now weak-minded. She cannot read or be trusted with the most simple errand. Before the convulsions, however, she is said to have been bright and intelligent, and could say several words. It may be of interest to add that the mother attributed the convulsions to the falling of the child into a dirty stream two or three weeks before their onset. The child was nearly drowned, and after being pulled out vomited much dirty water. In this case it is obvious that blockage of the right external or common iliac vein is present, which may with reason be attributed to thrombosis occurring at the time the enlarged abdominal vein was first noticed. The evidence of thrombosis in one vein suggests that the associated cerebral symptoms were due to thrombosis within the cranial cavity. This thrombosis may have been of infective nature consequent upon the entrance of micro-organisms contained in swallowed dirty water into the blood.]

REFERENCES.

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SYPHILITIC MENINGO-MYELITIS : ERYSIPELAS : RECOVERY.

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THE following case presents several points of interest :

History.—A woman, aged 23, had a miscarriage a few months after her marriage three years before the onset of the paralysis. Her first and second child died 7 and 2 months respectively after birth. She herself had been under treatment at the Leeds General Infirmary for syphilis, and her husband was known to be syphilitic. Six weeks before the commencement of the paraplegia she complained of pain and cramp in the left, and later in the right leg. This was followed by girdle pain. On February 24th, 1900, her right leg became weak on her way up to the dispensary, and she could only continue her journey with difficulty and with assistance. When she woke up on the morning of February 26th she had lost power in the left leg, and the paraplegia soon became complete. There was loss of sensation in the legs, and all the reflexes were slightly exaggerated. There was incontinence of urine, and a large sacral bed-sore was noticed on March 17th.

Condition on Admission.—The patient was admitted into the Leeds Infirmary under my care on March 17th. There was at this time an almost complete paraplegia with considerable rigidity, but no wasting. The reflexes were all increased, but no ankle clonus could be obtained. There was incontinence of urine and to a less extent of feces. The urine was alkaline, and contained a small quantity of pus. Tactile sensation was impaired. There was the remains of a syphilitic rash on the chest, as well as scars about the mouth.

After-History.—On March 24th an attack of facial erysipelas supervened. Two injections of antistreptococcus serum were given. The temperature became normal on March 30th, but there was a further slight rise of short duration on April 6th. On April 3rd it was noticed that she could move her toes, and the power in the legs began to return rather rapidly. Unfortunately, owing to the cleaning of the wards she had to be sent home on April 14th, and she was again attended from the dispensary. On June 12th she walked up to the out-patient room with the help of a stick. The knee-jerks were exaggerated, especially on the left side. All forms of sensation appeared to be normal. The bladder had recovered completely. On June 28th she walked up without a stick, and on July 19th the recovery appeared to be quite complete. On April 11th, 1901, she was carefully examined. She was quite well, with the exception of a slight weakness of the anal sphincter; thus she said that she could not hold her motions for more than a minute. The sphincter did not grasp the finger at all strongly.

The diagnosis of an incomplete myelitis involving the lumbar enlargement and part of the lower dorsal cord was based on the spastic paralysis, loss of sensation, paralysis of the sphincters, and bed-sore. The presence of pains and cramps in the legs showed that the meninges were also involved. The meningo-myelitis was believed to be syphilitic for the following reasons: (1) There was not only undoubted evidence of past syphilis, but the patient still presented manifestations of active syphilis when the paralysis supervened. (2) The presence of paralysis combined with rigidity and exaggeration of the reflexes, is known to occur in spinal syphilis; and (3) the absolute absence of any other effective cause. There was apparently some variation in the severity of the symptoms during the early stage of the disease, and this also has been associated with spinal syphilis by some

writers. It was imagined that the lesions consisted of an inflammation of the pia-arachnoid, most probably of a genuinely syphilitic nature, and that the disease had spread into the cord which it affected in a patchy fashion, and not uniformly.

A point of peculiar interest in the case was the attack of erysipelas, which was followed, at least in point of time, by improvement in the paralytic symptoms. There is nothing preposterous in assuming a direct relationship between the erysipelas and the recovery, as one infection has been known both experimentally and clinically to antagonise another and independent infection. By some curious oversight the patient received no antisyphilitic treatment between the onset of the paralysis and June 28th. There is reason to believe, though unfortunately the out-patient notes cannot be found, that the patient was under antisyphilitic treatment during at least a part of the twelve months preceding the paralysis.

The importance of the recognition of cases of spinal syphilis is twofold, namely, on account of its relation to (a) prognosis and (b) treatment, although the above-related case hardly exemplifies the latter statement. Williamson (of Manchester) in his interesting paper on spinal syphilis¹ mentions 32 cases of various forms of the disease. Of the 32 cases 10 recovered and 9 died, the remaining 13 passing from observation without any great change in their condition. Five out of 6 cases of acute syphilitic paraplegia died. While insisting on the importance of antisyphilitic treatment Dr. Williamson makes the very guarded statement that the prognosis of spinal syphilis is on the whole better than that of other affections of the spinal cord. Obviously the extent to which the spinal cord is involved in these cases is of the first importance in the prognosis.

