since, gradually obliterating the lung and giving rise to increasing dyspnoea. Death was probably caused by the congestion of, and eruption of military tubercle in, the left lung, by which the remaining breathing surface was further reduced in extent. The rise of temperature between April 27th and May 4th was undoubtedly due to the tuberculosis. The case may be considered a typical one of fibroid phthisis; the dyspnoea and the changes in the shape of the thorax being very well marked, and the life-history and *post mortem* record filling up almost every link in the evidence.

I have notes of a somewhat similar case, where the left lung was the seat of fibroid phthisis, and caused a remarkable amount of displacement of the neighbouring structures. In this patient, the left side measured two inches in circumference less than the right; the spine was curved, the convexity being towards the right; the left shoulder was depressed; and the physical signs denoted an old cavity at the apex of the left lung, and nearly entire absence of breathing over the rest of the side. The right lung was drawn halfway across the left chest, the heart's apex tilted to the left, and the stomach raised as high as the fifth rib. The right lung was healthy. The patient died from abscess of the brain, and *post mortem* examination exactly confirmed the diagnosis of the state of the lungs indicated by the physical signs. The left lung contained a small cavity at the apex, and consisted of a hard fibroid mass, impermeable to air, and occupying hardly a third of its proper area, the rest of the space being filled up by the encroachments of the adjoining organs, as had been described during life. This patient lived four or five years from the date of the first symptoms, and might have lived much longer but for the cerebral disease, which seemed quite unconnected with the pulmonary lesions.

The *prognosis* depends on the amount of lung involved in the fibrosis, and on the rate of its contraction. Where half a lung, or at the most one whole lung, is permeated by alveolar fibrosis, and where this process is limited by the white bands of interlobular tissue—where, in fact, matters remain at a standstill—there the patient may live for years, provided he limits his exertions in proportion to his breathing powers, and does not place himself under circumstances to induce a fresh inflammatory attack or necrobiosis in the old fibrotic lesion. The patients who trace their disease to pleurisy seem to have the best chance of life, for their cases last longer than those arising from pure interstitial pneumonia. I have records of two cases, one lasting ten years and the other fifteen, each of which commenced in pleurisy. In both of these, excavation took place; but their health remained tolerably good until the opposite lung was attacked. In other instances, fibrosis has proceeded rapidly, permeating the whole lung, and, by obstructing the vessels, inducing dilatation of the right side of the heart and dropsy of the abdomen and lower extremities, the kidneys usually becoming affected towards the close of life. It is remarkable how general the rule appears to be that, when fibrosis predominates over the other morbid processes, the life-history of the case becomes at once changed; the typical consumptive character is lost; obstruction to the respiration and circulation, with accompanying dyspnoea and dropsy, close the scene, contrasting sharply with the termination of catarrhal phthisis or scrofulous pneumonia. It will be seen that the prognosis, therefore, depends on the amount of lung involved. Fibrosis strangles one patient; it saves another by limiting the area of consumptive disease, and thus preventing fresh absorption and tuberculosis.

Treatment.—Our objects in the treatment of fibroid phthisis are—first, to limit the area of disease, and to prevent any fresh portion of the lungs becoming involved either by fibrosis or by inflammation; secondly, to maintain, or possibly to raise, the standard of nutrition in the patient, and thus obviate any tendency to necrobiosis in the fibroid material. The patient must avoid all great exertions, which lead to lung congestion, and should live in an equable atmosphere, to guard against fresh catarrh or pneumonia. The dry warm climate of the Riviera, or, again, the still drier one of Upper Egypt, have been found most useful for this purpose; and many of these cases have lived on for years by wintering in these localities. The improvement of the nutrition must be attempted by the usual antiphthisical treatment, such as cod-liver oil, strychnia, the mineral acids and bitters, and plenty of easily assimilated food. The preparations of iodine have been recommended as likely to promote absorption of the fibroid material and counteract the advance of fibrosis; but in my hands neither these nor any other drugs have sufficed for this purpose, though I have seen great improvement take place in the general condition from steady perseverance in the use of the hypophosphites of soda, lime, or iron, and even of the less powerful phosphates and dilute phosphoric acid. The local inflammations of the pleura and lung are best combated by counterirritation with blisters or strong iodine liniments, and by promoting free expectoration. Where cavities or dilated bronchi exist, it is not uncommon to find foetid sputum, adding still more to the irritation, and

