Progress.—On August 15th, at 7.45 a.m., he complained of sharp pain over the left half of the forehead, and of a feeling of fullness in the left eye, which was aggravated by light. The pain was temporarily relieved by vomiting, which, however, was scanty, and not very frequent. He also complained of pain in the epigastrium, and there was slight tenderness in this region. The action of the heart was quick and somewhat irregular, and the pulse feeble; there was no rise of temperature, and the urine remained free from albumen. Beyond a slight increase in the degree of ptosis and some congestion of the conjunctiva there was no change in the ocular condition at this time. During the course of the day the headache continued unabated, and paresis of the left eye muscles steadily increased. Next morning there was absolute ptosis, and the whole of the muscles supplied by the third nerve were completely paralysed, the eye being strongly everted and the pupil dilated to its utmost extent. The fourth and sixth nerves were unaffected. The exaggeration of the knee-jerks was more marked than before, and there was just a trace of to-and-fro movement on testing for ankle clonus. The plantar reflexes were of the flexor type. The urine was still free from albumen and the temperature remained normal. By the following evening the pain had nearly subsided, the eyelid could be partially opened, and there were signs of some return of power in the paralysed muscles. On August 17th the patient was free from pain and felt well; the eye had returned to its original condition, except for some remaining congestion of the conjunctiva. Figs. 2 and 3 show the con-

Fig. 2.

dition of complete paralysis. The next attack began with headache at 7.30 p.m. on August 25th, and was followed by vomiting at 9 p.m. The attack took exactly the same course, and on August 28th recovery had taken place. Since that time I have had the boy under observation in the out-patient room, but I have only seen him in one further attack, when I was inclined to think that there was some little paresis of the left superior oblique, in addition to the third nerve paralysis. During the three months following his discharge from the hospital attacks occurred on September 18th, October 1st, 14th, and 30th, and November 9th and 20th. Latterly there has seemed to be a slight increase in the length of the interval, but I should hesitate to attribute this result to the effects of treatment, which has seemed to be quite powerless to influence or relieve the condition. Diet, regulation of the bowels, stomach treatment, salicin, potassium iodide, mercury, bromide, and citrate of caffeine have all been tried, but the result has been equally disappointing in each case. The patient was at one time of opinion that doses of nitrite given on the first appearance of symptoms tended to shorten the attack, but he no longer holds that view, and I fear that we have both lost hope of improving the condition.

The association of persistent ocular paralysis with migraine is probably somewhat more common than would be gathered from the small number of cases on record. In English medical literature I have only succeeded in finding reports of the following six cases: Dr. Saundby¹ records the case of a girl, aged 19, whose attacks dated from her 12th year, and were accompanied by paralysis of the left third nerve. In the

intervals there remained complete paralysis of the superior rectus, with some paresis of the other recti. The pupil was also persistently dilated. Dr. Saundby recorded another case² of migraine with ophthalmoplegia, but in this there was complete recovery in the intervals between the attacks. Mr. Simeon Snell brought two cases before the Ophthalmological Society.³ In the first, a girl aged 8, had been liable to attacks of migraine with third nerve paralysis from the age of 18 months. There remained persistent external strabismus and slight ptosis, with marked enfeeblement of both upward and downward movement; but the power of convergence was only slightly impaired. In the second, a man aged 27, had suffered from attacks of migraine from his 12th year, complicated for the last seven years by third-nerve paralysis. Recovery was incomplete. In discussing the former of these two cases, Mr. Ernest Clarke mentioned a similar case under his own care. Dr. Suckling⁴ reported a case in which the attacks dated from infancy, and Dr. Colman⁵ showed a patient at the Neurological Society in whom the first attack occurred at the age of 11, and was followed by persistent paralysis. In the Index Catalogue of the Surgeon-General's Library of the United States army, I have found no reference to the condi-

Fig. 3.

tion in foreign medical literature, but Dr. Saundby quotes a case recorded by Horner,⁶ and in the discussion of one of Mr. Snell's cases Dr. Ormerod also mentioned a case published by Thomsen of Berlin. I have not been able to refer to either of these reports.

REFERENCES.

¹ *Lancet*, September 2nd, 1882. ² *Ibid.*, January 10th, 1885. ³ *Ophthalmic Review*, 1885, p. 181; and 1893, p. 219. ⁴ *Brain*, vol. x, p. 241. ⁵ *Ibid.*, vol. xxiii, p. 348. ⁶ *Monats. f. Augenheilk.*, 1869.