Unless the actual lesions present in the membranes and cord are syphilitic (gummatous) in character or perhaps due to syphilitic vascular disease, little hope of any good can be expected of antisyphilitic treatment. As, however, we are mostly not in a position to say whether these lesions are genuinely syphilitic or not, a course of antisyphilitic treatment consisting in mercurial inunction and potassium iodide should always be had recourse to unless it is certain that the patient has recently been efficiently treated in this way. If no result is obtained in a few weeks such treatment should be abandoned, at least for some time, as it may possibly do injury instead of good.

I have to thank Mr. H. R. Cross, one of the resident medical officers to the dispensary, and Mr. Hirst, the clinical clerk to the case, for additional notes of the case.

REFERENCE.

- ¹ *Edinburgh Medical Journal*, October, 1900.

CASE OF CEREBRAL TUMOUR AT THE PARIETO-OCCIPITAL FISSURE.

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THE following record of a case of cerebral tumour presents several points of interest with respect to the localisation of cerebral lesions.

G. P., aged 43, was admitted to the Ancoats Hospital on December 31st, 1900, complaining of headache and failure of sight.

History.—His illness had commenced nineteen weeks previously with troublesome headache and sleeplessness. At that time vision was not affected. After the headache had continued for two or three weeks he began to be troubled with slight twitches in the left leg; these continued from time to time all the morning and afternoon, and prevented him from walking about. The same day, about 5 P.M., the twitches in the left leg became suddenly more severe and rapid, and developed into a true clonic convulsion. The twitches spread from the left leg to the left arm and left side of the face. The face was drawn to the left, and the tongue bitten. After a few minutes the patient lost consciousness, and finally the right side of the body was convulsed also, but not so markedly as the left. The attack was therefore a true unilateral convulsion (Jacksonian epilepsy) beginning in the left leg. The duration of the fit was about twenty minutes. The patient fell asleep afterwards, and he remained in a semi-conscious state for four days. About thirty hours after the first convulsion there was a second of a similar nature, and again about twenty hours later there was a third. In all three fits the spasm commenced in the left leg; it then spread to the left arm and left side of the face, and remained unilateral for some time before the right side was affected. The patient recovered consciousness completely at the end of four days. There was no paralysis. The sight began to fail

about sixteen weeks before his admission into hospital, and vomiting occurred occasionally. There had never been any paralysis of the limbs. There was no history of injury to the head, of syphilis, or of any antecedent to which the illness could be attributed.

State on Admission.—The patient could walk, and perform all the movements of the limbs quite well. The knee-jerks and plantar reflexes were present; ankle clonus was absent. There was no anaesthesia. The bladder and rectum were not affected. The patient complained much of headache, which was most severe in the right frontal region. There was a tender spot on percussion just above and behind the right frontal eminence. There was no affection of the facial, ocular, or tongue muscles. The senses of smell and hearing were not affected. There was no anaesthesia in the distribution of the fifth nerve. The pupils were equal, and reacted to light and accommodation. The vision was impaired, but he could count fingers and read large print with each eye. Ophthalmoscopic examination revealed intense double optic neuritis, with marked swelling of the discs. The fields of vision were tested with the finger, but no hemianopsia could be detected. There were no mental symptoms. Examination of the heart, lungs, and abdomen reveal nothing abnormal. The urine had a specific gravity of 1025; albumen and sugar were absent.

Progress.—The patient's condition gradually became worse; vomiting occurred occasionally. The fields of vision were examined a second time, but no hemianopsia could be detected. The failure of sight increased, owing to the optic neuritis, and further examination of the visual fields was impossible. Soon the patient became quite blind. The changes in the optic discs were intense, and numerous hemorrhages were scattered over the retina. The headache became very severe and continued to be most intense in the right frontal and temporal regions. There was marked tenderness just behind and above the right frontal eminence; this persisted until the operation. When the skull was auscultated with a stethoscope in various regions, whilst the patient was whispering, the whispering sound was best heard at the region of tenderness just behind and above the frontal eminence. At this point the whispering sound was better heard on the right side than on the left. The patient became more and more apathetic, and in a few weeks after admission to the hospital he appeared to be in a semi-conscious condition. He remained quiet all day long, he never spoke to anyone, and always appeared to be asleep, but if spoken to he would answer quite clearly and describe his symptoms in the most intelligent manner. On January 10th it was found that he was not able to dorsiflex the left foot so well as the right; all the other movements of the legs could be performed. The knee-jerks were present. No paresis of the arms, facial, ocular, or tongue muscles could be detected. There was no impairment of sensation in the face, arms, or legs. There was no affection of the muscles of the trunk. As the headache in the frontal region was so intense and prevented him from sleeping at night, the patient wished to have some operative treatment.

