these that I refer; I am dealing with those cases in which the tension is persistently raised. Now, Mahomed's view was that these were the early initial stages of granular kidney, and this I take to be also Goodhart's view.

I do not think Mahomed's work has been estimated at its true value. The term he introduced may have had something to do with this, for the expression, the pre-albuminuric stage, of which he considered high tension to be the chief indication, seemed to assume entirely what he had to prove. His work was based on careful clinical observation, and I believe that he was in the main correct, though possibly he attributed too much to high tension and too little to the thickening of the arteries to which I have referred. Even his own descriptions show that high tension was not the only factor in the pulse, but that the arteries were also thickened.

Sir William Broadbent, differing as he does from Mahomed in theory, yet attaches quite as great significance to the increase of pulse tension. He describes as the ultimate results of this increased tension a variety of symptoms—for example, cardiac failure, general disturbance of health, a sort of cachexia, and even hæmorrhage, which, as he stated, could be truly predicted long before it occurred.

These are the same symptoms as those produced by advanced granular kidney. If the kidneys are not granular in these cases—and of this no proof is given—the arteries must become diseased if hæmorrhage is to occur, for it can hardly be maintained that mere increase of tension can lead to the rupture of healthy vessels. If the vascular change is not atheromatous, nor associated with granular kidney, what is it?

So far as the results to which high tension leads, there is general agreement, but opinions differ as to whether these results are to be referred to granular kidney or not. These differences in theory would remain a matter of opinion still, if there were not some fresh facts by which to elucidate them. These have been obtained by the more exact and careful study of the early eye changes met with in granular kidney.

As I shall show shortly, our knowledge of the early stages of albuminuric retinitis has been greatly extended of recent years. If in many of these doubtful cases of high pulse-tension and thickened artery albuminuric retinitis or the early changes which lead to it are found, and that even in cases in which albumen may not be present in the urine the diagnosis of granular kidney will be surely justified. Considering that albuminuric retinitis is not invariably found even in advanced granular kidney, the fact that it can be discovered in these cases of doubtful nature is a significant fact which throws a light upon many of those other cases in which for the present the doubt must still remain more or less unsolved.

None of the writers mentioned lay any stress upon eye changes in their papers—at any rate, not as a means of diagnosis; but the importance of these early retinal changes was not then so fully recognised as now. Some, at any rate, of these doubtful cases must be referred to the group of granular kidney, and the probability that a much larger number of them may be fairly so referred is so far increased.

It appears to me, therefore, that there is presumptive evidence in favour of regarding these cases of high pulse-tension, as well as those of thickened arteries in the young, as the initial stages of granular kidney which we are in search of.

I do not wish to go beyond my facts or to speak dogmatically upon points which may still be open to question, but this appears to me the conclusion to which the facts point. At any rate, a larger number of these cases can now be proved to be granular kidney than has been hitherto supposed.

REFERENCES.

¹ For these figures I am indebted to Mr. Woodbridge. They will be published *in extenso* in the *St. Barth. Hosp. Reports*. ² This paper will be published *in extenso*. The short abstract now given of it is of the nature of a preliminary report only. ³ BRITISH MEDICAL JOURNAL, August, 1883. ⁴ Bradshaw Lecture, 1885, BRITISH MEDICAL JOURNAL, ii, 327. ⁵ *Lancet*, January 11th, 1879, etc.

PROFESSOR VON ESMARCH has resigned the Chair of Surgery in the University of Kiel, in which he has been succeeded by Professor Helferich, late of Greifswald.

THE late Sir William Gray, shipbuilder, has left the sum of £2,000 to the West Hartlepool Hospital.

SOME UNCERTAINTIES IN THE DIAGNOSIS OF CEREBRAL TUMOUR.

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IN looking over the records of brain surgery one is struck by the very large proportion of cases in which no tumour was found at the operation. Thus in Starr's¹ list of 162 cases no tumour was found in 48; in Knapp's² list of 405 cases none in 92, and Bergmann³ only found a growth in 1 case out of 6. Besides these there are many isolated cases of the same kind scattered through literature. It seems therefore to be important and interesting to inquire into the causes of these deficiencies.

