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 consistency, 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.

seemed no worse, was suddenly seized with great dyspnoea and symptoms of collapse, which ended in death in less than an hour.

POST MORTEM EXAMINATION.—There was a large quantity of dark fluid blood in the peritoneal cavity. There was no aneurism. The capsule of the spleen was greatly distended; on the anterior aspect of its peritoneal surface there was a rent an inch long, through which the finger passed into a large space full of fluid blood. At first sight, it seemed as if the enlarged spleen were split longitudinally into two halves, which were separated along their whole length by this layer of fluid blood about an inch and a half thick; it was found, however, that the capsule had been stripped off the whole peritoneal surface of the spleen by blood effused between it and this surface, and that one of the apparent halves before mentioned was simply a firm layer of dark clot, about three-quarters of an inch thick, forming a thin cake applied closely to the inner surface of the capsule, and separated from the spleen itself by the layer of fluid blood before described. The exact source of the hæmorrhage could not be found. The splenic substance was of a brick-red colour, and very soft and pulpy (there had been considerable *post mortem* change). The spleen and clot together were eight inches long, five inches and a half broad, and three inches and a half thick, and weighed twenty-six ounces. The rest of the organs were very anæmic, but otherwise healthy.

REMARKS.—Rupture of the spleen is not a very uncommon occurrence; but almost invariably it is caused by external violence, such as blows, crushes, falls, wounds by fractured ribs. Instances have been met with, however, where the organ has ruptured spontaneously; in conditions where it is intensely congested, as in typhus, cholera, and the cold stage of ague, where it is probable that weakened texture has been combined with extreme congestion. But I have been unable to find any record of a parallel case to the one that I have related, where, at the time of the rupture, the patient was suffering from no acute disease, and had been quietly lying in bed for hours.

If we consider the state of the spleen *post mortem*, we see that the first event was hæmorrhage within the capsule, giving rise to the large clot before described, which, from its characters, must have been formed some time before the fluid blood was effused and the capsule gave way. There was the following sequence:—first, hæmorrhage between the spleen and the capsule; then cessation for a time; then more blood poured out, and in such quantity that the capsule gave way. Hence the actual rupture of the capsule was secondary and accidental.

Was there any congestion of the spleen as the first event? I think not; rather the opposite. The man had just been blanched by severe epistaxis, and there is no reason to suppose that the spleen would be other than anæmic, like the rest of the body. The conclusion must be that the same weakness of vessel that caused the repeated bleeding from the nose led to the hæmorrhage into this organ, especially as this occurred at a time when the force of the circulation had been much reduced by loss of blood.

I now turn to the medico-legal aspect of the case. Some months ago, a trial took place in India which excited much interest. The following short account I take from a medical periodical. "A curious medico-legal point was involved in a recent trial for manslaughter in Ceylon. A coolie was treated with some severity by the superintendent of a plantation, and died almost immediately from rupture of the spleen. The man had had fever lately; the spleen was large and soft. It was urged for the defence that the spleens of coolies sometimes rupture spontaneously on severe muscular exertion; that the exertion of running away might have ruptured the spleen; and that death would not be the direct result of the action of the accused. This theory was discountenanced by the medical witnesses, and we think rightly. An accident so rare, and even doubtful, as such rupture of the spleen must be, cannot be allowed weight in the presence of evidence of direct violence. The Chief Justice ruled that the rupture was distinctly the result of the violence. The prisoner was found guilty." If I remember rightly, the exact occurrences were these. The coolie received a box on the ear and fell down; he then got up, ran away, climbed over two walls, and, in so doing, fell from the top of one or both of them; he then quickly became collapsed and died. Here was plenty of muscular exertion; besides that, the fall from the wall represented a greater degree of violence than did the previous simple fall of the coolie off his feet. If the assault caused the rupture, it did so indirectly only by causing the fall; for there was no direct violence to the abdomen. And, if this were enough to produce the laceration, how much more were the muscular exertion and the accident that followed, with which the accused had nothing immediately to do! It seems to me also improbable that a man with a ruptured spleen would run away with the activity that this coolie showed.