only evacuated by fits of coughing which close with vomiting. Here the stimulant expectorants, such as compound tincture of benzoin, tolu, and turpentine, are the most likely to relieve; while much may be done by antiseptic and stimulant inhalations of tar, creasote, carbolic acid, and thymol. The increasing dyspnoea and cardiac asthenia can be temporarily relieved by spirit of ether, by carbonate of ammonia; and the dropsy by purgatives and diuretics; but, if the obstruction in the lung have reached this point, we cannot hope for more than a temporary improvement by any medical aid.

PATHOLOGICAL ILLUSTRATIONS OF THE LOCALISATION OF THE MOTOR FUNCTIONS OF THE BRAIN.*

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THE object of the following communication is to record several cases which have come under my observation illustrating the localisation of cerebral disease, and tending, I believe, to show that the facts elicited by experimental research have a direct applicability to human pathology.

The experiments of Hitzig, Ferrier, Nothnagel, Schiff, Carville, Duret, and many others, are now universally known; and, although many varying explanations have been advanced by different schools of physiologists to account for the phenomena, yet, in the main, the broad fact that stimulation of certain regions of the cerebral cortex is followed by definite movements has been established on a firm basis. The results of these experiments have shown that, in the convolutions bounding the fissure of Rolando in man and those surrounding the crucial sulcus in the dog, cat, etc., there is a zone, the excitation of which causes movements on the opposite side of the body, and the destruction of which causes disorders of voluntary motion, or even persistent paralysis, while in regions closely related to these no such effects are produced. Since these facts became known, their application to human pathology has been a matter of the deepest interest and importance. Dr. Hughlings Jackson, from whom, if I may so speak, the "doctrine of localisation" took its inception, has recorded many cases illustrating the effects of localised lesions of the cortex in the production of definite symptoms varying in extent according to the position of these lesions; the recent annals of the various French scientific societies abound with similar records, and the almost daily observations of M. Charcot and his pupils at the Salpêtrière tend universally in the same direction. In addition, the histological researches of Betz, verified by those of Mierzejewski, give a kind of anatomical confirmation to these results by showing that, in the vicinity of the fissure of Rolando, and particularly in the paracentral lobule (the internal free surface of the ascending frontal and ascending parietal convolutions in the great longitudinal fissure), there exist large pyramidal cells similar to those of the anterior cornua of the spinal cord. As the result of his extended pathological observations, M. Charcot has now mapped out on the cortex of the human brain regions, destructive lesions of which cause paralysis more or less extensive, according to the extent of surface affected. These regions closely correspond to those mapped experimentally by Ferrier on the monkey's brain; and, in order to illustrate this clearly, I here exhibit a prepared hemisphere of the human brain in which the so-called *motor zone* is coloured black, while the non-motor region is white, the individual convolutions being indicated by different coloured discs lettered to correspond to this index. From this preparation, it can be seen that the motor zone includes the ascending frontal and ascending parietal convolutions (anterior and posterior central of Ecker), the posterior terminations of the three frontal, the anterior edge of the postero-parietal (superior parietal lobule, Ecker), and that of the inferior parietal lobule. The non-motor zone comprises the anterior portions of the three frontal convolutions, the posterior parts of the postero-parietal and inferior parietal lobules, the three occipital and three temporo-sphenoidal convolutions, together with those at the base of the brain and the island of Reil. Of course, between these two regions, there can be no hard and fast pathological line of demarcation drawn, the motor region insensibly blending into the non-motor and extending probably in one brain farther than in another, according, it may be, to the size and configuration of the individual convolutions.

The cases I am now about to relate are illustrations of lesions occurring in the non-motor and motor regions, and they tend, I believe, to

* Read in the Section of Psychology at the Annual Meeting of the British Medical Association in Manchester, August 1877.

support M. Charcot's views, that in man lesions within the motor zone are accompanied by motor disturbance, while lesions in the non-motor areas produce no such symptoms. Taking first the non-motor region, the three following cases afford examples.

