PRISCOL AND RETINAL ARTERY OCCLUSIONS*

PRELIMINARY REPORT

BY

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OCCLUSION of the retinal arteries often ends tragically in various grades of visual loss chiefly because it is seen too late for effective treatment. This is especially so in central artery occlusions, where there is sudden, total loss of visual acuity that is seldom overcome, despite heroic treatment. Branch occlusions have the consolation of a retained, perhaps restored, central vision with some degree of visual field.

Few instances of complete or partial recovery of vision after central artery occlusion have been reported in the literature. Levitt (1948) reported a case in a young woman aged 19 years who suffered sudden loss of vision from occlusion of the cilio-retinal artery. This recovered almost completely after treatment which included daily intravenous injections of sodium nitrite for 8 days, followed by full courses of nicotinic acid and erythrol tetranitrite orally for a whole month. Galla (1948) had a case of total obliteration of the central retinal artery in a man aged 65 years. The patient was treated with retrobulbar nicotinamide, with paracentesis and nicotinic acid intravenously and amyl nitrite inhalations; there were no immediate effects, but the vision had improved to 5/12 by the next day. Michaelson (1948) treated a woman aged 41 years who had an occluded macular branch of the inferior temporal artery with visual loss amounting to "counting fingers" at one metre. She was given retrobulbar acetylcholine. In a description of the subsequent changes in the retina, Michaelson stated that the visual acuity 6 weeks later was 6/24 with correcting glasses. Thompson (1948), in a case of central retinal artery occlusion diagnosed 25 minutes after the onset of symptoms and treated with vasodilator drugs, reported a return of normal central vision and visual fields. Thiel (1951) reported five cases of occlusion of the central retinal artery treated with retrobulbar priscol. There was complete recovery in one case, and failure in the remainder was attributed to delay in beginning treatment. Cantrell (1953) reported a case of embolism of the macular branch of the superior temporal artery giving a central scotoma. Retrobulbar priscol was administered and resulted in full recovery of vision. While the retina was being examined the embolism was observed to shift from the first bifurcation to the second and then to disappear as it was carried out towards the periphery.

The present report was suggested by the rather remarkable coincidence of three successive cases seen at the Western Ophthalmic Hospital within 2

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months. They were given retrobulbar priscol with or without amyl nitrite inhalations. Two patients recovered central visual acuity, though with the field defects still present; the third improved subjectively.

Priscol (2-benzyl-4:5-imidazoline hydrochloride)* is one of a series of imidazoline compounds first synthesized in 1937. Its action is sympatholytic and adrenolytic, and whether given orally or parenterally, it produces generalized vasodilatation. Its successes in the field of peripheral vascular disorders prompted its use in retinal vascular conditions (retinal artery occlusions, senile macular degeneration, and retinitis pigmentosa). According to Grimson and others (1947; 1948), the drug acts on the sympathetic nerve terminals in the smooth muscle of the peripheral arteries and arterioles, producing its effect by two mechanisms:

- (i) blockage of the sympathetic vascular receptors,
- (ii) direct histamine-like effect on the small vessels.

When it is given by retrobulbar injection, dilatation of the retinal vessels results, combined with a fall in retinal arterial pressure (Gandolfi, 1947).

When it is given intravenously the results are variable. According to Fanta (1949) intravenous priscol may even produce constriction of the retinal vessels; he advocates a simultaneous combination of retrobulbar priscol with amyl nitrite inhalations.

It has been noticed clinically that the effect on the general blood pressure is very variable; when given intravenously or in very large doses, the blood pressure may be reduced, but in most cases it is unaffected, probably because by its direct action on the heart muscle together with coronary dilatation, the increased cardiac output balances the fall in pressure due to peripheral vasodilatation.

As in other vasodilator therapy, mild side-effects (flushing, feeling of warmth, gooseflesh, nausea, abdominal discomfort, and tachycardia) have been reported; but these are transient, occurring only in the early stages of treatment. In the cases reported here, flushing of the face appeared within a few minutes of treatment, but no other general disturbances were noted.

Case Reports

Case 1, a married woman aged 59 (Fig. 1, overleaf), was seen in the Casualty Department of the Western Ophthalmic Hospital on the morning of October 15, 1953, complaining of sudden loss of vision in the left eye, first noticed at about 10 a.m. that same morning. There was no previous history of visual loss.

Examination.—Visual acuity 6/5 in the right eye, counting fingers at 6 in. in the left eye. Both pupils were active. The right fundus showed marked nipping of veins at arterio-venous crossings, but otherwise nothing abnormal was detected. The left fundus showed a disc normal in colour, and arteries constricted into thin lines with distinct fragmentation of the blood column in the superior temporal artery. No to-and-fro movement was noticed. There was a localized oedema over a broad area on either side of the affected artery and extending nasally to the superior nasal artery. This oedema presented in the form of fine striae extending from the disc outwards. The macula was normal. There

* Manufactured by Ciba Ltd., London.

