brain tissue proper is capable of this process, but of a type *per se*, and affecting the outer layers of the grey matter only in great intensity. But this process does by no means explain the real nature of general paralysis. The necessarily progressive character of that disease which is its most characteristic and essential feature, is not, as yet, explained. The crudest and most backward notion that has been lately broached as to the real pathological nature of general paralysis is that the local adhesions of the pia mater to the convolutions is the one "specific" pathological feature of the disease. Such a selection of one morbid appearance only as being the grand feature of a most complicated and subtle disease, carries its own refutation. What are we to make of the cases of epilepsy, &c., in which there is adhesion of the pia mater to the convolutions found after death? I had one such case the other day in which this was universal over one hemisphere. The unlikelihood of the adhesion theory reaches its climax when the further theory is tacked on to it, that the local spots, where those adhesions are so strong that portions of the brain tissue are removed with the membrane, are the only convolutions affected by the disease. It would not be a "general," but a local paralysis were this so.

CLINICAL NOTES AND CASES.

Pathological Notes. By A. Harbinson, M.D., M.R.C.S., Assistant Medical Officer Lancaster County Asylum.

Multiple Apoplexies.—The subject of these notes was a demented, undersized, ill-developed female epileptic, aged 36, subject to fits for a long time, but whose history is unknown.

On April 18th she had two severe and protracted fits with a short interval between. She recovered consciousness slowly and imperfectly, answered questions reluctantly, refused milk, but allowed herself to be fed with it, and remained in a state of passivity and torpor contrasting with her previous querulousness and irritability.

During the night and on the following morning she had three more slight fits without any further mental change. She died on the afternoon of the 19th. There is no doubt cerebral hœmorrhage occurred during the fits on the 18th; and although a suspicion of this arose at the time, yet it was

not entertained, owing to the return of consciousness, the pupils being normal, deglutition possible, and the extremities capable of voluntary movement (the feet and ankles alone escaping observation). The altered look and collapsed condition were not cause for wonder, as independently of the accession of fits she could not have survived for many days the exhaustion due to diarrhœa, tuberculous ulceration of the bowels, and perforation of the caput cœcum coli, with localised abscess in the peritoneum. The latter curiously found exit through the umbilicus, having previously destroyed the peritoneum and tissues over the iliac bone, and exposed and isolated the cords of the lumbar plexus.

On post-mortem examination the abdominal state was as described. In the lungs there were deposits of tubercle and slight excavation. There was no marked hypertrophy of the heart, renal degeneration or atheroma of the vessels. The brain was small, but heavy (50¾ oz.), the cerebrum being sclerosed in the posterior half. The membranes were thickened on the vertex and the dura mater adherent to the calvarium. The veins and sinuses were very full of blood and dense clots. The arteries were not atheromatous, but very tough and resistant, pulling out on slicing the brain.

Symmetrically situated in each postero-parietal lobule was an apoplexy the size of a walnut, containing in its centre several separate and distinct, soft, recent clots (three on the left and five or six on the right side), from the size of a pea to that of a cherry. In the third left occipital convolution was another clot, the size and shape of an almond, splitting up the white fibres; and in the second left temporo-sphenoidal was a rather smaller one. For some distance round all these clots the brain substance, but particularly the grey matter, was broken down and mixed with blood, forming a soft red pulp. The convolution round the end of the left parieto-occipital fissure and the first right occipital convolution were similarly reddened and disorganised. Besides these six extravasations there were found three calcareous tumours, on the left side. The largest, weighing 21 grains, honeycombed, and irregular in shape, was situated in the ascending frontal convolution, half way up, and just reaching the surface in the fissure of Rolando. The second was in the tip of the occipital lobe, and the other in the first temporo-sphenoidal convolution. The left anterior cerebral artery was only about half the size of the right, and they were connected by two communicating branches.