With regard to muscular exertion as a cause of rupture of the spleen, cases illustrating this have occurred in this country; thus Sir James

Simpson refers to three cases of fatal rupture which occurred respectively during the pregnant, the parturient, and puerperal states; in each instance, after some unusual exertion. Another case is recorded in the *BRITISH MEDICAL JOURNAL* for 1874, by Mr. Atkinson of Leeds, where the accident was apparently due to violent vomiting set up by indigestible food.

The case I have related differs from all those that I have been able to find recorded in this important particular: that there was entire absence of any special muscular exertion; for the patient had, in fact, been in bed for hours before the rupture occurred. And I think that I am warranted in concluding that this instance has established the fact that rupture of the spleen may occur purely spontaneously, in the absence of any condition ordinarily associated with congestion of the organs, and apart from all external violence or special muscular exertion.

CLINICAL MEMORANDA.

CROUP: DIPHTHERIA: TRACHEOTOMY.

As Dr. Bowles of Folkestone has mentioned my name, among others, as holding some rather decided opinions (I hope not dogmatic ones) on the subjects specified above, perhaps he will excuse me for offering a few remarks on his very interesting cases published in the last number of the *JOURNAL*, and which I think may readily be explained in accordance with the pathological views now, as he states, fast gaining ground.

He has described three cases, two of which were fatal, tracheotomy being performed in both, but in neither of which was there any *post mortem* examination. Thus it is not absolutely certain what the disease really was; but, considering that Dr. Bowles distinctly saw the diphtheritic patches on the tonsils in each case, and that both of them died rapidly and unexpectedly, even although tracheotomy was successful, there is little doubt in my mind that the disease in both cases was diphtheria, first of the fauces and then of the larynx and trachea. His third case, which was most skilfully treated and was successful in its result, I believe to have been one of severe infantile laryngitis. It is true that Dr. Bowles says that "doubtless false membrane already existed" on the second day of the illness, but he gives no evidence of its existence; and, indeed, he distinctly states that he carefully examined the throat for false membrane on two succeeding days, and found none. It was not till ten days after the operation of tracheotomy had been performed, and after abundance of mucus and muco-pus had been discharged through the wound, that it is stated that on expiration, "false membrane was seen flapping through the wound". But as none of this false membrane ever escaped, and as the child was convalescent in two or three days afterwards, is it not possible that a part of the lining membrane of the trachea, or even of the integuments, may have presented the appearance of false membrane? It is certainly contrary to all experience that a tube of false membrane should be present in an infant's windpipe on January 20th, and that all bad symptoms should disappear and the tracheotomy-wound be closed on the 24th. The false membrane, if it existed, must have been discharged or extracted before the patient could recover.

It is by no means alleged, by those who maintain that the so-called membranous laryngitis is really laryngeal diphtheria, that ordinary laryngitis is not a dangerous disease in some instances and never requires tracheotomy. The narrowing of the glottis in young children renders laryngitis, with its complication, œdema glottidis, a very formidable malady. What is alleged is that such cases are far less serious than laryngeal diphtheria, and that in the worst of them, as is shown in Dr. Bowles's third case, the operation of tracheotomy has a much greater chance of success. There is no false membrane, and there is no specific poison; the absence of which conditions reduces laryngitis to the level of an ordinary inflammation.

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NOTE ON THE INCUBATION OF MUMPS.*

ON November 17th, 1877, I was asked how long a lady, who had accidentally been thrown in the way of mumps three days previously, need fear lest she had contracted the disease. Never having thought about the incubation of such a slight and usually home-treated ailment, I hazarded "a fortnight"; but was annoyed eighteen days afterwards to find I was wrong in my guess. I then consulted all the books on which I could lay hands, with the following result. Medical and surgical text-books both describe parotitis; but Tanner (sixth edition), Erichsen (sixth edition), Trousseau (Sydenham Society's edition),

* Read before the Norfolk and Norwich Medico-Chirurgical Society.