surrounding mesoderm (precursor of choroid and sclera) with a later extension of the resulting scar tissue."

Johns offered as explanation a failure in development of the papillo-macular bundle the fibres of which would normally occupy the area of the cup. Such an explanation is inconsistent with vision such as that of the present patient, *i.e.* 6/5 (left).

The classification of such cases usually follows that given by Caspar who described three groups.

(1) Cases in which all the vessels are coming from the lower portion of the pseudo-disc, even those which later turn upwards.

(2) Cases in which vessels emerge at or a little above the centre—their arrangement being almost normal.

(3) Cases in which the vessels appear at the circumference of the disc and appear to bend sharply around its edges.

The present case falls into the third group. The diagnostic features are: (1) Enormous size of disc, or what corresponds to disc. (2) Deep cup. (3) Dead white pallor.

The differential diagnosis must include for completeness: (1) Glaucomatous cupping. (2) Cavernous optic atrophy.

The first is excluded by a study of tension and fields. The latter occurs at a much later period.

I am indebted to Prof. Arnold Sorsby for the diagnosis of this case.

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A CASE OF PSEUDO-GLAUCOMA

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CASES showing typical glaucomatous cupping, but without increased intra-ocular pressure, have always aroused general interest. The various authors give them different names: amaurosis with excavation (v. Graefe), glaucoma without hypertension, primary cavernous optic atrophy (Schnabel), but I think Duke-Elder is right when he suggests that these terms should not be used. He calls these cases pseudo-glaucoma.

The following case is of interest because calcification of the internal carotid artery could be shown radiologically.

Robert Henry B., aged 74 years, was first seen on November 23, 1945. He had no complaints of anything in particular, he just wanted new reading glasses.

On examination the vision of the right eye was:—

$$\text{With } \frac{+0.5}{-3.5 \downarrow 90^\circ} = 6/9 \text{ to } 6/6 \text{ (2 letters)}$$

that of the left eye:—

$$\text{With } \frac{-1.75}{-2.75 \downarrow 90^\circ} = 6/9 \text{ (3 letters)}$$

With the addition of +2.50 reading small print (J.2).

RIGHT EYE

FIG. 1.

Both fundi showed deeply cupped discs, which were slightly pale.

The blood pressure was 160/80, and the intra-ocular pressure $\frac{5.5}{6} = 19$ mm. Hg in both eyes. This pressure did not rise after one hour's stay in the dark-room, nor after repeated instillation of homatropine for one hour.

The right field showed marked nasal step, reaching the fixation point, and a typical arcuate nerve fibre bundle scotoma; the left one general peripheral depression. The field for red was, in both eyes, disproportionately smaller than the field for white. It is of the atrophic type (see Fig. 1).

FIG. 2.

See Schema on p. 695, Fig. 2.

The X-ray of the sella shows calcification of the internal carotid artery (see Fig. 2).

The question arises—is this case a glaucoma? The difficulty of the diagnosis lies in the fact that the fundi and the field defects appear to suggest this disease. The radiological finding is, therefore, of great importance, because it furnishes us with a reasonable explanation for the field defect, because the intra-ocular pressure was always found quite normal, even after provocative tests. The calcified internal carotid pressing on each temporal side of the chiasma has caused the nasal contraction, the nerve fibre bundle

FIG. 2.

scotoma being most likely due to atrophic changes in the optic nerve itself.

Pickard pointed out that in glaucoma the fields for white and colours are similar, since all conduction is equally destroyed by pressure, but that in cases of glaucoma without hypertension, due to cavernous optic atrophy, the field for red shows a general contraction not in proportion to the contraction for white objects. The above case shows this disproportion of the fields.

It is difficult to find a reasonable explanation for the cupping of the discs, unless one assumes either a direct damage of the nerve fibres through pressure, of calcified blood vessels, or areas of degeneration and softening within the optic nerve itself, through end-arteritis or spastic contraction of the nourishing blood vessels. If these areas of degenerated nerve fibre tissue are situated just behind the lamina cribrosa the latter will recede, and produce a glaucomatous cupping.

Redslob published in 1941 a number of cases of glaucoma without increased intra-ocular pressure, where the histological examination revealed typical cavernous atrophy of the optic nerve. These cases showed extensive vascular changes, due to venous stasis and Redslob thinks that the vascular upheaval is the responsible cause for the formation of the cavernous atrophy and the glaucomatous cupping.

Weekers, on the other hand, speaks of incomplete glaucoma. He says:—Glaucoma originates chiefly from a change in the intra-ocular blood vessel system. In the case of chronic glaucoma, *i.e.*, idiopathic glaucoma, the vascular alteration affects the uvea, the retina and the optic nerve, causing the three principal symptoms of glaucoma:—increased intra-ocular pressure, narrowing of the field of vision, and glaucomatous cupping of the disc. The three symptoms are usually, but not always, found simultaneously. There is an incomplete glaucoma: in certain cases the increased intra-ocular pressure may be absent. The incomplete glaucoma is observed in grown-up and aged patients, whose vascular system is more or less defective and the affection progresses very slowly. The cupping of the disc may remain isolated for a long time, but may, in course of time, be complicated by field defects. Occasionally hypertension may appear. These accompanying symptoms show that the lesion of the optic nerve must be attributed to glaucoma. There are a series of intermediate stages between monosymptomatic cupping without hypertension and complete glaucoma with ocular hypertension.

Taking all the various points into consideration I feel strongly that the above case does not fall into the category of monosymptomatic glaucoma, but that the calcified internal carotid artery proves a vascular cause. In favour of this are the atrophic type of the field changes, and the perfectly normal intra-ocular pressure, in spite of provocative tests. The field and fundus changes have remained stationary for over one year, a fact stressed by Thiel, who gives these cases a good prognosis.

Summary

A case of pseudo-glaucoma is presented, which shows field changes, deep cupping of the disc, normal intra-ocular pressure, and calcification of the internal carotid artery.

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