Uncertainties in the diagnosis of brain tumour may be grouped into two large classes. (A.) When no tumour exists, but other diseases are mistaken for it. (B.) When a tumour is present, but (a) the symptoms are ascribed to some other disease; (b) there is a mistake in localisation; (c) there is uncertainty in the nature of the tumour.

A. DISEASES WHICH MAY SIMULATE BRAIN TUMOUR.

1. *Chlorosis*.—Very rarely optic neuritis, generally of rapid onset, is present in this disease; and if it is associated with intense headache and perhaps with hysterical motor or sensory symptoms, a mistake in diagnosis may easily occur.

2. *Uræmia* with intense headache, optic neuritis, convulsions, and coma may simulate brain tumour, especially if the convulsions are unilateral in character. The unilateral form of uræmic convulsion, without cerebral hæmorrhage, is spoken of by Sir William Broadbent in his book on the pulse, and I remember seeing a case of the kind at the Manchester Royal Infirmary (under the care of Dr. Graham Steele) some years ago. The character of the optic neuritis, together with the history and symptoms of Bright's disease, should of course make the diagnosis clear.

3. *Chronic lead poisoning*, if associated with lead encephalopathy, with convulsions, delirium, sometimes acute mania, and coma with optic neuritis and severe headache, may suggest the presence of brain tumour. This idea may be strengthened if hemianæsthesia is present, and particularly if there is any paralysis of the cranial nerves. Buzzard (quoted by Gowers) has described paralysis of the third cranial nerve in lead poisoning; and I have at present under my care a man suffering from chronic plumbism with gout and paralysis of both sixth cranial nerves; a few weeks ago the right nerve was totally paralysed, the left slightly so; this has now almost recovered, so that he has no apparent strabismus, but there is double vision when strain is put on to either of the external recti.

4. *Hysteria* may of course simulate brain tumour, particularly when there is headache, vomiting, convergent strabismus, and various motor and sensory disturbances.

5. So-called *reflex epilepsy*, in which the convulsions are unilateral, is sometimes difficult to distinguish from symptoms due to coarse irritation of the cortex of the opposite side. These convulsions may be caused by irritation of the dura mater due to old injury, by subdural hæmatoma, or by irritation (in dogs) of the inner surface of the exposed dura mater (Franck quoted by Mills⁴). In many of these cases the convulsions are on the same side of the body as the irritation, and are considered to be due not to irritation of the cortex but to irritation of the fifth cranial nerve supplying the dura mater, and especially distributed to its inner surface. Similarly reflex epilepsy, unilateral in character, may be set up by pressure on old scars in the region of the fifth cranial nerve, or by any peripheral irritation of the fifth nerve as from lesions of the ear, eye, nose, teeth, face, or scalp. Injuries of nerves or an isolated fibroma on a nerve in other parts of the body have been known to set up these unilateral reflex epilepsies.

6. Many *anomalous cases* have been reported from time to time in which there were symptoms apparently due to coarse irritation of the brain cortex, but in which the patients have either recovered without operation or after an operation in which nothing was found. Thus Pearce Gould⁵ reported a

case in which there was headache, aphasia, twitchings, facial palsy, and paralysis of the right side of the tongue with slow pulse. A tumour was supposed to be present in the frontal lobe and the skull was trephined; nothing was found; the patient gradually improved, and, with the exception of an occasional epileptic fit, the symptoms all disappeared. Phillips⁶ mentions a case of old injury to the head with severe headache, vomiting, slow pulse, and a tender spot on the head, and slight local twitchings just before the operation. Nothing was found on trephining, but the patient recovered perfectly.

I had the following curious case under my care at the Manchester Workhouse Infirmary:

The patient was a girl, aged 17 years. Her mother stated that the patient had a fit when she was 2 years old, and again when she was 6 years old. After this the fits occurred every year until she was 15; each attack consisted of a series of fits, which lasted about a fortnight. After the last attack she was paralysed on the left side for about three months, gradually recovering. About November 18th, 1897, the fits commenced by twitching of the muscles of the left eye, probably the orbicularis, while the patient was at her work making cigars. She went home and the spasms then spread over the whole of the left side, and she was apparently unconscious until she was brought to the hospital.

