Cerebral Localisation. Illustrated by a Case of Brain-Injury.

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In the human subject, strictly limited traumatic brain-lesions are among the pathological conditions which bear most clearly upon the problems of cerebral localisation. Necessarily these accidental experiments upon the human brain are sometimes as sharply localised as are the experiments of the physiologist upon the brains of lower animals. For the purposes of the physician, indeed, they are more instructive than the latter.

As one lesion in the following case, portions of the so-called motor-region of the cerebral cortex were partially or completely destroyed. They were those in which Ferrier places the centres for the following movements, mainly :— Movements of opposite leg and foot, as in locomotion; retraction and adduction of opposite arm; prehensile and other movements of hand; supination and flexion of the forearm; retraction and elevation of the opposite angle of the mouth; and, far less obviously, movements which open the eye, raise the lid, dilate the pupil, and turn the head and eyes to the opposite side; as well as certain other movements. The clinical phenomena agree with this fairly, so far as relates to the movements of the opposite lower extremity, and the retraction of the angle of the mouth; but not so far as concerns the movements of the upper limb of the opposite side. Even to nearly the last the biceps acted well, and the hand could readily be raised to the mouth, nor was the grasping power of the right hand relatively affected as much as it should have been according to Ferrier's conclusions.

As to isolated movements of the arm, this case supports the view of Carville and Duret, rather than that of Charcot and Pitres.

A summary as to the relations of the case to cerebral localisation follows its history.

J. H., Pte. 57th Regiment; 4 $\frac{9}{12}$ years' service; age 22; admitted Sep. 25, 1875. This, the first attack of mental disease had commenced insidiously, and therefore the exact date of its appearance could not be assigned, but the patient

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had been under treatment for it in Ceylon from Feb., 1874 to Apr., 1875, and then for several months whilst on the voyage home, and at Netley. The family history was not obtainable, but before entering the army the patient incurred a severe fracture of the skull by a fall from horseback. His character in the army was good, his education moderate. There was no record of syphilis. Since Nov., 1873 he had been known to be subject to nocturnal epileptiform seizures. These seizures became more frequent, as many as five often occurring in the course of the night, and on one occasion he became very violent after a seizure, and assaulted his orderly. The proneness to violence after the fits continued; and mental confusion, incoherence, and dementia appeared and waxed greater. No suicidal tendency was observed. Latterly incomplete right hemiplegia supervened.

His height was 5ft. 4½ in., weight, 131lbs. Without describing the physical condition in detail, on admission, it may be said that a marked cicatrix showed at the left temporal region, and in the left parietal a semilunar, reddish cicatrix, with marked irregularity of the bone. The left ear was misshapen from old hæmatoma. The tongue was protruded without tremor or twitch, and without decided deviation from the median plane. Speech was clear. The equal pupils were not quite normally susceptible to light. Slight lower facial paresis could be detected on the right side. The grasping power of the right hand was impaired, and in walking the right toes dragged, and the right leg was swung round somewhat at each step. Of the two, the lower limb was the one more paralysed. Sensibility was diminished in the right limbs, and their temperature was lower than that of the left, being in the right axilla 96·8°, as compared with 97·5° in the left. No other sensory failure was noted. Pulse 60; respiration 11; heart's action and circulation rather weak.

Dementia was well marked, but in some respects, and probably in relation with the convulsions, the obscuration of mind was more or less fitful and shifting. Thus, at times he appeared to be utterly dull, stupid, and void of comprehension, inattentive, and incapable of expressing himself; at others, each question only elicited one and the same irrelevant reply; at others, he made, to simple questions, replies which were relevant, although often incorrect by reason of amnesia. On his usually vacant and expressionless countenance a silly smile at times was seen. (KI, KBr. Tonics.)

As to the further clinical history of the case, it need only

be added that the epileptiform attacks still usually occurred at night, and therefore escaped my observation. Sudden quasi-syncopal seizures, however, occurred by day. At times the patient made *bizarre* movements; now lifting the head and gazing upwards; now slipping sideways, inertly on his chair; again rising, staggering, stumbling and falling towards the right side; and anon performing a turning movement, around and around, ending by his sinking in a heap to the ground. On occasions at night he was extremely restless, and even noisy, uttering incoherent ejaculations and unintelligible noises. By day he was often drowsy, heavy and languid, and at other times utterly dazed and confused. Latterly, the dextral hemiplegia became more pronounced, and the gait more insecure and tottering. The right toes scraped the floor, and the foot was planted heavily and clumsily, but the right hand could still be raised to the head. The mouth was slightly drawn to the left, and in protrusion the tongue deviated slightly to the right.