CASE I. *Glio-sarcomatous Tumour of the Left Temporo-sphenoidal Lobe; Softening of Middle Portions of Superior Middle and Inferior Temporo-sphenoidal Convolutions; Circumscribed Hæmorrhages into Softened Tissue: no Motor Symptoms.*—N. C., female, aged 80, died on March 25th, 1876. She had been an inmate of the asylum for many years, suffering from quiet dementia. She continued in good health up to a short time before her death, and to the last possessed normal power over the limbs and various muscular groups, for a few days previously being somewhat dull and lethargic, but sinking gradually without any further symptoms becoming apparent. On removing the scalp, a bulging was observed on the surface of the left parietal bone, near its lower border. The calvarium was thinned and with difficulty removed, owing to its inseparable adhesion to the dura mater. On forcibly removing the bone, the latter membrane was seen to be almost incorporated with it, so firm were the adhesions; and attached to the inner surface of the parietal, inferiorly, was a flattened tumour as large as a small apple, growing from the lateral surface of the left temporo-sphenoidal lobe, about the centre of its superior and middle tier of convolutions. This tumour was incorporated with the brain-substance, and was soft and of a reddish-grey colour. Subsequent microscopical examination showed it to be glio-sarcomatous in its nature, consisting of numerous widely dilated thin-walled vessels and masses of round ovoid and elongated cells closely aggregated, without any appearance of intercellular substance. These cells contained granular nuclei, and varied in size, some being as small as the nuclei of the neuroglia, while others were as large as those found in sarcomata occurring elsewhere. The groups of cells appeared to be arranged concentrically around the vessels, and interspersed amongst them were many large sized round cells (giant-cells). On the separation of this tumour, a softened cavity was disclosed deep in the substance of the temporo-sphenoidal lobe, as large as a good-sized walnut, and containing whitish curdy matter. The brain-tissue around this cavity was broken down, and above it were found two fresh circumscribed globular blood-clots as large as hazel-nuts. The pia mater generally was anæmic, and contained clear fluid beneath it; ramifying in it were very beautiful congeries of tortuous vessels filled with blood, closely resembling injected Malpighian bodies in the kidney. The membrane stripped easily, and the convolutions beneath were wasted and separated by broad and shallow sulci, but presented no further localised lesions. A section through the centrum ovale showed the brain-substance to be anæmic; an oval patch of a reddish colour blending with the surrounding tissue existed in the posterior part external to the posterior cornu of the lateral ventricle. The lateral ventricles, opto-striate bodies, and basal ganglia were normal in appearance. The arteries at the base of the brain were atheromatous in patches. Microscopical examination of the grey matter from the frontal parietal and occipital convolutions showed the larger nerve-cells to be in the second and third stages of pigmentary degeneration.

On this chart of the hemisphere (fig. 1), I have indicated the external

Fig. 1.—Glio-Sarcomatous Tumour of Left Temporo-Sphenoidal Lobe; Softening of Middle Portions of Superior Middle and Inferior Convolutions; Circumscribed Hæmorrhages into Softened Tissues.

site of the tumour by a black disc, while the zone of softening around it has been coloured yellow. From this, then, it can be seen that the lesions were strictly confined to the temporo-sphenoidal lobe, and, though they originated in a morbid growth, regarding which great

caution must be exercised, from the fact that such growths may displace tissues without destroying them, and thus produce indefinite phenomena, yet, from the isolated position of the tumour here, being separated from the remainder of the hemisphere by the Sylvian fissure, and from the fact that, though it did lead to destroying lesions of the adjacent brain-substance, no motor symptoms appeared, I think the case may be made use of for the purposes of this study.