On admission, she was suffering from well-marked attacks of left-sided Jacksonian epilepsy, always commencing in the face; the spasms rapidly passed to the left hand and arm. In a day or two the spasms also affected the left leg; each attack consisted of tonic then clonic spasm, followed by transitory paralysis. After recovery the patient told me she could not remember the attacks coming on nor what happened in the interval between the attacks. During the intervals, however, she was apparently conscious and answered questions. The number of fits a day increased rapidly, until the maximum of 275 on December 10th was reached.

The case exactly simulated one of cortical tumour, but as the physical condition of the patient remained good, and as the mother constantly asserted from former experience that the attacks would cease in a fortnight, the question of operation was postponed. On December 11th the number of fits was 125, and the next day she was apparently well, and after a few days she was discharged recovered. In spite of the extraordinary number of the attacks, which were always limited to the left side, the temperature was never raised. She was fed either by the mouth or rectum; she was incontinent as regards both urine and feces throughout the attack. The optic discs were normal, and there was no sign of paralysis of the left side when the fits had entirely ceased, but the left knee-jerk was increased, and there was marked left ankle clonus. During the attacks 30 gr. of chloral, with 45 gr. of potassium bromide were given every four hours, but without the slightest effect. There seemed to my mind no doubt that the attacks were genuine, and not in any way hysterical; there was no sign of hereditary or acquired syphilis. There was a large scar on the head with absence of the hair, but this dated from a ringworm contracted when the girl was 3 years old, and evidently was the remains of tinea kerion.

Another somewhat similar case was that of a lady, aged 60, whom I saw in consultation with Dr. John Scott, of Manchester, on January 25th, 1898.

The patient was said to have had rheumatism some years earlier, with secondary heart disease, but there was no evidence of heart mischief when I examined her. She had had an attack of "fainting" about three years before, and these attacks had recurred from time to time since; in some of these attacks she had been unconscious for about half a minute, and had fallen; in others she had momentarily lost power in the left hand and dropped anything she was holding in it.

About January 10th, 1898, she had severe pain on the top of the head, and this continued more or less day and night, partially preventing sleep until January 2nd. On this day she was feeling well, and was playing cards in the evening. About 1 A.M. on January 23rd she apparently had an attack of general convulsions in bed, "lasting about twenty minutes." Her husband was awakened by the bed shaking. She was then quiet for about an hour, when she had an attack of "spasms" on the left side of the face only. These attacks, which consisted of tonic and clonic spasms, and subsequent transitory paralysis, came on every five or ten minutes day and night, and affected principally the lower facial fibres, and never spread to any other part of the body, and though of course accompanied by loss of power to speak, were not accompanied by any unconsciousness. They were still occurring when I saw her on January 26th. The tongue was covered by a thick plastered coat such as one so often sees in cases of cerebral tumour: there was no optic neuritis, no vomiting, and no tenderness of the skull. As the patient seemed to be getting rapidly exhausted, and as the symptoms so strongly pointed to a gross irritating cortical lesion, an operation was strongly recommended, but the friends would not allow it. Large doses of chloral and bromide were prescribed together with stimulants and careful feeding.

The fits continued with unabated frequency on January 27th and 28th, but began to abate on the evening of the latter day, and she slept well that night, although several fits occurred which did not awaken her. On January 29th she had four attacks, on January 30th two attacks, and then complete recovery set in.

She remained well until the beginning of October, when she had a "fainting" attack in the street and fell unconscious, and the tongue was bitten. A few days afterwards she had a single attack of spasm without unconsciousness on the left side of the face, and a similar attack occurred the next day, and occasionally, she says, she has slight attacks of dizziness.

I saw the patient on November 10th, and she then appeared perfectly well in all respects.

