the notes of the patients, who were under their care at Brompton Hospital, London.

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OCULAR PARALYSES FOLLOWING MUMPS

BY

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The comparative rarity of the neurological complications of mumps and their unusual localization and termination in one of the two following cases have led us to put them on record.

Case I

A boy aged 9 came to the Coventry school clinic in December, 1929, because he had recently found that he could not see to read. While at the clinic he vomited, and it was noticed that a large quantity of the vomit came through the nose. He had had mumps three weeks previously, and a fortnight before admission he noted that he could not see to read. For the past fortnight food had regurgitated through his mouth.

There was complete paralysis of accommodation; the pupils were widely dilated and did not react to light. No external muscles were affected. The vision was as follows: right eye, 6/60, with +2 = 6/9; and the left eye, 6/60, with +2 = 6/9. There was slight blurring of the edges of both disks. The knee-jerk was absent on the right side, and its presence on the left was doubtful. The boy was admitted to the eye wards of the Coventry Hospital. A swab was returned as negative for the Klebs-Loeffler bacillus. He was transferred to the medical wards and lost sight of.

He was written for and examined on July 4, 1934, when he was aged 14. The disk edges were still somewhat blurred, but the eyes were apparently quite normal, and there was no evidence of any paresis of any extrinsic or intrinsic muscles. At the time of the first visit we found that Osler in his *Textbook of Medicine* had mentioned that mumps was occasionally followed by peripheral neuritis.

The case may be summed up as an example of the picture presented by post-diphtherial paralysis of the intrinsic muscles of the eye, but caused by the virus of mumps. It is probable that the slightly blurred disks were physiological and had nothing to do with the infection.

Case II

Another boy, aged 12, came to the Eye Department of Coventry Hospital on March 15, 1933, complaining of blurred vision in the left eye and inability to see things on the right side without turning the head in their direction. The symptoms had come on gradually a few days previously. One month before this date he had developed mumps. The attack was of ordinary severity, and he did not stay in bed. A sister had suffered at the same time. There had been no headache, fits, or any cerebral symptoms. His previous health had been very good. One brother had died recently from

pulmonary tuberculosis and a sister from meningitis, probably tuberculous.

We found that the vision in the right eye was 6/24, and with glasses 6/4.5. In the left eye it was 6/24, and with glasses 6/18. Both fundi were normal. All the intrinsic and extrinsic muscles supplied by the third nerve were paralysed on the left side. The pupil was widely dilated and inactive. There was incomplete left ptosis, with compensatory overaction of the left half of the frontalis. Food regurgitated through the nose. The general nervous system was normal. The Mantoux and Wassermann tests were negative. No Klebs-Loeffler bacilli were found. There was a history that twelve months previously the left eye had been struck by a golf ball and that two stitches had been put into the lids, one into each. The eye was not damaged, and there was no diplopia after the blow.

On March 17 paresis of the left external rectus appeared; the superior oblique remained active. On May 23 the paralysis of the eye muscles was reported to be improving, and on June 19 the eye movements were almost normal. On July 17 all the eye movements were normal except deorsum vergens. The pupil was still dilated. On August 21 the left pupil was still dilated and inactive, and upward movement of the eye was defective. No change was noted on October 2, December 11, or December 18. The vision of the right eye was 6/4.5 and of the left 6/18 partly (with glasses).

On January 15, 1934, there was slight defect of convergence and abduction. The pupil was still dilated and inactive on March 5. Deorsum vergens was still defective. There was no change on April 9. On May 14 there was still third nerve paralysis; the left pupil was fully dilated and inactive. The fundi were normal. The condition was not so good as previously. In November there was still no change or improvement.

Comment

Numerous investigations of cases of uncomplicated mumps have shown that a meningeal reaction, as indicated by a lymphocytic pleocytosis, is present in most cases of this disease, even in the absence of any symptoms or signs indicative of cerebral involvement. It seems probable that the virus of mumps is itself potentially neurotropic, since several cases are on record in which clinical meningitis and encephalitis have preceded the onset of the glandular swelling. The pathology would therefore seem to be distinct from that of the cerebral complications of the other virus diseases (such as vaccination, varicella, and encephalitis), in which it is generally held that the neurological complication is caused by a dormant virus which has been awakened to activity by the attack of the exanthem.

The peripheral nerve palsies accompanying mumps are often considered to be of the same nature as those associated with various toxins and infections, and to be a lesion of the peripheral nerve endings; but in view of the frequent meningeal reaction of parotitis it would seem equally likely that in some cases the lesion is one of the nerve trunk near its root (a meningo-radculitis). This would bring the pathology into line with that of the peripheral (cranial and spinal) nerve palsies associated with the various forms of meningitis, acute and chronic.

The time of onset of the neurological complications is usually at the height of the parotitis, while it is subsiding, or in the early period of convalescence (third to fourteenth day). Periods of three weeks have been recorded. The longer interval of six weeks in Case II is unusual.

It appears certain that in some cases cranial nerve palsy due to the mumps virus may occur in patients in whom there has been no apparent parotitis and no orchitis, as is shown by the occurrence of such palsies in contacts in barracks during epidemics, and in a member of the family of a definite parotitic patient. The possibility of such an

origin has therefore to be borne in mind in all cases of unexplained cranial nerve palsies. In the almost constant meningeal reaction of mumps may also lie the explanation of many cases of acute aseptic benign meningitis without parotitis.