CASE II. *Localised Deposit of Lymph on Dura Mater over Posterior Portion of Postero-Parietal Lobule of Left Hemisphere; Adhesion of Membranes; Superficial Erosion of Cortex: no Motor Disturbance.*—P. S., male, aged 66, died on September 9th, 1876, of diarrhoea. This patient had been an inmate of the asylum since April 1868, suffering from chronic dementia. Beyond the dulness and hebetude consequent on his mental condition, he presented no symptoms calling for special note; there were no motor disturbances, the man being able to move about freely until he became affected with the diarrhoea to which he eventually succumbed. On *post mortem* examination, the calvarium was soft under the saw; about four ounces of sanious serum escaped during the removal of the brain. The dura mater was strongly adherent to the frontal bone, requiring considerable force to separate it, which was, however, done without tearing the membrane. Over the posterior portion of the postero-parietal lobule of the left hemisphere was an irregularly ovoid patch of thick and shaggy organised lymph larger than a crown-piece, forming a growth on the inner surface of the dura mater, and inseparably connected with the latter membrane; this was slightly adherent to the pia mater, and the latter to the cortex of the postero-parietal lobule, leaving, when stripped from it, a patch of superficial erosion, which I have coloured blue on this chart (fig. 2). The convolutions generally were

Fig. 2.—Localised Deposit of Lymph on Dura Mater over Posterior Portion of the Postero-Parietal Lobule of Left Hemisphere; Adhesion of Membranes; Superficial Erosion of Cortex.

somewhat atrophied, but no further gross lesion existed. Microscopical examination showed the nerve-cells to be affected with pigmentary degeneration.

In this case, the cortical lesion was superficial and strictly confined to the postero-parietal lobule. In several of the cases recorded or alluded to by M. Charcot, in which motor disturbances existed, this region was found affected; but other and undoubtedly motor regions were also implicated. As there were here no motor disturbances whatever, this case would seem to indicate that, when involved alone, disease of this region will not give rise to such symptoms.

CASE III. *Softening of Gyrus Supramarginalis and Centre of Occipital Convolutions of Right Hemisphere; no Motor Disturbances of Left Side; Lesions of Left Hemisphere causing Aphasia and Right Hemiplegia.*—In this case, which I have already fully recorded elsewhere (*Journal of Mental Science*, October 1876), there were "foyers" of softening occupying the gyrus supramarginalis and portions of the occi-

pital convolutions of the right hemisphere, while no motor disturbances at the left side manifested themselves, the lesions in the left hemisphere sufficiently accounting for the motor disturbances at the right side. The lesions in the right hemisphere are indicated on this chart. (Fig. 3.)

Turning now to the motor zone, the following cases are illustrations of lesions occurring there, and accompanied by motor disturbances.

Fig. 3.—Softening of Gyrus Supramarginalis and Centre of Occipital Convolutions of Right Hemisphere.

CASE IV. Meningeal Hæmorrhage; Pressure on Left Ascending Frontal, Ascending Parietal, and Edge of Inferior Parietal Lobule; Right Hemiplegia.—M. N., female, aged 62, died on March 27th, 1876. She had been an inmate of the asylum for six years, suffering from chronic insanity, with periods of maniacal excitement. About a week before her death, she complained of a feeling of general weakness and undefined pains about the chest, which, however, could not be traced to any apparent cause; her appearance was florid and healthy, and, beyond some slight general weakness, she had no loss of motor power over the limbs. On March 25th, 1876, she became noisy and troublesome; and, two days afterwards, about eleven o'clock in the forenoon, while sitting quietly before the fire in the day-room, she suddenly slid off the form and became semi-unconscious. On being placed in bed, she was found to be semicomatose and paralysed on the right side, the limbs being quite flaccid. When spoken to loudly, she was able to mutter indistinctly; but did not appear to comprehend what was said; the face was cold and collapsed; the pulse slow and weak. The coma gradually deepened, and she sank in about two hours. *Necropsy.*—The skull was asymmetrical, the right parietal eminence bulging; the calvarium was soft and easily sawn through. The dura mater was intimately adherent to the left half of the frontal bone, and was thickened and opaque, especially over the right hemisphere, where its inner surface was lined with a thin layer of gelatinous material easily scraped off with the scalpel, and probably the decolourised remains of a former hæmorrhage. The great longitudinal sinus contained a partially decolourised thready clot. The right middle meningeal artery was atheromatous, and appeared torn in several places. There was much hypostatic congestion of the vessels of the pia mater over the posterior lobes of both hemispheres. On the left hemisphere, confined to the lower half of the ascending frontal, the ascending parietal, and portion of the anterior edge of the inferior parietal lobule, a soft clot lay on the pia mater, from which it readily separated. On removing the brain, the surface of the bones forming the anterior and middle fossæ of the left half of the skull were covered with a very thin layer of recent blood-clot, which extended from the olfactory groove on the ethmoid, along the orbital plate of the frontal bone, the petrous and lower part of the squamous plate of the temporal, and the inferior and lateral surfaces of the occipital. This extravasation was in great part little more than a colouration of the osseous dura mater with blood; it was somewhat thicker at the olfactory groove, and had insinuated itself beneath the left olfactory nerve, where a small elongated coagulum was also found. This extravasation had probably been continuous with that found lying on the convolutions, and was effused from a ruptured meningeal vessel, which, however, could not be found. The convolutions generally were atrophied and diminished in consistence, especially those of the parietal lobe of the left hemisphere, where, at the junction of the gyrus angularis and gyrus supramarginalis (Ecker), a depression as large as the top of a finger existed, over which was a bulla of clear yellow fluid (fig. 4). On section, the brain-substance was anæmic; the grey matter was thinned, especially in the occipital lobes. The ventricles and commissures were normal, and the opto-striate bodies

