abdomen two days before admission, with cold shivers. There had been some indefinite trouble with the bowels about 12 years before. On admission her temperature was 101° F. and the pulse 120; the abdomen was distended and very tender on the right side, where a soft swelling could be felt. Operation was as follows. A median incision was made. Free fluid was present in the peritoneum, serous generally, but puriform in the right loin. The bowel was then examined and a gangrenous broken-down mass was found, originating in a Meckel's diverticulum, and necessitating the resection of 15 inches of the small intestine. The appendix was normal. The patient remains well.

The specimen (see figure) is preserved in the museum of the Bristol Royal Infirmary, and Professor I. Walker Hall gave

The abdomen contained free fluid, with localised stinking pus in the right loin. The appendix was normal. The omentum formed a hood adherent over the mass, and is represented as having been partly cut away to show the structures beneath it. (Surgical Specimen No. 522, Museum, Bristol Royal Infirmary.)

the following microscopical report which he has subsequently confirmed: "Sections taken from separate parts of the tumour show masses of endothelioid cells and dilated blood sinuses, adult in type. The lesion is therefore a hæm-endothelioma."

Although the instances are so few in which inflammation of Meckel's diverticulum may simulate appendicitis, yet this should be borne in mind; and it may be of interest to append references to such recorded cases, grouping them together.

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Clifton, Bristol.

INDICATIONS FOR OPERATION ON CEREBRAL TUMOUR FROM THE POINT OF VIEW OF THE OPHTHALMIC SURGEON.¹

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I FEEL that my intrusion in the discussion on indications for operation in cerebral tumours demands some apology, but there are one or two aspects of it where the opinion of the ophthalmic surgeon may become requisite, and with those alone I shall deal as briefly as possible. His opinion may be asked firstly as to whether operation is necessary in a particular case to prevent loss of sight, or whether operation will be of any avail in procuring any recovery when vision has already failed or begun to fail. If the tumour can be definitely localised, the sooner operation, either palliative or radical, is done the better, not only for the sight but also for the life of the patient. But if the position of the tumour has not been determined, and if marked papilloedema is present, it becomes the duty of the ophthalmic surgeon to give his opinion as to whether a palliative operation must be performed to save sight, and, if so, when it must be performed. In other words, he must be prepared to say what delay may be allowed in the hope of definite localising signs manifesting themselves without serious risk to the sight of the patient. In attempting to give an answer to this question I must briefly refer to some of the pathological features of tumour papilloedema.

In the early stages of tumour development, if the tumour itself is not directly pressing on the chiasma or on one or other of the optic nerves, the increased pressure in the chiasmal neighbourhood is probably mainly a fluid pressure; at any rate, it is not sufficient to interfere with the normal conduction of nerve impulses from the retina, except possibly in a temporary way. We do get fleeting attacks of blurred vision or possibly, according to Cushing and Bordley, temporary dyschromatopsia, but there is no definite diminution of visual acuity and no obvious loss of peripheral field. In the optic disc at this stage the principal changes seen are due to the oedematous fluid filling up and squeezing apart the meshwork formed by the interlacing of the nerve fibres and the fibres of the pars choroidalis of the lamina cribrosa. There is as yet no degeneration of the nerve fibres and no increase in the connective tissue elements in the disc. If at any time while this stage persists intracranial tension be restored to the normal level, there will be a restoration to the normal condition of all these affected tissues, and no trace may be left to show that they have ever been affected. Even where the oedema has been so great as to overflow the disc neighbourhood and run along the fibres of the papillo-macular bundle and give rise to a macular fan, complete restoration of normal appearances may take place on relief of pressure, and I have seen such cases 12 months after operation in which it was almost impossible for any candid observer to say that there had ever been optic neuritis.

¹ A paper read at a combined meeting of the Sections of Neurology and Surgery at the International Congress of Medicine, London, 1913.

In the next stage, however, changes commence which do not allow of such complete recovery. First, as regards the intracranial portion of the optic nerves and the chiasma, the fluid pressure becomes replaced by a more solid pressure. We know that as the tumour develops the convolutions become flattened, the medulla is forced down into the foramen magnum as a pressure cone, and similarly the frontal lobes and temporo-sphenoidal lobes are forced on to the optic nerve, and, as Behr and Saenger have shown, the dural fold that overlaps the optic nerve may actually leave its impress on its surface. At the same time the distended floor of the third ventricle may be pressing down on to the chiasma. If this more solid type of pressure is prolonged or if the tumour itself grows so as to press directly on the optic nerve, then the papilloedema may subside and a descending or pressure atrophy take its place, and so you may get the seeming contradiction of a descending atrophy appearing in a disc which has been definitely affected by optic neuritis. And it is a point worthy of consideration whether the early onset of optic atrophy in cerebellar tumours is not in many cases brought about in this way.

When we turn to the disc itself we find that if the oedema persists secondary changes inevitably follow. The stretched nerve fibres, distorted and forced out of their usual course and bathed in cedematous fluid, begin to show moniliform swellings, and here and there these swellings increase and the constituent fibrillae break up and cytoïd bodies are formed which ultimately undergo further disintegration. The neuroglial cells at the crest of the swelling begin to proliferate, and if these degenerative and proliferative processes are allowed to proceed no subsequent relief of pressure will benefit vision. Indeed, in these cases the visual failure may progress even after pressure is relieved, for the subsequent cicatricial contraction of the newly formed neuroglial tissue causes a further atrophy of any nerve tissue that is still functional at the time of operation. The visual loss that is due to direct pressure on the nerves intracranially may be recovered from if operation is undertaken before much pressure atrophy has developed. Visual loss that is due to degenerative changes in the disc itself may, and probably will, be progressive even after operation. So long only as the papilloedema remains in the first stage without evidence of visual loss and without evidence of secondary disc changes is it justifiable to delay palliative operation.