Finally, there was some bronchitis and congestion of the lungs, at first with vomiting, ending in slight pulmonary collapse at the front of the right lung, and in slight inflammation about its base posteriorly. The pulse ruled high (114-120); so did the temperature (103·5°, 103·6°, 103·2°, 103·2°); the respiration was deep (17 to 30). There were cerebral oppression, drowsiness and somnolence. The conjunctivæ were injected; the pupils rather small, sluggish, and equal. The palsy of the right arm increased. And now a half-rhyming, singing delirium appeared, single words or unmeaning phrases being monotonously repeated or chanted. Intestinal tympanites supervened, and, after four days' illness, death on Nov. 5th, 1875.

Passing now to the Necropsy, the calvaria was extremely unsymmetrical. The line of ancient fracture was marked on the external surface of the skull by an irregular ridge on the left parietal bone. Nearly 4in. in length, it began below and in front at the temporal ridge, and passed upwards and backwards to the sagittal suture, where it formed a bold elevation beside the parietal foramen. Along the middle of this rough ridge ran an irregular depression, grooving the straggling heaps of bone callus. Exactly corresponding with this groove on the external ridge there was, on the *internal* surface of the cranium, an angular depressed furrow in the bone 3¼ in. in length, and in front of this furrow were four bosses of spongy callus. The upper end of the fracture

adjoined the sagittal suture at a point 3in. from the anterior extremity of the suture and 1¾in. from its posterior. The lower end of this internal line of old fracture was 2⅜in. from the front end of the sagittal suture, and ¾in. from the coronal suture. To the furrow of fracture, and to the exostoses in front of it, were adhesions of the dura mater, and on removal of the calvaria the dura mater was left in a ragged condition and full of gaps to the width of ⅛in. in parts. Along this line, too, had portions of the pia mater and arachnoid both adhered to the dura mater, and separated with it, thus leaving areas of the grey brain-cortex denuded. Facing the line of cranial fracture, was a belt of cortical change which had ended in wasting and sinking of the grey matter, to which also the intervening girdle of locally thickened, tough and fibrous meninges had formed adhesions. There was some induration of the white cerebral substance immediately underlying the belt of atrophied grey matter. This belt of surface lesion started immediately at the great longitudinal fissure, 5in. from the tip of the frontal lobe, 3in. from the tip of the occipital, and trending downwards and forwards over the left hemisphere, ceased at a point 2⅜in. from the tip of the frontal, 5⅜in. from the tip of the occipital, and 2¼in., by the shortest cut, from the great longitudinal fissure. In this course it affected the anterior and median portion of the postero-parietal lobule, the upper portion of the ascending parietal convolution, a considerable portion of the middle third of the ascending frontal, the posterior part of the second frontal, and it terminated at the inferior frontal sulcus.

Scattered adhesions also existed between the left frontal lobe and the dura mater lining the left anterior fossa of the base of the skull, and between the dura mater of the right and the anterior surface of the right frontal lobe for ¼in. adjoining the great longitudinal fissure. Connected with these adhesions were marked cortical changes. For, on the orbital surface of the left frontal lobe, was an area of wasting of the cortex of the gyri, irregular in shape and with sinuous margins, occupying the anterior three-fourths of the orbital surface, destroying nearly all the cortex of the second, and of the front half of the first, and invading that of the third orbital convolution. Over this area the inner meninges were thick and tough, and adhered to the greyish and yellowish, gelatinous-looking, softened relics of the cortex. The cortical destruction also affected part of the adjoining surface

of the gyrus marginalis near its tip, and was limited above by the calloso-marginal fissure. A less degree of a like change obtained in a smaller area of the right frontal surface, especially the middle portions of the gyrus rectus and second orbital convolution. A minor degree of a similar condition affected a portion, the size of a shilling-piece, of the inferior surface of the tip of the left temporo-sphenoidal lobe, invading parts of the third temporo-sphenoidal gyrus, "uncus," and lobulus fusiformis, and here, in the anterior part of the left middle fossa of the skull-base, were some brain-adhesions to the dura mater, but without meningeal thickening.

There was also a patch of adhesion, but here of the soft meninges only, over an area the size of a sixpenny bit, on the left first frontal gyrus, one inch from its termination posteriorly.

Independently of the local inflammatory thickening already mentioned, there were some general thickening and increased consistence of the meninges on both sides, and the velum interposition participated in these changes.

