8. ECKER, ARTHUR.—Treatment of high fever following head injury and suggestion concerning ventricular drainage for acute traumatic hydrocephalus. *The Military Surgeon*, Vol. XCI, pp. 670-675, 1942.
9. BEATON, L. E.—Neurogenic hyperthermia and its treatment with soluble pentobarbital in the monkey. *Arch. Neur. and Psych.*, Vol. XLIX, pp. 518-547, 1943.
10. NATHAN, P. W. and TURNER, J. W. A.—The efferent pathway for pupillary contraction. *Brain*, Vol. LXV, pp. 343-352, 1942.
11. WILLIAMS, DENIS.—The electro-encephalogram in acute head injuries. *Jt. Neur. and Psych.*, Vol. IV, pp. 107-136, 1941.
12. FITZGERALD, GERALD and HALLPIKE, C. S.—Studies in human vestibular function. 1. Observations on the directional preponderance (Nystagmus-bereitschaft) of caloric nystagmus resulting from cerebral lesions. *Brain* Vol. LXV, pp. 115-138, 1942.
13. LILLIE, W. I. and ADSON, A. W.—Unilateral central and annular scotoma produced by callus from fracture extending into optic canal. *Arch. of Ophthal.*, Vol. XII, p. 500, 1934.
14. TRAQUAIR, H. M., DOTT, N. R. and RUSSELL, W. R.—Traumatic lesions of the optic chiasma. *Brain*, Vol. LVIII, p. 398, 1935.
15. TRAQUAIR, H. M.—An introduction to clinical perimetry. Kimpton, London. 1942.
16. HUGHES, E. B. C.—Injury to optico-chiasmal junction. A case report. *Brit. Jl. Ophthal.*, Vol. XXVII, 8, pp. 367-371, 1943.
17. VAIL, DERRICK.—Syphilitic opticochiasmic arachnoiditis. *Amer. Jl. Ophthal.*, Vol. XXII, p. 505, 1939.
18. HOLBOURN, A. H. S.—Mechanics of head injuries. *Lancet*, p. 438, October 9, 1943.
19. ECKER, ARTHUR D. and ANTHONY, EUGENE, W.—Exanopic central scotoma—a pitfall in diagnosis. (To be published).

TRANSPARENCY OF THE LENS FOLLOWING TRAUMATIC CATARACT*

BY

Major H. J. STERN, R.A.M.C.

It is well known that the typical posterior cortical cataract due to contusion or perforation of the lens sometimes clears up completely. This particular form of opacity in the lens depends on the entrance of fluid underneath the capsule and between the lens fibres. The vacuoles thus formed are of a refractive index different from the lens substance and the lens appears opaque although its fibres are still transparent. At this stage the vacuoles may be absorbed and the "cataract" appears to resolve.

The case to be described presents a most unusual picture in addition to this phenomenon.

A West African negro, aged 25 years, presented himself in the out-patient department with a note from his Medical Officer saying that his left eye had been injured by a flying particle of stone. The report stated that the stone splinter had been seen in the cornea and that a small hyphaema had been present. At the time of the examination, however, several hours after the

* Received for publication, September 29, 1944.

injury, only a small corneal abrasion was observed and the anterior chamber was clear. Otherwise the eye appeared normal, with the exception that an anterior-capsular cataract was present. Exactly the same abnormality was seen in the other eye. The normal fundi were clearly visible and the visual acuity was 6/6 in both eyes. It was assumed therefore that the stone splinter had been washed off the cornea and the patient was treated as a corneal abrasion. On the next day the abrasion was nearly healed, the pupil well dilated with atropine and two days later the eye was almost quiescent.

Three days after the original injury the patient came again, complaining of violent pain in his left eye. On examination a

most surprising picture presented itself: A brilliantly white mushroom-shaped mass was protruding from the lens into the anterior chamber. There was a marked ciliary injection and the intra-ocular pressure was 40 mm. Hg. X-ray of the eye showed no radiopaque foreign body. The tension could be lowered by cautious massage of the eyeball and application of heat. The eye settled down during the next days with no further accidents.

On the sixth day the opaque lens masses had markedly diminished in size and the next day they were less than half the diameter of the original mushroom. A normally transparent lens was visible behind them. This surprisingly quick absorption of the opaque lens masses continued rapidly, and 13 days after the injury only a few very fine, grey capsular opacities were left, surrounded by brown iris pigment. At this stage a feathery posterior cortical cataract was observed, the vision being 6/60. This, however, cleared up almost completely, and on the 23rd day the eye was again white and perfectly normal apart from the unchanged anterior capsular cataract and a fine corneal opacity near the centre. Only with the slit-lamp some fine, linear opacities could be observed in the anterior capsule. Vision was 6/6 and retinoscopy revealed emmetropia.

It might be assumed that the stone splinter had actually perforated the cornea and the lens and disappeared somewhere in the periphery where it could not be seen; as most stone splinters are not radiopaque the negative X-Ray findings do not contradict this conception although the aspect of the lens on the day of the

injury makes it highly improbable. If no perforation had taken place the contusion alone of the eyeball might have produced a rupture of the capsule. In both cases it is difficult to explain the delay of three days in the appearance of lens masses in the anterior chamber.

Another feature difficult to explain is the complete closure of the tear in the capsule after the loss of cortex. After a minute tear in the anterior capsule the wound lips curl outwards and never reunite end to end. If no lens matter appears in the wound—which may happen under certain conditions—a fibrinous exudate is formed on the anterior surface of the lens and the defect in the epithelium is replaced by the neighbouring epithelial cells which slide over it. Later the fibrinous membrane is absorbed and the epithelial cells produce homogeneous lamellae which form a new capsule. It is, however, difficult to visualise this mechanism with cortex gushing out of the tear in the capsule.

Perhaps the presence of the anterior capsular cataract offers an explanation. This abnormality is due to a proliferation of the cubical cells which line the anterior capsule, and these cells might have succeeded in closing the wound where a normal epithelium would have failed to do so.

The last, and greatest difficulty is the surprising fact that after the absorption of part of the lens the refraction was finally normal.

At the height of the condition the "mushroom" had about half the size of a normal lens. The lens had suffered a substantial loss of cortex—but the eye remained emmetropic. (No myopia can have existed before the injury as the vision was normal on the day of its occurrence). Only two guesses can be put forward: Either a genuine regeneration of the lens had taken place, or the proliferation of the capsular epithelium during the healing process had given the capsule an additional refractive power.

This latter contention might be supported by the fact that members of certain coloured races—*e.g.*, West Africans and Sudanese—have a definite tendency to an abnormal fibroplastic reaction. Surgeons have noticed that these patients react very frequently with keloids, and it has been observed that corneoscleral trephining in glaucoma is frequently ineffective in members of these races as the trephine opening has a strong tendency to become obliterated by fibrous tissue. The presence of a congenital anterior capsular cataract might be an additional factor producing a proliferation of capsular epithelium.

The attempted explanation of this case seems to override accepted principles. I should be very pleased to hear of a more orthodox solution of this problem.

I wish to thank Lt. Col. J. Amos, R.A.M.C. for his permission to publish this case and Lt. Col. G. I. Scott, R.A.M.C. for his advice.