Operation.—On February 13th my colleague, Dr. Tytler, trephined the skull at the seat of the greatest pain and at the region where there was tenderness on percussion, that is, at a point just above and behind the right frontal eminence. He removed a small circle of bone, but did not open the dura mater. A fine hypodermic exploring needle was pushed through the dura mater into the brain substance of the frontal region, but no pus or fluid was detected. No bad symptoms followed the trephining. The temperature remained normal and the wound healed by first intention. The trephining relieved the headache markedly, and after the operation there was very little headache. At the end of February the patient became more dull mentally, but there was no delirium. Finally coma developed and death occurred on March 13th, 1901.

Fig. 1.—Convex surface of right cerebral hemisphere. Dotted area = position of tumour. P.O.F. = parieto-occipital fissure.

Necropsy (head only).—The dura mater appeared normal on its outer surface. The surface of the brain was rather pale, but otherwise normal, except at the region of the parieto-occipital fissure of the right cerebral hemisphere. At this region there was a tumour growth which was slightly adherent to the dura mater. The growth was about 1 inch in the antero-posterior diameter, and about $\frac{1}{2}$ inch transversely (on the surface of the cerebral cortex). It extended both on the median and convex surfaces of the hemisphere; it appeared wedged into the right parieto-occipital fissure, and invaded the brain substance on each side of the fissure; but the growth extended more into the occipital than into the parietal lobe. On the convex surface of the occipital lobe it invaded the superior (or first) occipital convolution just behind the parieto-occipital fissure; on the median side it occupied the upper anterior angle of the cuneus lobe. In front of the parieto-occipital fissure it invaded, for about half an inch, the most posterior part of the superior parietal lobule on the convex surface of the cerebral hemisphere, and, for the same distance, the most posterior part of the quadrate lobule (precuneus) on the median surface of the hemisphere. (See diagrams.) On section the growth was seen to be roughly spherical, and its diameter was about 1 inch. The outer part of the growth was greyish-red, the centre slightly yellowish and somewhat gelatinous, and crossed by paler tracts of firmer tissue. Microscopically the growth was a mixed-celled sarcoma. In the centre there had been apparently some old blood extravasation. The frontal

lobes, the other parts of the parietal and occipital lobes, and all other parts of the brain were unaffected by tumour growth, and were quite normal.

Fig. 2.—Median surface of right cerebral hemisphere. Dotted area = position of tumour. P.O.F. = parieto-occipital fissure. C = cuneus. Q = quadrate lobule.

The symptoms of this case pointed clearly to a cerebral tumour. From the history of the three fits, each commencing in the left leg, and from the fact that the leg did not become paralysed, it appeared probable that the growth was situated in the right cerebral cortex close to, but not destroying, the leg centre (that is, just in front of, or just behind the leg centre). At the early period no hemianopsia could be detected, but soon the blindness, due to the optic neuritis, rendered further examination of the fields of vision impossible; so that no conclusions could be drawn with reference to the effect of the growth on the visual fields. The only mental symptoms were dulness and delay in commencing to answer questions.

In the absence of any sign indicating whether the growth was in front of, or behind the leg centre, the frontal headache, and the tenderness on percussion just behind and above the frontal eminence appeared to be in favour of the former localisation. But as the locality of headache and tenderness is so often unreliable as regards the position of a lesion, the localisation of the growth, during life, did not appear sufficiently definite to justify any attempt to remove it. Hence, though the skull was trephined, the dura mater was not divided. The removal of the bone was followed by very marked relief of the headache. The patient had been blind for several weeks before the operation, and, of course, no return of vision followed the trephining.

The chief interest of the case was the locality of the tumour and the three unilateral convulsions beginning in the left leg. The case shows that a tumour at the most anterior part of the occipital lobe and at the parieto-occipital fissure, may produce convulsions commencing in the leg on the opposite side, through the extension of the growth into the most posterior part of the superior parietal and quadrate lobules for about half-an-inch. This would coincide with the view that the leg centre extends backwards both on the convex and median surfaces of the cortex (in the superior parietal and quadrate lobules) almost up to the parieto-occipital fissure. The ascending frontal and ascending parietal convolutions and the paracentral lobule were unaffected. It was only the most posterior portions (half-an-inch) of the superior parietal and quadrate lobules that were invaded by the growth. Another point of interest was the impaired power of dorsiflexion of the left foot, noted a short time before death. This was due, no doubt, to the involvement of the most posterior part of the superior parietal and quadrate lobules.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

THE DURATION OF DREAMS.

As the opportunity for comparing the duration of a dream with the rate of thought of the dreamer is rare, the following instance may be of interest to some.

Some little time ago I had occasion to administer nitrous oxide gas to a sailor for the purpose of tooth extraction. He was a petty officer in the merchant service, and an intelligent man. He took the anaesthetic well, the tooth was duly extracted, and he recovered consciousness with the usual rapidity. However, just at the moment of waking up, he