What the exact diagnosis is in these two remarkable cases I am at a loss to say. The second case suffers probably from

occasional attacks of genuine epilepsy, both *grand mal* and *petit mal*, but the facial convulsions without loss of consciousness are a puzzle to me. Undoubtedly, both cases from the persistent localised epileptiform seizures suggested some gross cortical irritation very strongly, but the nature of this irritation seems a mystery.

7. *Syphilis* of the cranial nerves with severe headache, but with no signs of cortical irritation, may simulate the early signs of a brain tumour, and the diagnosis may only be cleared up by time and antisyphilitic remedies. If a syphilitic meningitis with local convulsions and optic neuritis be present, then it may be even more difficult to exclude the possibility of tumour.

8. *Meningitis* or brain *abscess* may be mistaken for tumour, especially if slight optic neuritis is present, if the symptoms have had a slow onset, and if there is no apparent cause for the meningitis or abscess.

9. *Polioencephalitis* may give rise to symptoms which simulate those of pontine tumour.

B. SYMPTOMS DUE TO BRAIN TUMOUR MISTAKEN FOR OTHER DISEASES.

1. *Hysteria* is not infrequently assigned as a cause of the symptoms of tumour, especially when the latter occurs in young girls, and there is no optic neuritis or paralysis of cranial nerves. It should be remembered that in neurotic subjects true hysterical symptoms may be present with or precede symptoms of organic disease such as tumour or disseminated sclerosis, and so give rise to error.

2. *Reflex epilepsy* may be assigned as the cause of the unilateral convulsions of brain tumour, especially if there is an injury or tumour on a nerve. I saw a case of this kind some years ago in the Manchester Royal Infirmary, where a patient with an old injury to and a scar over the left ulnar nerve suffered from left-sided unilateral epilepsy commencing in the left arm; there were no other symptoms for some time, and the case was diagnosed as reflex epilepsy. Some time after the patient died, and a gumma in the right motor cortex was found.

3. *Hydrophobia* has been diagnosed in cases in which there was a history of a comparatively recent dog bite, but in which the symptoms were really due to brain tumour. This occurred in a case related to me by Dr. Bevan Lewis (who made a *post-mortem* examination) in which a small parasitic cyst was found in the neighbourhood of the medulla.

4. Cases of *senile dementia* are frequently admitted into workhouse hospitals. They take no notice of their surroundings, have to be fed, are incontinent, will not answer questions, have no friends to give a history of their illness, and lie curled up in bed. In two such cases, presenting no other symptoms, which occurred at the Manchester Workhouse Infirmary, brain tumours in the frontal lobes were found at the *post-mortem* examination, and had been quite unsuspected during life.

5. *Circulatory changes*, if the symptoms of brain tumour come on suddenly, especially in old persons, may be assigned as the cause of the symptoms. In a case sent to me by Dr. Eatough, of Mossley, near Manchester, a man, aged 56, but looking older, and with no previous symptoms, suddenly became unconscious while sitting in his chair. This condition lasted for about 15 minutes, and his left side, especially the arm, was found to have lost power. This gradually increased. A few days afterwards he had an attack of "trembling" in the left arm and leg, without loss of consciousness, and there was some loss of sensation in the same limbs; the optic discs were normal, and there was no headache or vomiting. Subsequently, however, other attacks of left-sided convulsions with loss of consciousness occurred; there was rapid loss of power on the left side, with optic neuritis, and there was no longer any doubt that a tumour at or near the right motor cortex was present. Death ensued within three months after the commencement of the illness. No *post-mortem* examination could be obtained.

6. If *ear disease* be present, or there is a history of injury, the symptoms of tumour may be ascribed to abscess, especially if there is no optic neuritis; similarly, meningitis may be given as the cause of the symptoms of tumour, particularly if these develop rapidly.

