distance. But there seems to be no case in the literature in which receding reduplicated images have been seen.

Another unusual feature is that the images are multiplications of objects actually seen—in the absence of illumination there are no hallucinatory phenomena. As these seizures were also characterized by turning of the head and eyes to the right and smacking of the lips, a left temporo-frontal focus was sought. It is generally considered that the occurrence of organized visual hallucinations indicates a disturbance in one or other temporal lobe, usually in the dominant hemisphere; and Penfield and Erickson (1941) and Marchand and de Ajuriaguerra (1948) classify seizures characterized thereby in the group of so-called "psychical seizures." In the present case, however, no clinical or electroencephalographic evidence of such a lesion could be found.

Conclusion

These two children have electroencephalographic records and clinical histories which suggest that they are suffering from a paroxysmal cerebral dysrhythmia of non-focal type. In each case the seizures are ushered in by unusual introductory phenomena, which, on current physiological grounds, might be expected to be related to disturbance in the anterior temporal lobe or island of Reil, but in which no evidence of such dysfunction can be found. It seems that these are cases of "idiopathic" epilepsy with unusual aurae, and as such have responded to treatment with phenobarbitone, with disappearance of seizures over the past year, during which time they have been under regular supervision.

My thanks are due to Dr. W. Ritchie Russell for permission to report these cases from his clinic, to Miss F. M. Taylor for carrying out the electroencephalographic examinations, and to Miss A. Arnott for working up the patient's drawings.

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Arguing that blood does not normally clot in the vessels because of the chemical nature of the endothelial lining of the vascular system, which is probably an acidic polysaccharide such as mucoitin, Drs. Lovelock and Porterfield have prepared (see *Nature*, January 6, p. 39) an artificial surface with a similar high negative (anionic) charge. They show that polystyrene tubes after a brief treatment with sulphuric acid are much better than untreated tubes and very much better than glass-ware in holding blood both fluid and free of clots.

SIXTH-NERVE PALSY AFTER LUMBAR PUNCTURE AND SPINAL ANALGESIA

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Palsy of the sixth cranial nerve following spinal analgesia has been reported regularly since soon after the introduction of this method of pain relief, and it is perhaps a reflection on anaesthetists that most of the communications on this subject come from ophthalmologists. The incidence of the complication is difficult to determine. Mild degrees of abducens paralysis probably pass unnoticed or unheeded; and although the highest figure given, 1% of all spinal analgesics, by Terrien (1923) is considerable, it is possible that the figure should be considerably greater.

Paralysis of the various cranial nerves, except the first and tenth, has been reported, but there is general agreement that the sixth is most often involved. The lesion is discussed by many, including Mühsam (1906), Babcock (1913), Tovell (1933), Fairclough (1945), Steinberg and Bishop (1946), Rose and Pritzker (1947), Azambuja (1947), and Parke (1948).

Aetiology

The sequence of events is remarkably constant. The paralysis is not noticed until 3 to 21 days after the spinal analgesia, but it is preceded by a period during which the patient complains of severe headache and perhaps dizziness, nausea, stiff neck, photophobia, and diplopia. The delay in onset of the palsy has given rise to many theories about its causation, none of which is subject to proof. They may be summarized as follows:

1. *Mechanical.*—A rise in intracranial pressure causes a "coning" of the brain through the foramen magnum so that the sixth nerve (see Diagram), anchored between its entry into the cavernous sinus and its attachment to the pons, is stretched over the apex of the petrous portion of the temporal bone (Fairclough, 1945).

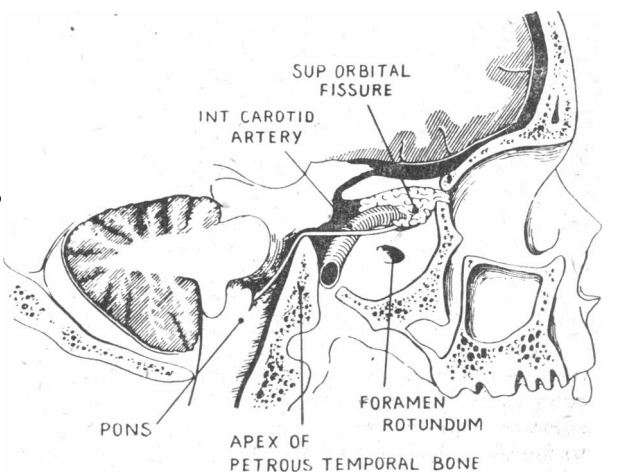

Diagram showing course of the sixth nerve. (After Wolff.)