CASE OF CEREBELLAR ABSCESS FOLLOWING MIDDLE-EAR DISEASE.

FAILURE OF RESPIRATION DURING ADMINISTRATION OF ANAESTHETIC: TREPHINING: RESTORATION OF RESPIRATORY FUNCTION FOR NEARLY THIRTY HOURS: DEATH.

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THE following case of cerebellar abscess presents some features of interest which appear to justify its publication:—

History and State on Examination.—The patient, a man, aged 26, had had for several years a purulent discharge from his left ear, but so small in amount as to cause little or no inconvenience. About the middle of June of last year he sickened, but with no definite symptoms. On June 30th

he was admitted to the Victoria Infirmary, Glasgow, under the care of Mr. Maylard. He looked ill, complained very much of his head, referring the pain chiefly to the occiput and nape of the neck, and of sickness and vomiting. His face was flushed, his eyes suffused, his pupils were equal and reacted to light. His temperature was high, 103.6° (afternoon, July 1st), his pulse was full, but numbered only 80 per minute. His tongue was dry and furred. There was no tenderness whatever over the mastoid, and none along the course of the internal jugular vein. The surface veins over the mastoid, temporal, and occipital regions were not prominent. The tympanic membrane on the left side was perforated; the discharge from the ear small in amount, and very foul-smelling. No gross lesion was noted elsewhere.

First Operation.—On July 3rd Mr. Maylard performed the usual mastoid operation. The whole postero-superior wall of the external auditory canal was removed, and the tympanic, attic and mastoid cells laid into the middle ear forming one large cavity. Granulation tissue and a small quantity of pus were found in the mastoid cells. As the bone was eroded over the sigmoid groove the sinus was exposed. The blood in the sinus was fluid. The resultant cavity was loosely stuffed with iodoform gauze, and everything left open. The operation considerably relieved him, the headache and sickness ceased. For some time, however, after the operation (ten days) the temperature remained remittent. There was very little discharge from the wound, and what there was was free from smell. The temperature gradually returned to normal and convalescence seemed established. The patient put on weight, the wound behind the ear was allowed to close. On July 15th Mr. Maylard left for his holiday, and the patient then came under my charge and more direct observation.

For nearly four weeks after the subsidence of the temperature, and for five weeks after the operation everything seemed satisfactory. The lowness of his temperature was noted, but as the pulse remained normal and no subjective symptoms manifested themselves, it was decided to await the development of events.

On August 11th, the fifty-seventh day of his illness, and the thirty-ninth since the operation, the headache recurred, at first slight, and again referred to the nape of the neck. On the evening of that day he vomited without any sickness, and apparently without relation to the food taken. On the following day, August 12th, the headache had increased in intensity, but unaccompanied by sickness or vomiting. The next day, the 13th, the headache persisted, and vomiting again occurred. Though a slight rise had taken place in his temperature, a degree in the evening, a decided lowering of his pulse-rate was observed. This at times numbered 50 per minute, and once not noted in the chart 40 per minute. It was this fall of the pulse-rate, with a corresponding diminution in the number of respirations per minute, and the feeling of depression and prostration not before manifest that caused me to come to the conclusion that some intracranial mischief was in progress.

The lowered pulse and respiration, the absence of a definite rise of temperature, pointed to intracranial pressure as a cause; the continued reference of his headache to the nape of the neck suggested the cerebellum as its seat. All other signs were negative. There was no tenderness along the course of the internal jugular vein, no emaciation, no yawning. His answers were intelligent. The condition of the surface granulation tissue along the track of the former incision behind the ear was suggestive of some further septic process. Its former red and healthy appearance disappeared at the onset of the headache, and a dirty-grey slough formed on its surface.

On the morning of August 15th he was taken to the theatre with the object of reopening the wound and tracing the sinus onwards towards the cerebellum.

Second Operation.—It is customary to have the patient anaesthetised in an adjoining room before being brought into the theatre. This was done in the present case by the anaesthetist of the institution, Dr. David Lamb.

After the patient was placed on the operating table, and before anything operative was done, he suddenly stopped breathing, the pulse-rate continuing satisfactory. Artificial respiration was at once resorted to, but no voluntary attempt at respiration followed. As long as artificial respiration was continued the pulse remained good.

