Cerebro-spinal Localization.—Destructive Lesions of Encephalon.—Disease of Cord. By W. Julius Mickle, M.D., M.R.C.P.

Localized destructive lesions of cerebrum, basal ganglia, pons, and cerebellum, and secondary descending disease; also cornual atrophy.

J. S., admitted 1869; died March, 1881, aged 74 years.

Hereditary influences were supposed to have assisted in bringing on mental disease. The patient had naturally been of a somewhat irritable disposition, and was a pushing, active, energetic man of business. Mental failure appears to have come on gradually.

It is on record that when admitted here he was depressed, apathetic, and for some time had been troubled with suicidal inclinations, which he was too irresolute to carry into effect; that though in fair general health, he spoke much of his general debility and incapacity; that he was confused in ideas, of very defective memory, simple and childish in conversation, slow and hesitating in replies, clean in habits; and that he took no part or interest in any occupation or amusement. As for the condition subsequently:—In the early years he was rather better, was fat, in fair health, moderately active, but quite unable to take care of himself, occasionally restless, depressed, and intolerant of musical sounds. In the later years, at least, he was always demented, the principal characteristic being the great failure of memory, so that he scarcely could recollect anything about himself or what had happened to him a few minutes before, or what he wished to tell one, or how he felt. He was, however, clean, good-tempered, and no moral failure was perceptible, only an intellectual debility and mnemonic failure. He was polite, and appeared to be placid, cheerful, and happy.

In 1877 was an attack of vomiting after dinner, apparently with unconsciousness for a few seconds.

In 1878, 1879, and 1880 he gradually became more and more feeble, scarcely able to walk, and was constantly wet.

In March, 1880, some 12 months before death, left hemiplegia came on suddenly, without loss of consciousness, or convulsions, or coma, but with some mental confusion, and thick, muffled, paralytic speech. Subsequently speech was more or less similarly affected, being thick, somewhat muffled, and slightly stammering, though better for months than it had been for a few days immediately after the attack. The pupils were somewhat sluggish, irregular; the right one was slightly the larger. The food was apt to collect in the left cheek. Very soon after the seizure the left lower limb became rigid, straight, immovable, and so stayed until death, the foot being in the mild talipes equino-varus position. The left arm was rigid, sharply flexed; the fingers were tightly flexed into the palm and adducted; the

wrist was flexed and abducted. There was no ankle clonus. In both legs the patellar tendon-reflex was diminished and very slight. In the left leg were diminished sensibility and reaction to pinches; in the left arm these were more obvious, and in the right limbs were rather in excess. The right leg was usually flexed and adducted, so that the sole of the foot rested on the bed to the left side of the rigid, extended left leg. He shouted or groaned aloud on the slightest manipulation of any kind, such as the necessary dressings and cleanings, or on any attempt to straighten the left arm or flex the leg. He was constantly wet and dirty; troublesome, also, by restlessly grasping objects with the right hand. The saliva drivelled from the mouth. He could not recognise persons, though their voices at times seemed in some way familiar to him. He addressed people not present, or when no one was by, repeating the same phrase again and again, and was profuse in his thanks, though somewhat mechanical in their expression. At times it seemed as if he saw indistinctly, or not at all, to his right side; and the head was sometimes turned to the right, the eyes to the left. A gangrenous slough came on the left heel. Finally, dysphagia became marked, so that at last he could not swallow at all, and life was supported by nutritive enemata. Bronchitis, and, latterly, hypostatic congestion and pneumonia of a chronic kind, even as if part of the mode of dying, assisted to cut life short.

Abstract of Necropsy.—Medium height, large frame, traces of obesity. Dura-mater too adherent on lateral aspects of calvaria, somewhat thickened; slight delicate false-membrane tissue between dura-mater and visceral layer of arachnoid, especially in frontal regions. Very marked atheroma of intracranial arteries, the left vertebral artery being extremely atheromatous; the right less affected; the basilar moderately; the superior cerebellar slightly; the posterior cerebral arteries, especially the left, considerably; the middle cerebrals and their branches extremely; and the anterior cerebrals moderately. Almost complete blocking of right Sylvian artery at level of outer edge of insula.

Widespread, but slight, arachnoidal opacity.

Brain flabby, and much wasted; much intracranial serum, and ʒ vi. of it in the lateral ventricles of the brain. Cerebral grey cortex slightly pale generally, and rather wasted in the frontal and parietal regions; it and the white substance flabby. Olfactory bulbs and tracts slightly soft and shrunken. Insulæ free from gross alteration. Left cerebral hemisphere, 16¼ozs.; right ditto, 16ozs.; cerebellum, 4ozs.; pons and medulla oblongata, ½oz.