Of the cranial nerves involved, the optic, the facial, and the acoustic are the commonest in the recorded cases. The third nerve and the post-diphtherial type of palatine and accommodation paralysis are much rarer. The benign and often transitory character of the effects on the nerves which result from the penetration of the sero-fibrinous exudate into their trunks at their origins and the pressure on these from the inflamed meninges have been emphasized. The two notable exceptions to this are the optic and acoustic nerves, where atrophy is apt to follow, with disastrous consequences. The two cases here recorded are therefore of interest in the type of nerves involved and in the absence of recovery in one.

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Clinical Memoranda

Local Anaesthesia in the Repair of Torn Perineum

Anyone who has had any experience with cases of prolapse of the uterus and vagina cannot fail to be impressed by the enormous weight of evidence in favour of lacerations of the perineum as a contributory factor in the aetiology of this distressing condition. While holding an appointment as a house-surgeon I saw a large number of such cases, and took a careful obstetric history from each. There were two points which struck me as being of great importance: (a) practically every patient gave a history of a perineal laceration; (b) a large number of patients said that either their doctors did not repair the tear or that the midwives did not call in a doctor to see the tear.

Margaret Salmond and Gertrude Dearnley (*J. Obstet. Gynaec. Brit. Emp.*, 1935, p. 446), in an investigation into this subject, showed that midwives were inclined to overlook tears and that results obtained by general practitioners were not very good. However, they offered no explanation why the practitioners' results were poor. The reason is, I think, that as the practitioner usually has no skilled anaesthetist at his disposal he is forced to do the operation without an anaesthetic at all, and in his effort to spare his patient as much discomfort as possible he hurries the procedure, with the result that it is not properly done. Therefore we need a method whereby anaesthesia can be produced easily, effectively, and single-handed by a general practitioner. When the procedure is carried out in the patient's home naked lights such as fires, gas flames, etc., preclude the use of inflammable anaesthetics. A gas-and-oxygen apparatus is expensive and clumsy, and needs a second skilled person to manage it. A small bottle of novocain solution, a 5-c.cm. syringe, and a needle in a sterile container occupy very little space, and may be carried in the pocket. The practice of allowing midwives to repair the tear while the practitioner administers the anaesthetic is one to be most strongly condemned.

TECHNIQUE

The patient having been placed in the lithotomy position with sterile towels in place and the vulva thoroughly cleansed and painted with such antiseptic solution as the operator is in the habit of using (Bonney's blue, flavine, etc.), a 5-c.cm. syringe is filled with 4 per cent. novocain solution. The skin edges of the tear are infiltrated throughout their entire length. The infiltration should be done internally, and the skin not punctured at all. This is usually all the infiltrating that is necessary, since the torn deeper structures are as a rule insensitive to pain. In tears involving the levators, however, I have found that the muscles are sensitive, and I recommend their infiltration. About 4 c.cm. of 4 per cent. novocain solution is sufficient for the average repair. The largest amount that I have used was 10 c.cm. for a complete tear involving the whole of the posterior vaginal wall, the rectum, and external sphincter. I recommend the repair being done within two hours of delivery, as at the end of this time sensation begins to return to the deep structures.

RESULTS

All the patients have expressed their gratitude for not having been "put to sleep," and some who have had previous repairs have compared this method very favourably with the others they have had with general anaesthesia or with no anaesthetic at all.

The healing has in all cases been most successful, and I have had none which have broken down or gone septic. It has been suggested to me that the novocain might cause soddening of the tissues in this potentially septic area, and therefore the procedure might be considered risky. Such quantities of novocain which would cause this are never necessary to produce the required anaesthesia, and no ill effects have followed its use in any of my cases. These observations are based on over 100 cases in hospital and on the district.

CONCLUSIONS

1. Suture of perineal lacerations is essential, and unless done thoroughly is useless.
2. The anaesthetic of choice is local infiltration.
3. Infiltration of the skin edges is usually all that is necessary, and should be done internally and the skin not punctured.
4. It is an extremely practical method for general practitioners.
5. It is very much pleasanter for the patient.
6. The use of general anaesthesia for the repair of even large tears is both undesirable and unjustifiable.

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P. Klotz (*Thèse Paris*, 1937, No. 35) maintains that polyneuritis is most likely to develop in female subjects, as they are particularly susceptible to endocrine disturbance. Experimentally a previous upset of the endocrine system by ovariectomy makes it impossible to produce that habituation to alcohol which it is easy to obtain in the healthy animal. On the other hand, such an attempt at habituation results in the production of a state of hypersensitivity to the poison. Alcohol in polyneuritis develops after a more or less prolonged period of dyspepsia, with atrophic gastritis and degenerative hepatitis, and is more often produced in such cases by infections, of which tuberculosis is the most important. It sometimes appears in an individual who has abstained from alcohol for some weeks or months. This polyneuritis is very often associated with anaemia and pronounced endocrine disturbances, among which thyro-ovarian insufficiency predominates.