plump and regular; the basal ganglia were healthy. The left internal carotid artery was atheromatous; the basilar and vertebrals were studded with similar patches. Microscopical examination in the recent state showed the larger ganglion-cells to be in an advanced state of pigmentary degeneration, and, in some instances, in what appeared to be a condition of calcification.

Fig. 4.—Meningeal Hæmorrhage; Pressure on Left Ascending Frontal, Left Ascending Parietal, and Edge of Inferior Parietal Lobule.

In this case, an acute apoplectic seizure occurred, the necropsy revealing recent meningeal hæmorrhage; hence there can, I think, be no doubt that this lesion and the paralysis which supervened stood to each other in the relation of cause and effect. Now, although the extravasated blood was effused over a considerable extent of surface, yet it was only on the ascending frontal and ascending parietal convolutions, and in the olfactory sulcus, that it had assumed the characters of a regular clot, and was attached to the surface of the brain; hence I think it equally certain that to the pressure or presence of the clot in this portion of the motor region the right hemiplegia was due, the extent of the paralysis and the fatal result being probably accelerated by the already weakened and degenerate condition of the nerve-centres generally, which were in consequence unable to bear up against the sudden shock.

[To be continued.]

SPONTANEOUS RUPTURE OF THE SPLEEN.*

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IN the following case, splenic apoplexy, and consequent rupture of the capsule of the spleen, took place under what I believe to be very rare and, perhaps, unique conditions. The case is of interest clinically, and also has a medico-legal importance to which a recent trial in India calls special attention.

The patient was a shepherd fifty-three years of age, admitted into the Bristol General Hospital on July 4th. He stated that, up to two months before admission, he had enjoyed good health; then he began to suffer from nausea and pyrosis, and entirely lost his appetite. He had done no work for six weeks. A month before admission, he had an attack of epistaxis, which lasted about twenty-four hours, during which time he lost much blood. Since this, he had noticed a sensation of beating in the upper part of the abdomen. Just before admission into the hospital, another severe attack of epistaxis came on; the patient was, however, able to walk up into the ward without help.

When I saw him next day, he was almost blanched from loss of blood, but cheerful and anxious to give all the information in his power. The heart's impulse was weak, and there was a faint systolic apæmurmur; the pulse was feeble and the vessel degenerate. The abdominal aorta was pulsating to a marked degree, but no distinct dilatation of the vessel could be made out; a systolic *bruit* was plainly heard in the vessel, but was found to be only due to pressure. There was no pain or tenderness in the abdomen.

I concluded that there was no aneurism of the aorta, and that the pulsation and *bruit* were due to the anæmic condition consequent upon the loss of blood.

Soon after 9 P.M. the same day, the patient, who up to that time had

* Read before the Bath and Bristol Branch.