The following were the more localized lesions:—

1. A large yellow patch of wasting and disappearance of the cortex, with local meningeal induration and pigmentation, affecting

the median surface of the right hemisphere, the middle part of the *right* paracentral lobule, and the adjoining gyrus marginalis for one inch in front of the lobule, the fissura calloso-marginalis forming the middle line of this change, and the subjacent white substance being slightly indurated.

2. Complete disappearance of the cortex and brain-substance on part of the under surface of the *left* occipital lobe and occipito-temporal region, the overlying and thickened meninges bagging here, and forming the wall of a fluid-full cavity continuous with the posterior horn of the left lateral ventricle. The cortical destruction, commencing $\frac{1}{3}$in. from the tip of the occipital lobe, and extending to the sulcus hippocampi, involved nearly the whole of the lobulus lingualis, a little of the lobulus fusiformis, that part of the hippocampal gyrus which is opposite to the posterior border of the crus cerebri and in front of the calcarine fissure, and it extended right through to the posterior horn of the lateral ventricle. This lesion was three inches in antero-posterior length, but its width was narrowed by the falling in and encroachment of the sound tissues on each side as the degenerative and absorbtive changes had advanced. There was gelatinous-like thickening of the ependyma of the posterior ventricular horn.

3. Yellow and drab-coloured superficial spots and patches, with slight superjacent adhesions to the thickened meninges, on the upper surface all along the posterior two-thirds of the peripheral border of the great longitudinal fissure, especially on the occipital lobes. The same, to a slight extent, also one inch from the upper end of the right ascending frontal gyrus.

4. Yellow, drab, or brownish degeneration, softening and destructive, forming a lesion the size of a sixpenny bit, at the middle of the outer border of the caudate nucleus of the *right* corpus striatum, extending to the depth of a third of an inch, and affecting also the internal capsule, the wasting, mingled softness, and stringiness descending into the crus cerebri. Also a minute and very superficial patch of drab-coloured degeneration at the middle of the ventricular aspect of the left corpus striatum.

5. Wasting and discolouration about the middle and posterior parts of the right crus cerebri, entering the pons Varolii.

6. Softening, wasting, and yellowishness of the posterior part of the inner edge of the *left* optic thalamus, extending about a sixth of an inch into its substance.

7. A small cavity in mid-depth of the pons Varolii, towards the upper part, near the median plane, but slightly towards the right side; firm-walled, and containing turbid fluid.

8. Five patches of yellowish wasting and sinking on the under-surface of the left lateral hemisphere (or lobe) of the cerebellum—namely, on the digastric, the slender, and the inferior posterior lobes

of the cerebellum. These patches were similar to those affecting the cerebrum, as above described.

9. Spinal grey matter of small dimensions. Some wasting of the left posterior grey cornu. Slight incipient induration of posterior part of left lateral column.

Under the microscope, the cortex of the right frontal region showed granular degeneration of the nerve-cells, and changes in the walls of the minute blood-vessels.

For the rest, the following only need be stated here :—

Heart, 10ozs. ; mitral and aortic valves somewhat atheromatous ; fibroid patches in columnæ carneæ of left ventricle ; heart-muscle pale, and slightly fibroid in parts ; coronary arteries highly atheromatous, aorta slightly. Bronchitis, hypostatic congestion, and pneumonia ; cicatrix at left lung apex. Kidneys red and cirrhosed ; $3\frac{3}{4}$ozs. and $3\frac{1}{2}$ozs. Liver, $49\frac{3}{4}$ozs. ; spleen, $3\frac{1}{2}$ozs.

Remarks.—1. In this case, with left hemiplegia, there were destructive lesions affecting both the right corpus striatum and the right paracentral lobule, the former being probably the one mainly efficient in producing the palsy, as also the secondary descending lesion.

2. With rigid contractures of the left limbs—the arm being in flexion, and the leg in extension—and with diminished patellar tendon-reflex, and no ankle clonus, the descending sclerosis was only slight, while the corresponding posterior grey cornu was wasted. Possibly the lesion of sensory elements more than neutralized the descending lesion of motor elements in this respect.

3. The destructive lesion of the under surface of the left occipito-temporal cortex, together with the partial lesion of the left optic thalamus (let alone the pontine lesion), were of interest, and somewhat puzzling, in relation to the incomplete anæsthesia of the left leg, and the appearance as of some hyperæsthesia of the right limbs. The state of the cord, however, affords some explanation as to these symptoms, and the pontine lesion may have been contributory ; while, as concerns the impairment of visual perception of objects towards the patient's right side, the same lesions were noteworthy, and, of these, the occipital in relation to results recently published by Munk.

4. The absence of any moral degradation accompanying the extreme intellectual (mnemonic) failure is worth keeping in mind for comparison of the lesions here with those occurring in clinically similar cases.