BIBLIOGRAPHY

- CANTONNET.—L'ionisation de l'oeil. Technique Resultats Editions Medicales N. Malione, Paris, 1927.
- ERLANGER —On Practical and Scientific Significance on Iontophoresis in Ophthalmology. International Congress of Ophthalmology, Amsterdam, Vol. I, p. 196, 1929.
- On the Scientific and Practical Value of Ionisation in Ophthalmology. *Brit. Jl. Ophthal.*, Vol. XX, p. 213, 1936.
- Iontophoretic Medication in Ophthalmology. *Arch. of Phys. Ther.*, Vol. XX, p. 16, 1929.
- KARBOWSKI.—Iontophoresis in Ophthalmology. *Ophthalmologica*, Vol. LXLVII, p. 166, 1939.
- LE CAM.—De la valeur de l'ionisation en therapeutique oculaire (On value of Ionization in Therapeutic Ophthalmology), Thesis of Medical Faculty of University of Paris, 1926.
- LE COUILLARD.—Ionisation dans les sclerites (These de Paris, 1933).
- LEDUC.—Introduction des substances medicamenteuses dans la profondeur des tissue par le courant electrique. *Ann. d'Electrobiol.*, Vol. III, p. 545, 1900.
- MORIZOT.—Treatment des Affections Oculaires par l'ionisation. *Jl. de Med. et Chir.*, 10, 8, 32. Also 110, 329, 1939.
- ROCHAT.—On iontophoresis of mydriatics in treatment of iritis and iridocyclitis. International Congress of Ophthalmology, Amsterdam. Vol. I, p. 199, 1929.
- ROHOZINSKI.—Penetration electrolytic les substances medicamenteuses dans les milieux oculaires. Paris. E. le François, 1936.

INJURY TO THE OPTICO-CHIASMAL JUNCTION—A CASE REPORT*

BY

E. B. C. HUGHES

THIS interesting case of injury to the visual pathway presented a difficult problem in localisation and in pathology. I can find no case quite comparable to it in the literature, and it has the added advantage that the lesion has been inspected at operation.

Major H. W. B., aged 31 years, sustained a severe head injury as the result of a head-on motor crash, the site of the injury was the left frontal region, and he had a fissured fracture running down in the left frontal bone, traversing the left frontal sinus, running along the floor of the left anterior fossa lateral to the optic canal, and then passing down into the left middle fossa. The actual course of this fracture has been verified in the course of two operative procedures. No definite fracture into the left optic canal was located, either by X-Ray or by direct vision; X-Ray, however, five months after injury showed this canal to be a little irregular in outline and larger than its fellow. No fracture of the left anterior clinoid process was demonstrable by X-Ray, or seen at operation.

He was admitted to another hospital, and some thirty-six hours

*Received for publication, May 25, 1943.

later was transferred to an E.M.S. Neurosurgical Unit. Here, two days after injury, a left epidural haematoma was exposed and removed through a temporal craniotomy. Until that time he had been stuporose, but he recovered consciousness in the operating room, and within a few hours of operation severe loss of vision could be demonstrated in the left eye. His condition improved steadily, and six days after injury adequate clinical examination could be carried out. This disclosed, amongst other signs, a partial left third nerve paresis, severe loss of vision in the left eye, with doubtful perception of light in the upper nasal field only. The visual field in the right eye was passed at that time as normal. Both then, and on two further occasions, our consultant ophthalmic surgeon, Mr. F. Ridley, and myself, passed the right visual field as normal to rough confrontation tests during the next week.

At about this time he had a clinical attack of meningitis, this lasted a few days only, and responded rapidly to sulphapyridine. At this time also the left third nerve paresis began to progress and thirteen days after injury had become complete. Visual acuity in the left eye was by this time good finger counting in the upper nasal field only, the right field again being passed as normal. During the next few weeks he remained confused mentally, a left frontal intracerebral aerocele was suspected and confirmed by X-Ray. Owing to our anxiety over this matter clinical examination of the visual fields over the next few weeks appears to have been confined to the left eye only. This remained about stationary with a large upper nasal island of vision, of 1/60 acuity.

In time the frontal aerocele absorbed and filled with fluid. Operation had been decided upon for this condition some time previously but was postponed owing to minor skin sepsis from the former operation site. It was not until ten weeks after the injury that his condition had improved enough for us to take our attention from the cerebral condition and turn to the more academic study of the visual fields, and full quantitative perimetry was undertaken for the first time. (Fig. 1.) This showed a large island of vision in the upper nasal field on the left, including a little macular vision; acuity was still 1/60.

In the right field, somewhat to my surprise, there was a large upper temporal quadrantic defect and some depression in the lower temporal field to 1/2,000 and 2/2,000 white. Acuity was 6/18. This field pattern remained stationary until the second operation some sixteen weeks after his injury. This was undertaken to drain the aerocele cavity and repair the dural defect over the frontal sinus. A left frontal bone flap was turned down. The line of fracture was seen as described above, the dural defect had healed soundly and was lightly adherent to the fracture line. On

FIG. 1.—Fields ten weeks after injury.

RIGHT EYE: Upper temporal quadrant defect to 7/330, 3/330 and 10/2000, with severe depression in the lower temporal field as well to a 2/2000 and 3/2000.

LEFT EYE: Upper nasal island of vision only, with some sparing of the macular field.

