all the muscles of both extremities, and there was no paralysis of the bladder. Nevertheless, there seemed good ground to expect systematic disease of the pyramidal strands, and lesion of the anterior cornua.

The lesions found on *post-mortem* examination were (1) complete degeneration of the lateral pyramidal tracts of both sides in their

whole length, and of the anterior pyramidal tract of the right side down to the lower dorsal region; (2) degeneration less intense, of the cerebellar tracts; and (3) degeneration of the posterior columns. The anterior cornua were quite healthy. The cells of Clarke's columns were abnormally few in number in the lower dorsal region. The disease nowhere extended into the medulla oblongata.

The distribution of the disease in the posterior columns was remarkable. The condition of each column was as follows: (1) a narrow belt of normal tissue bordering the posterior fissure; (2) external to this, the degenerated Goll's column, triangular in shape; (3) external to this again, the degenerated 'postero-external region of the posterior column,' also triangular in shape; (4) in the anterior part of the column, and with its apex directed back between the apices of (2) and (3), a third triangular area, of normal tissue; (5) in the mesial line immediately behind the posterior commissure, and at the apex of Goll's column, a small band of degeneration; and lastly (6) a narrow intact strip on the inner side of the posterior roots. The symmetry of the disease on the two sides, and the occurrence of the same sharply defined areas in widely separated parts of the cord, are strong arguments in favour of the systematicness of the affection. (1), (2), and (5) together, make up what is usually regarded as Goll's column. (3), (4) and (6), constitute Burdach's column. The somewhat indefinite 'bandelettes' of Charcot probably correspond to (4).

Viewing the lesions in connection with the symptoms, this case seems to teach us (a) that sensory conduction does not take place either in the pyramidal strands, the cerebellar tracts, Goll's columns, or in the postero-external divisions of the posterior columns; (b) that with very extensive degeneration of the pyramidal tracts we may only have symptoms of paresis; and (c) that the posterior columns may be diseased in the lumbar region, and yet there be excessive tendon reflex.

In a second case, the symptoms were paralysis and contracture of the lower extremities, heightened tendon reflex, incontinence of urine, and a degree of analgesia in the legs. The autopsy gave degeneration of the lateral pyramidal tracts (particularly in the inferior dorsal and lumbar regions), the cerebellar tracts (degeneration most marked here), Goll's columns, and the postero-external divisions of the posterior columns. The anterior cornua were normal. The degenerated Goll's columns were traced to the upper part of the lumbar region.

Allusion is made to a third case, reported by Kahler and Pick,

in which the symptoms bore a strong resemblance to those observed in Friedreich's cases of hereditary ataxia, and where after death there was found systematic degeneration of the pyramidal strands, the cerebellar tracts, and Goll's columns. These three cases, Strümpell thinks, leave no doubt as to the existence of primary combined systematic diseases. The morbid process in these diseases is not a chronic inflammation originating in the connective tissue or blood-vessels, but is a true parenchymatous degeneration of the nerve fibres, the increase of connective tissue being secondary.

The paper closes with a case of tabes dorsalis, in which there was paralysis of the legs and ataxia and paresis of the upper extremities. The autopsy showed complete degeneration of the posterior columns with the exception of a small area adjoining the posterior commissure, and also systematic degeneration of the cerebellar tracts in their whole extent and of the lateral pyramidal strands in the lumbar and lower dorsal regions. A narrow belt of marginal degeneration surrounded the cord. The affection of the lateral columns in this case was too systematic to be accounted for on the usual theory of the direct extension of the disease from the posterior columns. The only feasible explanation is that tabes dorsalis belongs to the group of primary combined systematic diseases.

W. J. DODDS, D.Sc.

Syphilis and Tabes Dorsalis.—Westphal (*Archiv f. Psych.* xi. p. 230) concludes that an aetiological relation between syphilis and tabes is unsupported either by clinical or pathologico-anatomical facts. In 75 cases, whose histories were comparatively well ascertained, he found chancres in 14, chancres and secondary symptoms in 11. Nineteen out of 20 cases occurring in women had no history of chancre, and the twentieth case was a doubtful one. In one case only were secondary symptoms present whilst the patient was under observation. Of 16 cases that came to autopsy one only showed evidences of syphilis, and in another the appearances were doubtful. Further, Westphal has never seen a case of grey degeneration of the posterior columns cured by anti-syphilitic remedies. Remak (see *Centralblatt f. med. Wissensch.* 1880, No. 43) obtained a history of syphilis in 25 per cent. of his cases of tabes, nevertheless he does not admit a direct causal connection between the two. Bernhardt admits that syphilis may