The fact that a cerebellar abscess is a common cause of this phenomenon is well known, several cases having been recorded. When it was seen therefore that artificial respiration did not restore this function I lost no time in exploring the cerebellum. Some cerebro-spinal fluid escaped when the membranes were divided, but there was no bulging of cerebellar tissue, and pulsation of the exposed cerebellum was clearly visible. The absence of bulging and the presence of pulsation were remarked upon at the time and considered disappointing, as I felt sure that an abscess large enough to press on the respiratory centre would have caused some bulging at the trephine opening, and have been sufficient to render pulsation invisible. A hollow needle was inserted in a direction forwards and outwards, but failed to find pus. No reinsertion was made. Tracheotomy was performed at the anaesthetist's request.

It was thought that the patient was likely to die on the table, and failing to find pus in the cerebellum, I explored the temporo-sphenoidal lobe in the hope that I had mistaken the seat of the mischief, but with negative result. Voluntary respiration became re-established after a stoppage lasting for one hour and twenty minutes, during which time artificial respiration was continuously performed. On the restoration of respiration I reopened the mastoid wound and re-exposed the sinus. The blood in the sinus was thrombosed, but not in a state of disintegration, and as I had failed to find pus in the cerebellum, reluctantly resolved to desist from further attempt to find an explanation for the symptoms. The surface wounds over the cerebellum and temporo-sphenoidal lobe were closed, a drain being inserted in each case; the tracheotomy wound was also closed, and a small gauze drain passed down to the sinus at the mastoid opening.

Progress.—Though the patient had a somewhat restless night, he had snatches of sleep. On the following morning I dressed the wounds and left him fairly comfortable. His headache, he said, was relieved, and he was able to take some nourishment. His temperature, pulse, and respirations were increased.

At 7 o'clock that evening, thirty hours after leaving the theatre, he suddenly became livid, and it was noticed that he had again stopped breathing, though his pulse was quite perceptible at the wrist. The house-surgeon reopened the tracheotomy wound, inserted the tube, and again had recourse to artificial respiration. After this had been practised for a few minutes he voluntarily respired, but only for a short time. Failure of respiration recurred, but still with a fair pulse. He succumbed, however, two hours after the original respiratory failure, the pulse gradually giving out.

Necropsy.—Performed by Dr. Townley. The body was well nourished. The cavity formed at the first operation was examined and found to be lined with granulation tissue, the tegmen was intact, and no communication was detected with the middle fossa. The opening into the sigmoid was lined with apparently healthy granulation tissue. On removing the calvarium, some slight evidence of a commencing leptomeningitis was noticed along the reflected portion of the occipital lobe and in the immediate neighbourhood of the superior occipital convolution. On separating the attachment of the tentorium cerebelli it was at once seen that that portion of the cerebellum which lies in contact with the lateral sinus just beyond the sigmoid groove was involved.

A greenish-yellow slough of cerebellar tissue was in contact with the sinus; this was extremely friable, and gave way on the most gentle handling. When detached, about a drachm to a drachm and a-half of pus escaped. The amount of pus could not be accurately measured, but it did not exceed the quantity stated.

Surrounding the cavity was an area about $\frac{1}{2}$ in. in extent of degenerate and dead cerebellar tissue—encephalitis—of varied hue, that near the cavity being of a dirty green, and passing through various gradations of colour till that of normal cerebellum was reached. The whole of the involved portion of the cerebellum would not exceed in size that of a small walnut.

The track of the aspirating needle was then sought for, and traces of it could be found right up to the abscess wall. That it penetrated the abscess cavity there is little doubt, but in all probability it had become choked with soft brain tissue before reaching the pus. Direct pressure on the pons was improbable.

Sections were made of the entire brain, and no evidence of further lesion was detected. The whole brain was, however, oedematous, and there was marked distension of the third and both lateral ventricles, which contained an excess of cerebro-spinal fluid.

The lateral sinus was opened up and found to contain a thrombus, which extended from the jugular foramen to the torcular. It bore no evidence of disintegration, except at the part corresponding to the involved portion of the cerebellum. There was no further manifestation of lepto meningitis other than that already mentioned; the basal membranes were healthy.

Beyond a congestion of the base of both lungs there was no other lesion.