FIG. 2.—Fields three weeks post-operative—nineteen weeks after injury.

RIGHT EYE: Upper temporal quadrant depression to 3/330, and 10/2000, with some depression in the lower temporal field to 3/2000, and 2/2000. The isopter for 7/330 shows a full field but with only dim vision in the upper temporal periphery (dotted area), and with an absolute scotoma in the upper temporal quadrant (cross hatched).

LEFT EYE: Shows a general enlargement of the nasal island of vision, with considerable return of the central field.

opening the dura and retracting the frontal pole, the posterior part of the anterior fossa was found to be the seat of massive adhesions, which extended back as far as the sphenoid ridge and enveloped the intracranial part of the left optic nerve as far as its chiasmal junction. The chiasm itself appeared to be normal. The nerve was freed from its adhesions and appeared to be rather shrunken and of a yellowish waxy colour. In view of the accepted pathology of these lesions the optic canal was not decompressed and the operation was terminated by drainage of the frontal cyst resulting from the aerocele.

Recovery from this operation was uneventful, apart from a very striking change in his mental state. From a state of mild confusion he returned to almost normal within a few days of operation. An equally startling and rather unexpected change took place in his visual fields. Vision in the left eye rapidly improved, and within fourteen days of operation had changed from 1/60 to 6/36. Perimetry showed this to be due to a general enlargement of the island of vision to include the greater part of the macular vision. The field in the right eye also improved, the quadrantic defect rapidly dwindling, and twenty-one days after operation a very dim peripheral rim appeared in the upper temporal field, leaving him with a moderate sized upper temporal scotoma. (Fig. 2.)

Discussion

In the evaluation of this confusing lesion of the visual pathway, one must postulate two pathological processes at work. There can be no doubt that he suffered a lesion of the left optic nerve and possibly of the anterior chiasmal angle, which was present as soon after the injury as adequate examination could be made. This must be presumed to be the result of the original injury. Most authorities now believe that these lesions are the result of vascular damage in the nerve or chiasma (Traquair, Dott, and Russell, 1935). They are maximal at first, and some recovery can be expected up to four weeks, but seldom after that time (Traquair, 1942). It must be assumed also, that if the lesion is the result of thrombosis and softening in the nerve, late operative procedures such as decompression of the optic canal or the division of adhesions, will have little effect on the recovery of vision. Other pathology has been suggested to account for chiasmal injuries. Coppez (1929) suggests antero-posterior tears as the cause, and Campbell and White (1938) postulate an intrasellar haematoma pressing up on to the chiasm; Dandy (1942) also mentions such a case. Neither of these lesions would seem to have been present in this case, and one must agree with Traquair that most of such lesions have a vascular basis. I have no doubt that this patient

suffered from such a vascular lesion of his left optic nerve, sparing upper nasal vision, and the greater part of the macula, and producing no gross defect in the right field. Subsequently he developed a gross field defect on the right, and the process of recovery in the left eye, if not actually retrogressing, was at least halted. I think that this was due to some secondary process, related to the attack of meningitis, and portrayed at the second operation as a mass of adhesions around the nerve. It is to be noted that the third nerve paresis, incomplete at first, progressed to completion during this same period. Following operative attack upon this secondary lesion, a rapid and marked recovery took place, leaving him with upper nasal vision on the left sparing the macula, and a small upper temporal scotoma on the right. This final state, now stationary, must portray the true extent of the initial vascular lesion of the nerve. It is interesting to note in this connection, that pressure on the chiasm from tumour commonly leaves such an upper nasal island until a later stage. So common is this sparing of the upper nasal fibres that Traquair (1942, p. 220) has even suggested that these fibres may have a separate blood supply, or at least a blood supply that is more "sheltered" than the rest of the direct fibres.

The site of this man's damage must be either in the termination of the left nerve, or in the anterior chiasmal angle. It is a lesion which corresponds to the "junction" type of defect affecting the fibres of the left optic nerve mainly, but also catching the knee of crossed fibres from the lower nasal retina of the right eye. The presence of this knee of fibres was postulated by Willbrand and Saenger (quoted by Traquair, 1942) but its actual location and depth of penetration into the terminal part of the opposite nerve still requires to be demonstrated. The case outlined above, while failing to bring positive proof of the presence of this knee of fibres, adds yet a little more to the great weight of circumstantial evidence suggesting their presence in this situation.

My thanks are due to Mr. Harvey Jackson for permission to report this case, and to Mr. Frederick Ridley and Major J. W. Aldren Turner, R.A.M.C., for their help and advice in its presentation.

REFERENCES

- TRAQUAIR, H. M., DOTT, N. M., RUSSELL, W. R. (1935).—*Brain*, Vol. LVIII, p. 398.
 TRAQUAIR, H. M. (1942).—*Intro. to Clinical Perimetry*, London, p. 201.
 COPPEZ, H. (1929).—*Arch. d'Ophth.*, Vol. XLVI, p. 705.
 CAMPBELL, E. H., WHITE, E. (1938).—*N.Y. St. Jl. Med.*, Vol. XXXVIII, p. 841.
 DANDY, W. E. (1942).—*Dean Lewis' Practice of Surgery*, Vol. XII, p. 299.
 WILLBRAND u. SAENGER, quoted by Traquair (1942), *loc. cit.*, p. 398.