Gairdner, of Glasgow, has written to me for fuller particulars of my patient's will making, as he is to be called as a witness in a case next month in which it is to be attempted to reduce a will made by an aphasic whom he attended. Dr. Gairdner says: "I touched briefly upon the subject of the testamentary capacity of the aphasic in my address at the Liverpool meeting of the British Medical Association, but it has always appeared to me one of great difficulty and depending essentially on details."

A CASE OF COMPLETE APHEMIA OF NINE WEEKS' DURATION: SUDDEN RECOVERY THROUGH AN ACCIDENT.¹

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THE patient, C. R., aged 34, a bricklayer, was seen by me on September 2nd, 1896. Complete loss of speech had occurred suddenly on the night before. There was no specific history, no history of previous blow or fall, and none of previous neurotic attacks of any kind. He had always taken a considerable amount of beer, but had been a teetotaler for seven months, and until a fortnight before the loss of speech occurred, when he took to his old custom. He had always been in good health, with the exception of occasional rheumatism and an illness seven years ago, which was stated to be inflammation of the lungs and kidneys.

His present attack came on quite suddenly on his way home from the public-house, and just after having left a friend with whom he was conversing as usual. As he left his friend to walk to his house—a distance of about 50 yards—he experienced a feeling of faintness and swimming in the head, with weakness in the left leg, which rendered him unable to walk steadily. He asserted that he was quite sober. This his wife corroborated.

On arriving home he discovered that he was unable to talk, and he had since been unable to articulate a single word, though able to express himself freely by aid of slate and pencil without the slightest hesitation or mistake.

The patient, a well-built dark-complexioned man, with a narrow retreating forehead, looked somewhat dazed, pale-faced, with anxious, emotional expression, and profuse perspiration of head and face, which increased on his being questioned, when he showed an amount of distressed effort. His temperature was slightly increased. The tongue was furred and moist, and on attempt at protrusion impinged on the teeth at the right angle of the mouth. There was difficulty of deglutition. The throat was relaxed and the uvula deflected to the right side.

He complained of occasional attacks of faintness and swimming in the head, of the same kind as he experienced at the onset of the attack. The apex beat was outside the nipple line, and the impulse weak. The first sound was indistinct, the second accentuated in comparison. There was a systolic murmur in the aortic area transmitted towards the neck. The urine was healthy. He complained of pricking or tingling sensation in the left leg as far up as the knee. There was no alteration in sensation or conduction. The pupils were equal and reacted well to light. There was general paresis of left leg and arm—of the leg more than the arm—and that mostly in the upper part.

His intelligence was good; he had no delusions, torpor, or coma, or much slowness of mental processes; attention and memory were good. Sleeplessness with restlessness had been troublesome since the attack. This he attributed to faintness and swimming in the head. No involuntary interjections were uttered and attempts at speech were abortive. He answered simple questions requiring affirmation and negation by nodding and shaking the head. He understood everything said to him, and promptly and intelligently answered all questions put to him by aid of slate and pencil, and without confusion of words or their sequence. He wrote the date of the month and the day of the week of the seizure, and gave dates and character of previous illnesses. He under-

stood and appreciated singing. He could read and understand what he read. Sight was good, and he recognised common objects and pictures of common objects, and named them correctly. He could copy words and figures, and wrote spontaneously and from dictation.

On September 3rd he could protrude his tongue with slight difficulty.

On September 12th he muttered "Yah! yah!" at the breakfast table without apparent application to anything. He tried to talk to a child, but unsuccessfully. He complained of weakness in the arm after getting out of bed for the first time.

His general condition improved, and he resumed his work on September 16th. He had a slight attack of lumbago and sciatica on October 1st, which soon passed away. Sleeplessness resulted if he worked in a stooping position. The loss of speech remained as before.

His general condition continued to improve until November 2nd, when, while building an oven at a height of 6 feet from the ground, he felt giddy, lost his balance, and fell backwards to the ground, landing on his head. He was stunned, moaned, and then shouted to his friend by name. His nose bled considerably, which it does occasionally and without apparent cause.

Next morning he informed me that he had been able to talk since, and spoke quite distinctly, but somewhat more slowly than usual. There was tenderness on pressure over the left occipital and parietal regions, with some effusion.

The exact pathology in this case is difficult. Hyperæmia, hæmorrhage, thrombosis, and embolus will suggest themselves, as also monospasm and hysteria.