The interest of this case to a large extent centres round the sudden failure of respiration, and of respiration alone, during the administration of the anaesthetic, its subsequent restoration after a period of one hour and twenty minutes, its normal continuance for thirty hours, its recurrence with a slight intermission of three or four minutes, followed by the death of the patient. What would immediately occur to one as an explanation of these facts would be that pressure had been exerted on the respiratory centre, and on the respiratory centre alone. It is questionable whether the abscess alone was sufficient to account for this pressure. The amount of pus was small, the whole involved part did not include an eighth part of the cerebellum, and, moreover, the situation of the abscess was far removed from this centre, though in close proximity to the pons. That the anaesthetic increased the intracranial tension there can be no doubt. There was found *post mortem* an oedematous brain and an increased amount of cerebro-spinal fluid with distension of the ventricles. The oozing from the trephine opening during the time that artificial respiration was being performed temporarily relieved the pressure, and was, I believe, the cause of the return of normal respiration. The closure of the trephine wound, though a small drain was used, again allowed of reaccumulation, a renewal of pressure, a recurrence of respiratory embarrassment, and death. "In a case of otitis media purulenta, when evidence of abscess exists..... the internal hydrocephalus and oedema of the brain may actually be the cause of death in such cases. It is always serious, and when the patient is under the influence of an anaesthetic the respirations must be carefully looked to, as a little increase of oedema may cause a fatal result."

The absence of bulging of cerebellar tissue at the trephine opening and the presence of visible pulsation are also worthy of remark. Both of these conditions are emphasized in the textbooks as of almost constant occurrence in cases of abscess or tumour, and so they are in the majority of cases when the abscess or tumour is near the surface, but when the lesion is

more deeply placed and of small size the absence of the one and the presence of the other must not deter the operator from exploration in various directions.

The use of the hollow needle is, I am convinced, unsafe for exploration. It is liable to become choked with brain tissue before the abscess cavity is reached, and where there is no tension the pressure of the pus is not sufficient to free it. The sinus forceps is a much more reliable instrument and not likely to do more damage. Had sinus forceps been used in the present case the abscess cavity would have been evacuated.

SOURCE AND TIME OF INFECTION OF THE CEREBELLUM.

The source of infection of the cerebellum forms another subject of speculation. One would naturally at once conclude that extension had taken place along the lateral sinus. But if that were the case, why were there no symptoms during life of infective sinus thrombosis? True, the temperature remained remittent for some ten days after the operation, but if that were due to a septic thrombosis of the sinus, why were there no extension along the course of the internal jugular vein and lung complications? The thrombus discovered in the sinus after death was in a state of disintegration only at the site of the abscess in the cerebellum. One would have expected if infection had occurred along this route that the thrombus would have been in a state of disintegration from the sigmoid groove to the cerebellar collection of pus.

It is probable that infection occurred along another channel, possibly a vein, and in the form of a septic embolus. It is also a matter of surmise when the infection of the cerebellum took place. It is possible and probable that this occurred early in the course of the illness and before the date of the first operation. One feature of the case seems to add some support to this view, namely, the want of correspondence between the temperature and the pulse-rate during the time that the temperature was high and remittent. With a temperature of 105.2° F. on the day after his admission one would have expected a corresponding increase of the pulse-rate instead of 98 as recorded, and on the following day a temperature of 104° F. with a pulse-rate of 70. The gradual fall of the temperature to normal or subnormal with a normal pulse was misleading. A corresponding diminution of his pulse-rate did not occur until the recurrence of headache four days before his death. During the time that his temperature was normal or subnormal between July 14th and August 11th there was no symptom or sign to excite one's suspicion. The temperature, pulse, and respiration were noted daily and he was watched on account of the subnormal temperature for a possible recrudescence.

As already stated, on the recurrence of headache the pulse-rate dropped, as did also the respirations per minute. It was this combination of signs, the subnormal temperature, lowered pulse and respiration, accompanied by depression, severe headache, and vomiting, that led one to suspect increased intracranial pressure, possibly due to abscess, and that abscess cerebellar. If this assumption be correct, then it is more than likely that an attempt was made by Nature to encapsule the abscess, and that the incidence of headache, etc., revealed a reinfection and lighting up of the residual collection.

REFERENCE.

¹ Macewen, *Pyogenic Diseases of the Brain and Spinal Cord*, p. 196.

MEMORANDA:

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

SALINE TRANSFUSION IN ECLAMPSIA.