The points to which I may direct attention are—

I. The number of extravasations. In Reynolds' system of medicine, Dr. Hughlings Jackson states that "sometimes two or more recent clots, even large clots, are found in different parts of the brain." He mentions a case of Mr. Llewellyn's, in which there were two clots, one of Dr. Ogle's, in which there were three, and one of Dr. Baümler's, in which four recent clots were found. Dr. W. A. Hammond says that "of 139 cases cited by Durand-Fardel twenty-one were multiple; eighteen of these were double and three triple. In my own experience two cases of triple lesions have occurred and two of double lesions." According to Niemeyer "usually there is only one hæmorrhagic effusion in the brain, rarely several."

II. The occurrence of apoplexy during an epileptic seizure. "Apoplexy is so rare a sequence of epilepsy that it is mentioned simply for the purpose of stating this fact" (Russell Reynolds). Niemeyer also refers to it as rare, and Hammond does not mention it.

III. The absence of such grave symptoms, mental and otherwise, as might be expected from so extensive lesions. Is it possible that the indurated, sclerosed condition of the cerebrum, in which all the extravasations occurred, neutralised to some extent the force of the blood in the ruptured vessels and prevented transmission of the pressure to more important parts, or had the brain become so habituated to epileptic congestion that the pressure was borne with comparative impunity?

IV. The position of the hæmorrhages. The upper surface of the hemispheres is not very frequently the seat of apoplexies.

V. Regarding the motor centres, it is interesting to note, as the probable starting point of the epilepsy, the presence of an irregular, jagged, calcareous tumour in that part of the ascending frontal convolution which, according to Ferrier, produces action of the elevators of the angle of the mouth. Her fits mostly occurred at night, and unfortunately having never seen her in the very beginning of one, I am unable to say what the initial movements were.

The extravasations avoided Ferrier's motor centres, except as regards the postero parietal lobule, and excluding the latter the absence of paralysis was therefore to that extent corroborative of his experiments. She was able to draw up her legs, but as already stated, it was not observed whether the

foot and ankle movements were affected by the hæmorrhage into the postero-parietal lobule.

As the following case bears some sort of resemblance to the foregoing, I may briefly mention the particulars :—

Lydia S—, an epileptic, aged 49, on the 26th of April, 1876, in a quarrel and violent passion, was in the act of whipping off her boot to strike another patient, when she suddenly fell forward on her face in a fit. She was turned over by the nurse, was severely convulsed, became very dark in the face, and died in a few minutes. Her fits had always been very severe, and without any warning.

The following extracts are from the post-mortem record :— "The right side of the heart is almost entirely replaced by fat." "Blood effused beneath the membranes of the brain on the upper surface, about the cerebellum, Sylvian fissures, and indeed nearly all over." Death was certified to result from apoplexy, but whether the apoplexy was preceded by an epileptic seizure must remain uncertain. My impression at the time was, and still is, that the sequence of events was first the violent mental perturbation and passion, second the epileptic attack, and lastly the apoplexy and sudden death.

Tumour in Second Frontal Convolution of Brain.—Isabella B—, aged 40, died on June 1st from acute pulmonary tuberculosis. On examining her brain, a cylindrical tumour—one line and a half in diameter by three long—was discovered in the superior posterior angle of the second left frontal convolution, one-third of an inch from its superior and posterior borders. It appeared to be of old standing, was soft, like inspissated pus, and was easily enucleated. This woman had a peculiar sort of squint from an early age, and when eighteen years old was actually operated upon, but without success.

Ferrier, on stimulating the posterior extremities of the first and second frontal convolutions, obtained lateral movements of the head and eyes, elevation of the eyelids and dilatation of the pupils. The tumour in this case was confined to a small part of this region, and whether the squinting condition of the eyes was due to the limited lesion, or was merely a curious coincidence, I cannot pretend to say. Perhaps in future experiments it would be worth while defining more exactly the effects produced by restricting the stimulation to the particular spot referred to.