There was inability to protrude the tongue, which was deviated to the right side. The left arm and left leg were paresed—the arm slightly and leg mostly in the upper part. The complete loss of speech are suggestive of a lesion in the left inferior frontal convolution or in that region, the patient being righthanded, although his work might help to make him partly ambidextrous. Can hyperæmia be so localised? There was no disease of the kidneys or of blood vessels which would tend to exclude hæmorrhage and thrombosis, and the lesion was probably not central. The heart was not satisfactory, and the multiple character of the lesions suggests emboli. If embolic, were both sides of brain affected? As to the sudden recovery through an accident, can it be owing to the sudden cessation of an enduring monospasm as Bastian would suggest? or to the resolution of the embolus or emboli? The leg on November 24th showed distinct weakness. As to the locality of the lesion as far as the total loss of speech in a case of aphemia such as this is concerned, Bastian suggests that the "defect is capable of being produced by damage to efferent internuncial fibres in any part of the course from the left glosso-kinæsthetic centre to the articulatory centre in the bulb," and Gowers, that the lesion is "probably so placed as to interrupt the fibres that go to the motor tract (internal capsule), and those to the corpus callosum, but has not destroyed the speech centre itself or the connection between it and the head centre."

TWO CASES IN WHICH THE SIGNS OF THE PRESENCE OF AN INTRACRANIAL TUMOUR WERE FOLLOWED BY RECOVERY.

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WHEN patients present themselves with signs of progressive organic intracranial disease, the outlook is, in the great majority of cases, of the most gloomy character. Sometimes, however, even when such signs are well-marked, an intermission or remission of the symptoms takes place, and, much more rarely still, a more or less complete recovery, and this in cases in which the character of the symptoms present seems to leave little room for doubt as to the correctness of the diagnosis.

With the exception of intracranial syphilitic disease, which is more amenable to treatment, and in which more or less complete recovery is frequently seen, the uniformly

¹ Read before the Staffordshire Branch of the British Medical Association at Stoke-on-Trent.

grave prognosis that attaches to cases of organic brain disease renders instances of recovery of great interest from the clinical point of view. That such recovery may occasionally take place where the symptoms indicated tuberculous meningitis has been shown by the accounts of published cases.¹ Sometimes cases are also met with, and are perhaps of especial interest, in which, in the absence of syphilis, the symptoms point to the presence of an intracranial tumour and yet the patients recover. Such seemed to be the condition in the two cases which form the subject of this paper. In each the prospect of recovery during the early part of the illness seemed, to say the least, extremely doubtful, and the favourable termination was all the more satisfactory.

CASE I.—L. E., aged 17, a cigar maker, was admitted into the Bristol General Hospital on January 28th, 1893, complaining of frontal headache, attacks of vomiting, and great failure of sight since the previous September. Her father died of pulmonary phthisis, and her mother was in delicate health; otherwise there was nothing of importance in the family history. The patient, although never strong, had never had any bad illness. The present illness began in the first week of September, 1892, with several attacks of vomiting, and severe pain in the frontal region of the head, which was intensified on lying down. For the eleven weeks following this attack the patient was laid up at home. She was frequently sick—as a rule about every other day, generally at 5 A.M., and two or three times later in the day. She stated that at this time the headache was practically constant, without intermission. She also suffered from dimness of vision, with sudden attacks of total blindness, which lasted for two or three minutes and then passed off. These attacks had occurred about three times a day ever since the middle of September; she was sometimes giddy in them, and once fell down, but there was never any loss of consciousness, convulsions, or muscular twitchings. There was some failure of memory. She returned to work at irregular intervals during the latter part of November, but then grew worse. There was no history of diplopia.

On admission she looked fairly well; she was rather pale, but not distinctly anæmic. The thoracic and abdominal organs were healthy. There was no sign of hereditary syphilis.

Nervous System.—No tenderness to percussion anywhere over the skull. No paralysis of the cranial nerves. No paralysis, wasting, or rigidity of any muscles. No affection of sensation to touch, pain, or temperature. Superficial and deep reflexes normal. Speech and swallowing normal. Special senses, except vision, normal. Gait natural. There was very intense double optic neuritis, most advanced in the left eye, which showed numerous specks of cholesterol at the edge of the optic disc; there were a few linear hæmorrhages around the right disc. The swelling of the optic discs was estimated at from 5 to 6 D.

She could move her limbs well, and there was no paralysis or wasting of any muscles. The feet presented the condition of pes cavus, which dated from infancy. Urine normal, contained no sugar or albumen. Catamenia absent for six months. Pulse 72, regular. Temperature 98°; it remained subnormal during her stay in the hospital. Bowels regular. Tongue clean and moist. There was no history or sign of aural affection. She suffered from very severe dull aching pain in the frontal region of the head, not quite continuous, though occasionally lasting for hours. She was unable to lie down in bed on account of the headache being aggravated and accompanied by giddiness in that posture.

