CRURAL MONOPLÉGIA.

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Within a comparatively short period of time six patients attended hospital complaining of weakness of one leg. In five of the cases the diagnosis has been established beyond doubt, and was proved to be different in each of them. The sixth case still presents some uncertainty. A careful examination of the history and course of their illnesses is instructive.

Case 1.

Male, clerk, aged 43 years.

Complained that for 4 years he had noticed a progressively increasing numbness, loss of control and weakness in the right foot.

His previous health had been good. No other nervous symptoms were remembered and venereal disease was denied. The family history was normal.

Three years previously he first noticed that after walking far the right foot "went dead and dragged." This got worse and in 6 months the abnormality became permanent. Gradually the weakness extended up the leg and spontaneous clonus occurred at the ankle but there were no convulsive phenomena. The left leg and both arms were normal. Speech, mentality and sphincter control were unimpaired. There had been no headache. He was right-handed.

Examination (24/5/33). The patient was a healthy-looking man of normal mentality and speech. The cranial nerve functions were all normal. There was slight weakness of the right-hand grip and some tremor of the left-hand in co-ordination tests. The right leg was spastic and weak in all muscle groups, particularly the distal ones. The left leg was normal. The arm reflexes were brisker on the right side than the left; the abdominal reflexes diminished on the right side; the right knee and ankle jerks exaggerated and the right plantar reflex extensor, the left flexor. Sensation was normal. The spine and skull were normal. Investigation of the cerebro-spinal fluid showed a completely normal fluid. The blood Wassermann reaction was negative. X-ray examination of the skull was normal. A diagnosis of disseminated sclerosis was made.

Subsequent Course. In October, 1933, it was noticed that slight dysarthria was present. In April, 1934, a sudden increase of the weakness in the right leg and some weakness of the left leg occurred. On his admission to hospital at this time there was a spastic weakness of both legs with bilateral extensor plantar reflexes. The mood was euphoric and intention tremor was present in both hands.

The weakness of the legs rapidly improved, but relapsed again twice during the next 6 months.

In October, 1934, precipitance of micturition was first experienced.

The progress of the disorder confirmed the diagnosis of **disseminated sclerosis**.

Case 2.

Male, chauffeur, aged 35 years.

Complained of dragging of the right leg for 6 months. His previous health had been excellent except for an attack of lumbago some years before. No previous nervous symptoms and no venereal disease were admitted. The family history was good. Six months previously he noticed that the right leg was becoming stiff and that it fatigued readily and dragged. During the past month similar symptoms had occurred in the left leg and precipitant micturition was experienced. The arms and cranial nerve functions were normal and no pain or paræsthesia had been felt.

Examination. The right foot dragged in walking. His speech and mentality were normal. Both optic discs were paler than normal in their temporal halves, but in other respects the cranial nerve functions were normal.

The arms were of normal size and power and apart from slight terminal tremor co-ordination was normal. The trunk muscles were normal but both legs showed a spastic weakness which was much more severe on the right side. The arm reflexes were abnormally brisk; the abdominal reflexes absent on the right side; the knee and ankle jerks much exaggerated especially on the right side and both plantar reflexes were extensor. There was no sensory disturbance and the spine was normal.

A preliminary diagnosis of disseminated sclerosis was made and the patient admitted to hospital for investigation.

The cerebro-spinal fluid was under normal pressure but contained 38 lymphocytes per c.mm and 0.02 per cent. of protein with slight excess of globulin. The Lange colloidal gold reaction was 2344321000; the Wassermann reaction +++ in the cerebro-spinal fluid and ++ in the blood serum.

The case was thus one of **syphilitic meningomyelitis** and on appropriate treatment has made a very gratifying recovery.

Case 3.

Female, married, aged 62 years.

Complained of gradual loss of the use of the left leg during 4 months. Her past and family histories were both satisfactory. The weakness and dragging of the left leg were noticed after a period of overwork. They steadily increased and were accompanied by a sore, gnawing pain in the left loin which started in the back and came round to the midline. Latterly the leg had felt a little numb below the knee. The right leg and both arms were normal and no other symptoms had been noticed.

Examination. The cranial nerve and mental functions were normal as were the upper extremities. There was weakness of the lower segments of the rectus abdominis and the lower five intercostal muscles on each side were inactive.

The left leg was spastic and weak in all its movements, the right normal.

The arm jerks were normal, the abdominal reflexes lost on the left side, the knee and ankle jerks were exaggerated on the left side, the left plantar reflex extensor and the right flexor.