C. UNCERTAINTIES IN THE DIAGNOSIS OF POSITION OF CEREBRAL TUMOUR.

Mistakes in localisation not infrequently occur, and in this connection may be mentioned those in which no tumour was found at the operation, but after death the tumour was found at the point of operation, sometimes even in the resulting hernia cerebri. This failure may have been due to want of deep exploration of the cerebral substance, but in other cases punctures and incisions were made into the brain but without disclosing the tumour which was present at the spot. Such cases are reported by Dinkler,⁷ Köster,⁸ and Schultze.⁹ The reasons for this failure should be given by the surgeon. I can only suggest that at present few individual operators are so sufficiently familiar with the aspect and feel of the healthy brain substance as to be always able to differentiate it for tumour growth, especially if this is a soft diffuse glioma. Sometimes a tumour is present on the side expected but not at the spot diagnosed. In the case of a woman at the Manchester Workhouse Infirmary, there were marked and recurring right-sided epileptiform seizures spreading over the face, arm, and leg, together with other symptoms pointing to tumour of the facial area of the left motor cortex. I referred the case to my surgical colleague, Mr. Joseph Collier, for operation, and he agreed that it was a suitable case, at any rate, for exploratory trephining. Before arrangements could be made, however, the patient had several more fits in rapid succession and died. *Post mortem* a tumour was found in the left second temporo-sphenoidal convolution involving the grey and white matter; the motor cortex appeared normal.

In a case recorded by Schiller,¹⁰ the tumour was supposed to be in the left arm centre. It was not found at the operation, but at the *post-mortem* examination one small tumour was found at the posterior part of the left posterior parietal lobe, and a larger wedge-shaped tumour on the median aspect of the left hemisphere in the quadrate lobe, and a cyst in the left corona radiata.

In one of Hitzig's¹¹ cases, where trephining over the left motor centre was unsuccessful, a tumour was found *post mortem* on the median surface of the left hemisphere, the oldest part being in the centrum ovale, and had invaded the gyrus fornicatus and the corpus callosum, and pressed on, but not invaded, the central convolutions.

Difficulties often arise in distinguishing between tumours of the cerebellum and those of the frontal lobe, as illustrated by a case of Hitzig's,¹² in which a diagnosis of tumour in the right cerebellar hemisphere was made, but *post mortem* a large cystic glioma of the frontal lobe, flattening the second, third, fifth, and sixth nerves, and the right half of the pons was found. As Bruns¹³ and others have pointed out, ataxia and disturbances of equilibrium are found both in frontal and cerebellar tumours, and a distinction is only possible if symptoms due to pressure on neighbouring structures are present.

In other cases tumours in the internal parts of the brain are mistaken for those near the cortex. Thus Bury,¹⁴ in a case where the symptoms were giddiness, left-sided paralysis, headache, nausea, blindness, and nystagmus diagnosed a tumour to be subcortical near the motor area, and an unsuccessful operation was performed. The patient died two months afterwards, and a tuberculous tumour of the optic thalamus was found. In Gibson's¹⁵ case, in which during life there were unilateral spasms and contractions, an operation revealed no tumour, but this was found *post mortem* in the caudate nucleus and anterior part of the internal capsule. Zeller's¹⁶ case complained of pain over the left parietal bone, double vision, loss of memory, double choked disc, difficulty in walking, and spasms of the right arm. A tumour of the anterior lower part of the motor region was diagnosed, but no tumour was found at the operation. After a slight improvement the patient became worse and died, and *post mortem* the tumour was found in the left caudate nucleus, and it sent a process laterally and obliquely upwards into the white matter, but the cortex was not involved.

As I have already mentioned, irritation of the dura mater may cause convulsions on the same side of the body and lead to errors of localisation. In a woman at the Manchester Workhouse Infirmary, suffering from primary carcinoma of the gall bladder with numerous secondary growths, unilateral

convulsions on the right side set in, but at the *post-mortem* examination the only cerebral growth was one limited to the dura mater of the right side.

D. UNCERTAINTY IN THE NATURE OF THE TUMOUR.

Intracranial aneurysm may be diagnosed as a tumour or *vice versa*, and a distinction is often impossible. Apart from an accurate diagnosis of a tuberculous or syphilitic growth, a distinction between the other forms of primary cerebral tumour is often impossible during life. As Gowers says, the diagnosis of the exact nature of the tumour is generally "hardly more than a guess," and "a guess that is almost as likely to be wrong as right." Yet it would be of great benefit if one could distinguish between the diffuse gliomata which are hardly suitable for operation, and the sarcomata which generally have a well-marked limit, whether they spring from bone or membranes, or are within the brain substance, and which can be thus more or less easily "shelled out" at an operation.