During the first month of her stay in the hospital she complained almost constantly of intense headache, and of nausea; she vomited on several occasions, the vomiting being of the cerebral type, and bearing no relation to the ingestion of food; the brief attacks of amaurosis came two or three times daily, and there was progressive deterioration of sight. The treatment adopted was the administration of cod-liver oil and of potassium iodide in increasing doses, with syrup. ferri iodidi. Antipyrin generally relieved the headache for the time, but on a few occasions hypodermic injections of morphia were necessary. Nausea, vomiting, and headache began first to improve about March 18th; she then had a slight eruption of labial herpes. The attacks of vomiting first ceased, then the giddiness passed off, and the headache began to lessen. The optic neuritis gradually subsided, only to be followed by atrophy of the disc. The temperature remained at 98° during the whole of her stay in the hospital, the pulse at about 72 to 76; there were no other symptoms, and she never at any time had any pains in the back of the neck or retraction of the head. She left the hospital in May, as she seemed well in herself, and the headaches and vomiting had ceased, and no further symptoms appeared. Vision was, owing to the consecutive atrophy of the discs, unfortunately reduced to very little more than perception of light. When seen some months later her condition was unchanged.

CASE II.—A mason, H. K., aged 23, was admitted on November 8th, 1893, complaining of headache, weakness, giddiness, and dimness of vision. He stated that his father had died of pulmonary phthisis, and that a brother and sister had died young—cause unknown. He had never had any previous illness, and was certain that he could not have contracted syphilis; he had occasionally suffered from headaches. He had never suffered from deafness or discharge from the ears, and had always been temperate. About two months before admission he had fallen out of a cart on to his head, and for two or three minutes after getting up he felt dazed and could not see, but was able to resume his work shortly afterwards.

The present illness began five weeks before admission. One day whilst at work he felt giddy and stupid, so that he could not go on, and was obliged to go home. During the next week he managed to continue at work, but his sight failed so rapidly that he could hardly see at all. A week later he had an attack of shivering (? tremor), together with violent headache and a sense of fullness in the head which made him feel very stupid. From this time his sight grew rapidly worse, so that he could not see persons with whom he was talking, and had to be led about in the streets; he had attacks of vomiting, not related to food, two or three times in each week. He also suffered from muscular twitchings and

cramps in the legs, from pains in the back of the head and left angle of the jaw, from noises in the ears, and from giddiness.

On admission he looked healthy, but his face was decidedly flushed. On his first morning in the hospital he felt cold and shivered for about half an hour, without, however, any rise of temperature. The headache was generally in the frontal, sometimes in the occipital region.

On examination, the heart's apex was in the sixth space, and a faint blowing systolic murmur was heard over the pulmonary area; otherwise the thoracic and abdominal organs were normal. The urine contained a deposit of oxalate crystals but no albumen or sugar. The pulse-rate was 108, respiration 24, temperature 97.6° F. He lay on his back, was listless, yawned repeatedly, and was extremely drowsy. His gait was feeble, and he was giddy when he stood up. There was no paralysis of any movement, although he was generally weak, and no muscular rigidity or wasting. Sensation to pain, touch, and temperature normal. The superficial reflexes and knee-jerks were normal; ankle clonus was not present; the cranial nerves were unaffected; the special senses other than sight were normal; vision was very defective; there was very intense optic neuritis with much swelling of the discs, most advanced in the left eye; the pupils were dilated, and reacted, though sluggishly, to light, accommodation, and sensory stimulation of the skin of the neck.

The night after admission he vomited several times, and for several days the headache was intense. He was very drowsy the whole time, being with difficulty roused; he was occasionally irritable. Ten days after admission his sight had become so bad that he could no longer feed himself.

On November 22nd his condition was much the same; the optic neuritis had increased, and there were numerous small hæmorrhages around the discs. He was now quite unable to stand, chiefly from weakness but partly from giddiness, which was especially bad in the early morning. There had been no recurrence of vomiting. Both pupils were dilated, and the left was persistently larger than the right.

On November 28th the headache began gradually to improve, and he felt a little better in himself. Steady though slow improvement as regards general symptoms took place. On December 12th he was delirious during the night without any obvious cause or rise of temperature. Soon after this he began to get up for a little time each day.