IN THE BRITISH MEDICAL JOURNAL of February 21st I see that the Clinical Society of Manchester proposes to make an investigation on eclampsia. The following case may have an interest bearing on the treatment of that disease:

On February 11th I attended a Mrs. U. in her fourth confinement, the last child having been born eight years previously. She had suffered much during the latter part of her pregnancy from great abdominal distension; otherwise her health had been good. There was no oedema of the legs or other signs of kidney mischief except occasional severe headaches. Her urine was tested in November, 1902, and found free from albumen or sugar.

Labour was slow, and no presentation could be detected at first owing to excess of liquor amnii, which on rupturing the

membranes escaped in large quantities, and which I should estimate at 3 quarts. Chloroform was administered and forceps applied, the child being delivered with the greatest difficulty, both head and shoulders offering great resistance. The child weighed 11 lb. 2 oz. and was stillborn. The patient did very well for two days, but on the third day (February 13th) she had a severe headache, pulse and temperature being normal. At 9.30 p.m. I was hurriedly called and told the patient was dying. On arrival I found she had suddenly been seized with violent convulsions a short time previously, and was in a deeply comatose condition. Severe convulsions occurred every ten or fifteen minutes without any return to consciousness.

Dr. Purslow kindly saw her with me, and we gave morphine, $\frac{1}{2}$ gr. hypodermically; and transfused a pint of saline solution into the subcutaneous tissue of her abdomen. The effects were striking, for in about ten minutes she passed into a quiet sleep lasting for three hours, and free from stertor. She awoke semi-conscious, and in a few minutes had another severe convulsion followed by deep coma, the fit was repeated in half an hour, and coma even more pronounced, with a pulse of 120.

At this time her condition appeared quite hopeless, but as a last resource I again transfused, and with the same beneficial effects, for in a quarter of an hour she was quietly sleeping, and the stertorous breathing, which previously could be heard all over the house, had entirely disappeared.

She had one more slight convulsion some hours later. Her urine was drawn by catheter, and found to contain a light cloud of albumen.

During the next twenty-four hours she was very delirious, throwing the clothes off and attempting to get out of bed. This was followed by a good night's sleep, and next morning she awoke quite rational.

The points of interest in this case to my mind are the striking results of saline transfusion; and, secondly, the violent delirium next day, which I took to be partly due to the extremely diluted condition of her blood.

Birmingham.

GEORGE ST. JOHNSTON, M.D.Lond.

THE TREATMENT OF CONGENITAL PHIMOSIS.

IN THE BRITISH MEDICAL JOURNAL of March 7th, p. 545, Dr. J. F. Woodyatt expresses his dissatisfaction with ordinary circumcision as performed for congenital phimosis, and describes "a much simpler operation." The latter is identical, if I am not mistaken, with one recommended forty years since by Mr. Furneaux Jordan, of Birmingham.

But even on Dr. Woodyatt's own showing, the simple operation he performed is hardly indicated. He says "the condition which prevents retraction of the prepuce is due to tightness of the mucous membrane and not to any contraction of the skin itself." If the word "adhesion" be substituted for "tightness," no description could be more accurate. Unless a mucous membrane has been structurally modified by disease or injury, it cannot well be associated with any idea of tightness or rigidity. All the preputial tissues are obviously soft and distensible.

The trouble in congenital phimosis depends on adhesion of two contiguous mucous surfaces with consequent retention of secretions. It ceases for good and all when one layer is gently peeled off the other and the smegma removed. Any one who attempts this under anaesthesia will be surprised to find how many seemingly tight prepuces, no matter what the age of the patient, can at once be retracted without any instrumental interference.

If, however, there be still difficulty, that can be readily overcome by the introduction of sufficiently long-bladed dressing forceps, and subsequent expansion of the handle, usually in two directions. The mucous membrane and skin are thus well stretched to the point at which the former is adherent. It should be peeled off with the fingers till the gland is free all round, and after removal of the smegma duly anointed. No further treatment is necessary.

Circumcision is of course but a "lingering reminiscence of human sacrifice." How it can still find favour as a justifiable surgical weapon for congenital phimosis I am at a loss to imagine. No one who fairly tries dilatation would resort to even the mild treatment advocated by Dr. Woodyatt. But some profess to have done so with non-success. There are two reasons for this. The most common is carelessness in not securing complete exposure of the corona glandis. The next is laceration by too much roughness at the time, or by meddlesomeness afterwards, as by daily retraction of the prepuce with a screaming infant. In this way adhesions may