Cutaneous sensation in the legs was normal but on the trunk all modes of sensation were more acutely appreciated above a point midway between the xiphoid process and the umbilicus. Sense of position and passive movement were defective in the toes of the left foot.

There was a general dorsal kyphosis and some tenderness to pressure over the upper dorsal spinous processes. A provisional diagnosis of compression of the spinal cord was made and the patient admitted to hospital for investigation. Examination of the cerebro-spinal fluid showed no evidence of any subarachnoid block. The protein content was 0.025 and 0.030 per cent. in two successive examinations and in all other respects the fluid was normal. A skiagram of the spine was normal.

Subsequent Course. After admission the patient's symptoms steadily progressed. The right leg became affected and incontinence developed. The sensory disturbance increased so that all forms were diminished below the 7th dorsal segment, pain and temperature being most severely impaired on the right side and the sense of posture and passive movement on the left side (Brown Séquard syndrome). Finally $\frac{1}{2}$ c.c. of heavy lipiodol was introduced by cisternal puncture and it was seen to be held up on the left side at the level of the arches of the 4th and 5th thoracic vertebræ.

Laminectomy was performed at this level and a small tumour measuring $1 \times \frac{1}{2} \times \frac{1}{2}$ c.m. was removed from the subarachnoid space on the left side of the cord, which proved to be an endothelioma.

Unfortunately, the patient died 48 hours later of an aspiration bronchopneumonia. Had she survived her operation one could have confidently promised a complete recovery.

Case 4.

Male, aged 18 years.

Complained of weakness of the right leg.

His past and family histories were perfectly normal. The weakness was first noticed in the right foot in May, 1933, and gradually extended up the leg. The left leg was normal and absolutely no other symptoms were complained of.

When examined in October, 1933, his mentality, speech, cranial nerve functions and upper extremities and trunk were all normal.

The right leg was slightly spastic and power was reduced in all groups, but particularly in the dorsiflexors of the right ankle which were virtually powerless. The right knee and ankle jerks were exaggerated, the right plantar reflex extensor in type, the left flexor. There was no sensory disturbance. The spine and skull were normal.

A provisional diagnosis of disseminated sclerosis was made and the patient admitted to hospital for investigation.

The cerebro-spinal fluid was under normal pressure and contained no abnormal constituents. The Wassermann reaction was negative in blood and cerebro-spinal fluid. Skiagrams of skull and spine were normal. While in hospital he complained

of diplopia and developed slight weakness of his left external rectus and it was noted that on phonation the palate was drawn slightly to the right and his right arm showed very slight inco-ordination and increase in its tendon reflexes.

In other respects the signs were as before and no headache had been experienced. He was discharged with the diagnosis of disseminated sclerosis.

Subsequent Course. Six months later he was re-admitted to hospital with a 3 weeks' history of headache, vomiting and mental disturbance and increasing paralysis of the right side of his body.

He was lethargic and had an ataxic speech. His fundi showed early papilloedema and there was a complete right-sided homonymous hemianopia. The right pupil was dilated and there was a severe internal strabismus of the left eye. Coarse nystagmus was present on looking upwards and to the right. On lateral deviation of the eyes there was a defect of conjugate deviation of the eyes to the left and the eyes showed skew deviation. There was bilateral retraction of the upper lids. The motor division of the left trigeminus showed great weakness and sensation was diminished over the left half of the face. The left side of the face was completely paralysed. Hearing was diminished and vestibular function lost in the left ear. The left half of the palate and pharynx and the left vocal cord were paralysed. The tongue deviated to the right on protrusion.

The right arm and leg showed a severe spastic paresis without complete loss of power. There was inco-ordination of cerebellar type in both arms.

The tendon reflexes were grossly exaggerated on the right half of the body, the right abdominal reflexes were lost and the right plantar reflex was extensor, the left being flexor.

Sensation to pain and temperature and touch as well as the sense of passive movement were all grossly impaired in the right arm and leg.

It was clear that the original diagnosis had been wrong and that the patient was suffering from a diffusely infiltrating **tumour of the brain stem** from the **medulla to the mid brain** chiefly on the left side.

From this he died some months later.

Case 5.

Male, painter, aged 53 years.

Complained of increasing weakness of the left leg for 2 years.

His past and family histories were both uneventful.

Some 2 years previously the left leg had begun to drag, and after an attack of influenza the weakness was more marked and gradually increased, spreading from the lower part towards the hip.