By January 11th, 1894, he was able to be up and about the ward; there had been no recurrence of the vomiting, and the headaches only recurred occasionally and were slight. Vision was reduced to bare perception of light; the optic neuritis was subsiding, leaving the disc atrophied. He suffered from clonic spasm of the right hand for a short time one day, which did not recur; otherwise there were no fresh symptoms. As his general health was now good and his condition appeared to be stationary, he left the hospital shortly afterwards. The treatment adopted was rest, application of ice to the head, the administration at first of mercury and full doses of potassium iodide, followed later by cod-liver oil and tonics. He was seen a few months later, and again in November, 1896, and it was found that he remained well, but was totally blind from atrophy of the optic discs. There had been no recurrence of any of the cerebral symptoms.

When these patients were admitted, and during the first part of their stay in the hospital, although the symptoms were general and no localising symptoms were present, the most probable diagnosis seemed to be that of an intracranial tumour. In each case the general signs of tumour—that is, optic neuritis, intense headache, vomiting of cerebral type, and vertigo—were well marked, and in the second case there was inability to stand. From the point of view of the symptoms themselves, the most significant was the character of the optic neuritis, which was of that very intense kind that is most frequently found in association with tumour.

The next most likely cause of the illness was meningitis, and there was, further, the possibility of the optic neuritis being of the idiopathic variety, a primary neuritis sometimes occurring for which no cause can be discovered. Against this latter was the very pronounced nature of the general head symptoms, and possibly the great intensity of the neuritis. With regard to meningitis, the absence of any past or present ear disease and of any other cranial affection that commonly causes meningitis, and the absence of all the characteristic symptoms—for example, retraction of the head, affection of cranial nerves, muscular rigidity, convulsions, delirium, and the presence of a subnormal temperature throughout the illness in both cases—would seem to be sufficient to negative this affection. The other causes of optic neuritis, including syphilis, anæmia, lead poisoning, and Bright's disease, could be satisfactorily and completely excluded.

On the whole, then, the presence of an intracranial tumour seems the most probable diagnosis even in the absence of localising symptoms, and the character of the optic neuritis offers further confirmation of this view of the cases. In the second case the appearance of symptoms some time after a fall on the head suggests that the injury might have started into activity some previously existing but quiescent mischief, and is certainly suggestive when the frequency of a history of a fall or blow on the head in cases of intracranial tumour is borne in mind.

With regard to the nature of the growth it is well known that under treatment tuberculous masses may occasionally cease to grow, become inert, and are encapsulated. This pro-

bably occurs more frequently in tuberculous than in any other form of intracranial tumour, syphilitic growths of course excepted.

In each of my cases the father of the patient had died from pulmonary phthisis, and in the second case there had probably been other deaths from phthisis in his family. Without going further one may suggest that it is at least probable that the cause of the illness in these patients was a tuberculous mass in some indifferent part of the brain, which ceased to grow or became encapsuled. The duration of the illness was about eight months in the first and four months in the second case.

Dr. Gowers² gives a short account of two cases, and says that he "has seen a considerable number of other cases with well-marked symptoms of a tumour, probably tuberculous, which passed away permanently under treatment," and a most instructive case in point, in which the accuracy of the diagnosis was confirmed at the necropsy made four years afterwards is reported by Dr. R. T. Williamson.³ The patient, a man aged 27, had suffered from Jacksonian epilepsy (left-sided), with impaired vision. He got better, the fits ceased, vision improved, and he returned to work, with a slight loss of power on the left side. Four years later he suffered from ataxia, headache, vomiting, and double optic neuritis, with tuberculous lung disease, and the necropsy disclosed an old encapsulated tuberculous nodule in the right cerebral hemisphere, with a recent tuberculous mass in the cerebellum.

There is one point with regard to the prognosis in such cases after the period of symptoms of active disease has passed away. Of course, in cases of tuberculous diseases there is always a liability, as is illustrated in Dr. Williamson's case, at some subsequent period to a recrudescence of the disease, but apart from this there seems to be also a danger of sudden death after the symptoms of the former illness have disappeared. Dr. Gowers mentions that in one of his cases referred to above sudden death occurred after there had been no recurrence of symptoms for three years. The same fact was recently exemplified in a patient who had been under my care for cerebral syphilis, due, as the necropsy showed, to the presence of a large syphilitic growth on the under surface of and involving the substance of the left frontal lobe. There was no other lesion. This man suffered at first from attacks of loss of consciousness, later from attacks of Jacksonian epilepsy (right-sided), with right hemiparesis; from headache, vomiting, vertigo, spasm of the left orbicularis muscle, fixation of the left pupil, and double optic neuritis ending in blindness. He became unable to stand or walk, was at first mentally dull and drowsy, and then for two or three weeks very irritable, noisy, violent, and at times quite maniacal, and lost all idea of his surroundings. After a stay in the hospital of three months' duration he recovered from all his symptoms except that he was left blind from optic atrophy, and with slight loss of power on the right side. In all other respects he left the hospital apparently well, and able to walk about nearly as well as ever except for the blindness. After he had been at home for a month, and was to all appearance in good health and perfectly free from all his former symptoms, he one day fell down dead quite suddenly whilst walking in the street.