CONCLUSION.

It will be seen from the above sketch of a very large subject how difficult it may be for a neurologist to give an accurate opinion on the precise locality and nature of a brain tumour when required to do so by a surgeon before he undertakes an operation; and it can be understood that the difficulties and uncertainties of brain surgery are not so much those of surgical technique or of untoward after-effects, but occur at the outset and with our present knowledge may be well-nigh insurmountable.

REFERENCES.

- ¹ Starr, *Med. Rec.*, February 1st, 1896. ² Knapp, *Epitome, British Medical Journal*, July 16th, 1898. ³ Bergmann, *Neurol. Centralbl.*, 1897, p. 920. ⁴ Mills, *Transact. Congress Americ. Phys. and Surg.*, 1888. ⁵ Pearce Gould, *British Medical Journal*, January 22nd, 1898. ⁶ Phillips, *British Medical Journal*, January 22nd, 1898. ⁷ Dinkler, *Neurol. Centralbl.*, 1897, p. 611. ⁸ Köster, *Neurol. Centralbl.*, 1897, p. 1057. ⁹ Schultze, *Deut. Zeits. f. Nervenheilk.*, 1896. ¹⁰ Schiller, *Neurol. Centralbl.*, 1897, p. 1056. ¹¹ Hitzig, *Theraps. Wochens.*, 1896, Nos. 19, 20. ¹² *Ibid.* ¹³ Bruns, *Neurol. Centralbl.*, 1897, p. 922. ¹⁴ Bury, *British Medical Journal*, November 28th, 1896. ¹⁵ Gibson, *Edin. Med. Journ.*, February 7th, 1896. ¹⁶ Zeller, *Neurol. Centralbl.*, 1896, p. 288.

ON THE REMOVAL OF A FRAGMENT OF STEEL FROM THE RETINA WITH THE ELECTRO-MAGNET, WITH NOTES OF TWO CASES.

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SINCE the year 1882,¹ when I described the electro-magnet devised by me for the extraction of splinters of steel or iron from the interior of the eye,² many instances have been recorded by me³ and others illustrating the use of the electro-magnet in ophthalmic surgery. Its value, and the class of instances in which it is of service, have become generally recognised. The record of isolated cases is hardly, therefore, of interest or value unless there are some special features pertaining to any particular instance. That there are, such, however, in the case related here will at once be recognised. To remove a foreign body from the background of the eye with the retention of perfect sight (§) is a satisfactory event in ophthalmic surgery.

CASE I. Removal of Fragment of Steel from the Retina: Result, Vision §.

History.—On November 5th, 1898, Mr. H. B. M., aged 22, consulted me on account of an injury to his right eye. On October 24th he was driving a wedge with a hammer, and a splinter flew off and struck the right eye. He cannot say whether the fragment was from the wedge or from the hammer. He felt a slight shock to the eye at the time and sought medical aid, and later he was referred to me.

Nature of Injury.—Examination at this visit to me on November 5th disclosed a small linear wound at the periphery of the cornea, at its outer edge, corresponding to about the centre of the palpebral opening. Beneath this corneal scar was a small opening in the iris at the periphery, which at once suggested the probability of the foreign body having penetrated and being located in the globe. Vision = §. The media were clear, but in the fundus towards the periphery on the outer side, just above the median line, a mass of whitish effusion was noticed, in size equal to about 2 diameters of the optic disc. There was only at one point anything resembling the glistening noticeable when a metallic body is embedded in the retina, and this was very trifling. The situation of the effusion corresponded exactly to the line a foreign body passing along the track indicated by the corneal scar and the wound in the iris would take, and no doubt was left in my mind that the fragment of steel was actually embedded in the retina and coated over with this whitish effusion.