The second point to which I wish to refer briefly is whether in the present state of our knowledge we can place any reliance on a difference in the amount of papilloedema in the two eyes as a valuable factor in the localisation of the tumour. I hold very strongly the opinion that we cannot do so. My own published statistics are based on 102 cases where the position of the tumour had been definitely ascertained by operation or necropsy, and where there were no signs of atrophy or subsidence in either disc. In 54 of these cases the swelling was either equal or the difference did not amount to more than 0.5 D. In 25 the swelling was greatest in the eye on the side of the tumour, and in 23 it was greatest in the eye of the opposite side. Time will not permit me to go into further details about these cases which have already been published. I hope in the course of a few months to be able to give detailed statistics of another large series of cases which I have seen since the last set was published.

At the Heidelberg Congress last year Uthoff gave the following statistics based on a summation of his own cases and cases collected from the literature. In the case of supratentorial tumours he found that one-sided choked disc occurred in 56 per cent. of cases on the side of the tumour, and in 44 per cent. of cases on the opposite side. If the disc changes were not marked enough to be called "choked disc"—i.e., less than 2 D. of swelling—the proportion was more in favour of the side of the tumour, 75 per cent., and 25 per cent. on the opposite side. When there was choked disc on both sides, but more marked on one side than the other, it was in 73 per cent. greater on the side of the tumour, and in 27 per cent. greater on the opposite side. When there was optic atrophy on one side and papilloedema on the other side the tumour was invariably on the atrophic side. When the papilloedema took on a hæmorrhagic character it was as often more marked on the contralateral side as on the same side. As regards cerebellar tumours, he points out that one-sided optic neuritis is very rare. When

it occurs "die Geschwulst ist ebenso oft auf der gleichen als auf der entgegengesetzten Seite."

I cannot refrain from quoting here the words Professor Rothmann used in his contribution to the discussion on cerebellar tumours: "Absolut trügerisch und irre führend ist die frühere Auftretung und die stärkere Entwicklung der Neuritis optica auf einer Seite; oft genug kommt sie zuerst auf der der Kleinhirn-Affektion gegenüber liegenden Seite zur Beobachtung." "The earlier appearance or stronger development of optic neuritis on one side is an absolutely deceitful and misleading sign. Frequently it is first seen in the opposite eye from the affected side of the cerebellum." Now I would extend this dictum to intracranial tumours generally, and say that in too many cases it is seen that the papilloedema either first shows itself or is more strongly marked in the contralateral eye for us to use this as a valuable localising sign. The appearance of descending atrophy in one eye and optic neuritis in the other is a sign of very great value, and indicates that the tumour is on the atrophy side pressing directly on the nerve. But until we have more knowledge than we have at present any difference in the amount of swelling or the appearance of the two discs will too often prove a misleading sign.

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AN UNUSUAL CASE OF CEREBRAL ABSCESS.

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THE patient, a male aged 41, was admitted to the Bradford Union Hospital on April 25th, 1913, in a comatose condition. The history of his illness was difficult to obtain, but he was said to have been treated by a medical man for influenza over a period of seven days. An examination of the patient revealed the following. He was comatose and was bathed in a profuse sweat. His cheeks were flushed, and his pulse full and bounding. The pupils were equal, of medium size, and reacting sluggishly to light; there was no squint and no optic neuritis. There was marked facial asymmetry, the left side showing partial or supranuclear palsy of the seventh nerve. There was no retraction of the head. The limbs of the left side were more flaccid than those of the right, but the reflexes were normal and equal on both sides. The plantar reflex was flexor in type on either side. The abdominal reflexes were not obtained. The heart was not enlarged and there were no murmurs, the sounds being forcible and steady. The chest was full of moist sounds, but there was no abnormal dulness. There was no wound of the skull, and no discharge from the ears or nose. The temperature taken in the left axilla was 101.6° F., and in the right axilla 100.4°. A catheter was passed, but no urine was withdrawn. During this operation the patient moved his right arm and leg, but the limbs of the left side remained immobile.

A diagnosis of hæmorrhage into the right side of the brain was made and the usual treatment carried out. Re-examination of the patient on April 26th showed that the left pupil was smaller than the right, and that the left nostril moved during respiration, the right one being apparently immobile. The patient was able to move his left arm and leg, but not to any marked extent. He attempted to speak, but the sounds he made were incoherent. Towards night he took a little milk. On the 27th he was quite unconscious. The temperature as shown by the four-hourly chart was hectic in character, and the patient was bathed in a profuse sweat. It was quite natural to conclude from these details that the original diagnosis of cerebral hæmorrhage was incorrect, and that the patient was really suffering from a purulent affection of the brain, the nature and situation of which were certainly not clear. Later in the day the nurse in charge of the case reported the presence of a foul greenish discharge which "came into the mouth," and necessitated frequent removal. The discharge was very free, and it was impossible to determine by what path it reached the mouth. The chest was carefully examined for the possible presence of an empyema, but without success; and an examination of the