The cerebral grey cortex generally was rather thin, pale, and indistinctly stratified, especially in the frontal and parietal regions, and more so in the left than in the right hemisphere. There were no marked changes in the insula. On both sides the white brain-substance was rather pale, and, except at the parts already specified, both it and the grey were of ordinary consistence. The grey commissure was unduly fragile, the fornix slightly softened. The left opto-striate bodies were less plump than the right, but no very marked difference between them or change, was observed on section. Basal arteries healthy; serosity moderate. The right hemisphere weighed $20\frac{7}{8}$ozs.; the left $17\frac{15}{16}$ozs., or almost 3ozs. less than the right; the cerebellum $4\frac{1}{2}$ozs.; the pons and medulla oblongata $\frac{7}{8}$oz.

It is unnecessary to describe the thoracic and abdominal viscera, most of which were fairly healthy, except for slight vegetations on the mitral and tricuspid valves, pulmonary congestion at the bases, and a patch of pulmonary collapse at the lower border of right upper lobe.

The following were the microscopical appearances:—

At the belt of left parieto-frontal lesion, and in the white substance beneath—much connective tissue overgrowth; proliferation of nuclei; grey non-staining rounded nucleated bodies; bodies resembling amyloid corpuscles. In the cortex

adjoining this belt—proliferation of nuclei, and some nerve-cell and vascular changes as described below.

At the lesion of left orbital surface—granule masses; meshes of wavy reticulate tissue, entangling bodies like amyloid bodies. The grey matter adjoining this change was also invaded by the amyloid-like bodies and increase of connective tissue, and its nerve-cells were obscured by molecular deposit, which also affected some vascular walls.

Tip of left frontal lobe—rather granular nerve-cells; scattered hæmatoidin masses, and the same fringing the minute vessels; interspersed fine reticular tissue; and round or oval cells $\frac{1}{1500}$ to $\frac{1}{1000}$ in. in diameter. Left third frontal g.—much the same, some of its nerve-cells shrunken. Somewhat similar neurogliar and cellular changes and formations, moderate in degree, as also granular deposits in the vascular walls, were observed in the left ascending parietal; and, to a less degree, in the right third frontal, and right ascending parietal.

In conclusion, I may summarise by saying that, in this case there were, *pathologically*:—

Brain injury, and resulting local inflammation.

Permanent local destruction of parts associated with local irritative lesions.

Secondary wide-spread morbid processes, nutritive failure, and degenerative changes, all mainly of the left cerebral hemisphere. The lesions of the orbital cortex had followed bruising and pulpification due to counterstroke.

And *clinically*:—

Epileptiform seizures.

Mental disorder and defect assuming the character of incoherence and dementia.

Dextral paralysis and impairment of tactile sensibility.

To explain these symptoms by the lesions, we have, as a factor, the chief injury, at once destructive and irritative, occupying a tract coursing through the very heart of the cortical motor zone.

(*a*). This sufficiently accounts for the epileptiform seizures.

(*b*). To this, in part, also may be assigned the right hemiplegia; to post-epileptic exhaustion of this motor cortex, and of the motor centres connected therewith along the line of convulsive incitation, may be referred the evanescent post-epileptic increase of paralysis; and its permanent increase to the general left cerebral atrophy. That the arm suffered less, relatively to the leg, was a point as to which the teach-

ing of the case was not in harmony with the localisation schemes of those who place important arm centres in the middle of the ascending frontal convolution.

(*c*). To the extensive degeneration of the left cerebral hemisphere, and to the more enfeebled circulation in, and lower temperature of the right limbs, may be attributed their defective sensibility.

(*d*). The occasional turning-movements were probably due to irritation reflected from the cortex to parts at the base.

(*e*). As for the mental symptoms, there were more or less loss and disorder of motor ideation by the lesion of the left cortical motor zone; some failure of power of attention, of mental concentration and ideational control by destruction of part of the frontal cortex; and general degradation of the mental powers by the secondary extensive nutritive changes, more particularly affecting the left hemisphere.

The Conditions Necessary for the Successful Training of the Imbecile. By David Brodie, M.D., Liberton, Edinburgh.

The education of the imbecile is an enterprise the inception of which dates from a very recent period. The generation has not yet passed away, which frankly insinuated that the idea of teaching the idiot could only enter the brain of one somewhat closely related to the class. It is now everywhere recognised as an important and imperative duty which cannot be neglected without shame and loss; yet for efficient practical effort, it is still but the day of small things.

The inquiry as to the extent to which idiocy prevails, the investigation of the causes which operate in its production and also the very varied conditions under which it is presented, we leave aside, while we invite attention to the conditions under which the improvement of imbeciles can be most efficiently prosecuted.

The relation legally established between imbecility and lunacy has supplied a most embarrassing complication in dealing with educable imbeciles. There is no reason in the world why juvenile imbeciles, in all their varied phases, should be classed with lunatics, or in any way subjected to the regulations which are in force in relation to that section of society.