2. *Toxic.*—This is specific degenerative change following the use of certain local analgesic drugs, and based on the known susceptibility of certain nerves to particular toxins—for example, the radial nerve to lead, and the posterior columns to ergot. If this theory were correct the lesion would be expected to be bilateral, whereas that is rarely the case. That procaine can cause nerve degeneration is known (Spielmeyer, 1908 ; Lundy, Essex, and Kernohan, 1933). Dinsdale (1947) has reported cauda equina lesions after the use of spinal analgesia, but the sixth nerve is never bathed in the high concentrations he mentions. Further, the complication has followed the use of a wide variety of drugs (Blatt, 1928) and even simple lumbar puncture for diagnostic purposes (Dattner and Thomas, 1941).

3. *Inflammatory.*—This implies a low-grade meningitis. Babcock (1913) attributed two cases of his to this. But the unilateral palsy and absence of an accompanying rise in temperature are good arguments against this view. The complication affects women slightly more often than men. The prognosis is favourable, and over 50% recover within a month of onset. On rare occasions the paralysis is permanent, and if after an interval of two years recovery has not taken place a corrective operation may be necessary. Generally the treatment is palliative. The troublesome symptom of diplopia is prevented by covering the affected eye with a shield, and the headache is relieved by suitable analgesics. Therapeutic lumbar puncture has been recommended with the object of lowering a supposedly raised intracranial pressure (Hayman and Wood, 1942), but the pressure of the cerebrospinal fluid may well be low and the procedure result in more harm than good.

Case Reports

Two cases have occurred recently in our hospital—one following simple lumbar puncture, the other after a spinal analgesic.

Case 1.—A woman aged 42 was to receive an extradural analgesic, but inadvertently the dura was pierced by a 16-gauge Tuohy needle, with the patient lying in the right lateral position. There was a rapid loss of about 5 ml. of cerebrospinal fluid through this large-bore needle. At this stage the technique was abandoned and a general anaesthetic given instead. The operation of cholecystectomy was uneventful. The next day the patient complained of nausea, pain in the back of the neck, and headache, aggravated on sitting up. Three days later double vision was remarked on. The next day there was a complete paralysis of the left lateral rectus muscle. It took three months for recovery to be complete.

Case 2.—A woman aged 49 was to undergo a total hysterectomy. An extradural analgesic was intended, but again an inadvertent spinal tap was made with a 16-gauge Tuohy needle. When the woman was lying on her left side, 8–10 ml. of cerebrospinal fluid was lost rapidly and the patient complained of severe frontal headache at that time. Light "nupercaine," 15 ml., was injected and the patient put lightly to sleep with a general anaesthetic. The next day complaint was made of headache, aggravated by sitting up, and slight nausea. Three days later double vision occurred, and on the seventh post-operative day partial paralysis of the right rectus muscle was evident. Recovery in this case was more rapid and was complete in six weeks.

These two cases have two points in common. A large-bore needle was used, causing a rapid loss of a comparatively large volume of cerebrospinal fluid, and external rectus paralysis developed in the eye opposite to the side on which she was lying. The brain, deprived of its water cushion through the sudden loss of cerebrospinal fluid, sags and a strain may be thrown on the sixth nerve, which is uppermost. The nerve is stretched, but the lesion is due primarily to reduced and not to raised intracranial pressure, as suggested by Fairclough.

When considering the pathogenesis of sixth-nerve palsy three facts should be borne in mind. The paralysis is preceded by headache. The causal relation of headache to prolonged seepage of cerebrospinal fluid through the hole made in the dura by lumbar puncture is now widely accepted (Franksson and Gordh, 1946). A patient suffering from a post-lumbar-puncture headache invariably assumes a horizontal position in bed, immobile and curled up on one side. These circumstances may account for stretching of the upper abducent nerve. In one-quarter of the reported cases the lesion is bilateral: the explanation may be that after some time the patient, by rolling on to the opposite side, exposes the other nerve to trauma.

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TORSION OF THE GREAT OMENTUM

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Torsion of the great omentum is a well-recognized condition, but the number of recorded cases is still comparatively small. The torsion may be primary or secondary, complete or incomplete, and partial or total. Primary torsion where there is no recognizable cause is the less common. In secondary torsion, a hernia, neoplasm, cyst of omentum, tuberculosis, or evidence of past or present intraperitoneal inflammation is present.

Marchetti (reported by Aimès, 1919) is credited with having reported the first case of torsion of the great omentum in 1851. Oberst (1882), however, was the first to describe the condition. In each of these cases the condition was associated with an inguinal hernia. Eitel (1899) reported the first case of primary torsion. Corner and Pinches (1905) collected 54 cases from the literature. Morris (1932) collected a further 161 cases, and added three of his own, bringing the total number reported to 218. Etherington-Wilson (1945), in a critical review of the literature, reported 190 cases of torsion of omentum, 73 of which were considered to be primary or idiopathic. The number of cases reported by different authors varies. This is due to the fact that some authors have included cases in which torsion has been