Six months before attendance very slight weakness was noticed in the left arm, but no other symptoms were elicited.

Examination. The mentality, speech, and cranial nerve functions were normal. The left arm was slightly weaker in all its movements than the right. The left leg was spastic and showed weakness in all its movements, which was slight at the hip, more marked at the knee and very severe at the ankle. The tendon reflexes were increased in the left arm and the knee and ankle joints much exaggerated on the left side. The abdominal reflexes were absent on the left side and the left plantar reflex extensor, the right being flexor. There was no sensory disturbance.

On palpation a thickening of the cranial vault could be felt to the right of the sagittal suture just in front of the coronal suture.

On this finding and the nature of the hemiparesis a diagnosis of **endothelioma of the meninges** arising in the neighbourhood of the superior longitudinal sinus was made.

X-ray examination of the skull showed an unmistakable erosion of the inner table of the skull in this area. The cerebro-spinal fluid was normal in all respects.

Craniotomy was performed and an endothelioma the size of a golf ball found and successfully removed.

The patient is well and at work 2 years later.

Case 6.

Male, carpenter, aged 17 years.

Complained of dragging of left leg for 3 months.

His previous and family histories were uneventful. The condition had come on quite gradually without any pain or other symptom. The other leg and arms were all normal. There had been no sphincter or visual disturbance.

Examination. He was a healthy youth of normal mentality and speech. The outer margin of the left optic disc was slightly pale. The pupils were normal but there was slight internal strabismus of the left eye and indefinite nystagmus on full deviation of the eyes to right and left. The arms and trunk were normal.

Both legs showed spasticity and some weakness, the left being much more severely affected than the right.

The knee and ankle jerks were exaggerated, especially on the left side. The left abdominal reflexes were diminished and both plantar reflexes were extensor. Sensation was normal throughout.

The cerebro-spinal fluid was normal and the blood Wassermann reaction negative.

At present the diagnosis cannot be regarded as finally established, but in view of the patient's age **disseminated sclerosis** is much the most likely.

Discussion.

The outstanding feature of this series of cases is the fact that although the patients all complained of identically the same symptom the cause and prognosis in each case was very different. Two important principles are illustrated. Firstly, that in nervous diseases any individual symptom depends upon the functional unit which is destroyed and not upon the site at which the lesion is situated or its precise pathological nature. To determine these the history of the disease and the other symptoms and signs present must be considered.

In all these cases the symptom depended upon the interference in function of one pyramidal tract somewhere between its origin in the motor cortex and its termination in the lumbar enlargement of the spinal cord.

Secondly, in any such case, a final diagnosis should not be made until every investigation has been carried out, and should be reviewed from time to time in the light of subsequent developments.

In practice the first difficulty in the diagnosis of a crural monoplegia is to determine whether the weakness is part of a paraplegia or of a hemiplegia. To decide this a close examination must be made of the other limbs and of the cranial nerve functions.

If the symptom proves to be part of an incomplete paraplegia the lesion is clearly one of the spinal cord.

The presence of local muscular atrophy, of sensory change below a definite spinal level or of root pain will usually indicate the site of disease.

The occurrence of other symptoms in the past such as transitory amaurosis, diplopia, or numbness in the case of disseminated sclerosis and of other signs, such as abnormal pupils in syphilis, will often indicate the pathological nature of the disease. The commonest causes falling into this category are disseminated sclerosis, syphilitic meningomyelitis and spinal compression from whatever cause.

The examination of the cerebro-spinal fluid often yields invaluable information.

In those cases in which the weakness of the leg is part of an incomplete hemiplegia the lesion is clearly situated in the brain, and the fact that only one limb is involved always suggests that the pyramidal pathway is damaged at a point where its units are spread out over a wide area, *i.e.*, near its origin from the motor cortex. The commonest lesions here are neoplasms, particularly endotheliomata, arising from the meninges in the neighbourhood of the superior longitudinal sinus. More rarely vascular lesions of the anterior cerebral artery may produce a disturbance of similar distribution.

The much rarer condition illustrated by Case 4, of involvement of the pyramidal tract by a tumour of the brain stem, reveals itself by an associated involvement of the cranial nerve nuclei on the side remote from the hemiparesis. In this patient the presence of weakness of the left external rectus and the left half of the palate should have warned one that a local lesion of the brain stem was present but the signs were erroneously attributed to the presence of multiple areas of disseminated sclerosis.