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A CASE OF CEREBRAL ABSCESS, PRESENTING SOME UNUSUAL FEATURES.

By MORLEY WILLIS, F.R.C.S.ENG.

I AM indebted to Mr. T. Chalmers Norton for permission to publish this case, which occurred in his practice at the Hospital for Sick Children and Women, Bristol, at the time I was house-surgeon at that institution.

E. L., aged 8 years, was admitted on January 28th, 1896. On January 24th she complained of headache, was sick once, and her friends for the first time noticed a discharge of matter from the left ear. They attributed the headache to a fall a few days previously, though the only evident injury due to the fall was a cut lip. On January 25th and 26th she was sick several

times, and on the last-named day her manner altered, and she was very excited and unruly.

For the greater part of January 26th and the whole of January 27th she was not conscious, and recognised no one. Her head and neck were markedly retracted on admission, and had been so since January 26th. When not disturbed she lay quiet and still, and curled up, that is, flexed generally, except at the neck, which was extended. She much objected to being disturbed, turning away, and crying petulantly on being roused. The temperature was 101° F. There were sordes on teeth and lips, and the tongue was dry. Though not profoundly unconscious it was obvious that she did not recognise her mother. There was no purulent or other discharge from either ear, nor any offensive odour from the meatus. There was no paralysis of any sort at this time.

The notes for the next two days show some improvement in her mental condition, and she gave intelligible replies to questions relating to feeding, and asked for bread and butter. Coincidentally with this improvement the temperature fell, until on the morning of February 1st, five days after admission, it was 98° F. For those five days it had been at 100° to 101° F., the latter on most evenings.

On February 7th a view of the optic discs was obtained, and they appeared slightly redder than usual. From this time she got worse, vomiting frequently, occasionally screaming, and appeared to indicate that she had pain in her head, although at this time her expressions were not very intelligible.

On February 16th (20 days after admission) some weakness of the right arm and hand was observed; and the wrinkles and folds of the right side of the face were less marked than those of the left, especially when she cried. Her speech had become more impaired, and she could not make herself understood.

These symptoms all became more and more marked until, on February 22nd, there was complete facial paralysis of the right side, and greatly impaired power in the right arm and hand. The tongue on being protruded was strongly deviated to the right of the middle line, that is, towards the paralysed side. With this there were now distinct evidences of optic neuritis.

Taken together, these symptoms appeared conclusive evidence of a lesion affecting Broca's convolution and those parts of the cortex lying in the immediate neighbourhood of the lower end of the Rolandic fissure. The history of the case indicated as the cause an abscess in the temporo-sphenoidal lobe of the left side, which, gradually enlarging, pressed upon the structures named.

It was, therefore, decided to explore the brain in this situation, and on February 23rd, the patient having been anaesthetised with chloroform, a disc of bone $\frac{1}{2}$ in. in diameter was removed with a trephine, the pin of the trephine being applied to a spot corresponding to the lower end of the fissure of Rolando. The dura mater was incised and turned up, and then, carefully avoiding some rather large veins on the surface of the brain, a silver trocar and cannula were passed into the substance of the brain in a direction slightly downwards and backwards from the centre of the area of cortex exposed by the removal of the disc of bone. It had been observed as soon as the disc of bone had been removed that there was an increase in the intracranial pressure; when the dura mater was incised the brain bulged somewhat into the hole in the skull, and pulsation in the brain itself was hardly at all visible. On removing the trocar pus welled up through the cannula.

The cannula itself was left *in situ* for drainage. The wound was sewn up entirely, a hole having been made in the flap to permit the passage of the cannula. A small superficial drainage tube was placed in the posterior inferior angle of the wound, which was then dressed antiseptically, and the patient was removed to bed.

On the day following the operation there was an obvious diminution in the signs of compression, the patient being conscious, and appearing to understand what was said to her. On the second day after the operation the discharge of pus ceased, and as it was thought that the cannula might have become blocked, a little chloroform was given, and it was removed and reinserted in the same direction, but, on removing the trocar, not pus, but a stream of pure blood, shot up at