FIG. 1.—Case 1, left eye. Target 1° white.

was some nipping of the veins at the arteriovenous crossings. Blood pressure 180/100.

Heart within limits, sounds normal, no murmurs.

Blood Wassermann reaction (done subsequently) negative.

Therapy.—At 12.35 p.m., prisol 0.01 g. was given retrobulbarly, followed immediately by an inhalation of amyl nitrite and ocular massage. At 2 p.m. visual acuity in the left eye was 6/60, and at 4 p.m. it was 6/24 (pupil still under mydriatic). The patient was then given one tablet prisol three times daily, and oral nicotinic acid 250 mg. daily in divided doses, and asked to report again. The next day, visual acuity in the left eye was 6/9 (iii). Oral treatment was continued for 2 months.

Result.—On her discharge on December 20, 1953, visual acuity in the left eye was 6/9 (ii), and blood pressure 180/90.

Case 2, a man aged 43 (Fig. 2), was seen on December 2, 1953. He stated that a “curtain” had come over his right eye at about 9.15 p.m. on the previous evening with complete loss of vision. The next morning he found that he could not see at all below the horizontal when looking straight ahead. He also volunteered a history of sudden complete loss of vision about 2 months previously which had lasted about a minute or two.

Examination.—Visual acuity in the right eye 6/36, in the left 6/6. In the right fundus a retinal oedema extended over a wide area from the disc upwards on either side of the two superior arteries; on the temporal side the oedema extended a short distance below the macular branch of the superior temporal artery. Off-shoots from this artery were not visible. The macular area was spared. The disc margin was clearly visible for three-quarters of its circumference, but slightly blurred above. All the arteries were very constricted, and showed as more or less straight lines. There was no nipping of the veins.

In the left fundus nothing abnormal was detected.

Wassermann reaction negative.

Blood pressure 140/100.

Heart, nothing abnormal.

Therapy.—3.50 p.m. retrobulbar prisol 0.025 g., with simultaneous administration of a capsule of amyl nitrite. At 4.30 p.m. visual acuity in the right eye (pupil under mydriatic)

FIG. 2.—Case 2, right eye. Target 1° white.

was 6/36. At 6.30 p.m. (with the pupil still dilated) it was 6/18. The patient was put on priscol and nicotinic acid orally, and the next day the visual acuity had come down to 6/24. He was given a second priscol injection of 0.025 g. retrobulbarly, and an hour later, had reached 6/18 again. His vision gradually improved over the next few days, being 6/9 4 days later after a third injection of priscol.

Result.—The retinal oedema had disappeared by December 14, 1953, and oral treatment was continued until March 5, 1954, when his visual acuity was 6/5.

Case 3, a man aged 71 attended the Western Ophthalmic Hospital on November 23, 1953, with a history of sudden loss of vision in the right eye 7 days before. There was no history of previous visual loss.

Examination.—Visual acuity in the right eye hand movements only; in the left eye 6/6 (iii). The right pupil was inactive to light. The right fundus showed marked retinal oedema with a cherry-red spot at macula. The arteries very irregular in calibre, and the blood column was broken in the superior temporal artery. The veins were full, with nipping at the arteriovenous crossings. The disc margins were blurred.

Blood pressure 180/110.

Heart, nothing abnormal.

Wassermann reaction, negative.

Therapy.—3.40 p.m. 0.025 g. priscol given retrobulbarly. 4.15 p.m.. Subjective improvement only. The patient was sent home on oral priscol and nicotinic acid.

Results.—The next evening the visual acuity in the right eye was 6/60 barely. Oral treatment was continued for a month but no further improvement was seen.

Discussion

Three cases obviously provide insufficient material for dogmatism, but a few points may be stressed.

It appears that an element of spasm is present in most, if not all, cases of occlusion of the retinal arteries from whatever cause; and alleviation of this spasm, even if it has been present for a long period (up to 7 days, in one case

reported here), does result in some restitution of vision. As a corollary, intensive vasodilator therapy should not be withheld in even apparently hopeless cases and should be persisted in for at least a month. Other observers are agreed on this point (Lijo Pavia and Lis, 1947; Levitt, 1948; Hoang-Xuan-Man and Bailliart, 1948). Parental vasodilator therapy, preferably by retrobulbar injection, should be given in the first instance as an initial dose, followed by prolonged treatment with the same type of drug orally. Of these, priscol appears to be the drug of choice by reason of its low toxicity and freedom from side-effects. Furthermore, when given retrobulbarly, it is quite painless, and appears to be free from the complication of ocular palsies associated with a retrobulbar injection of acetyl choline (Payne and Reed, 1954). To produce a heightened effect, a combination of such drugs is recommended, viz. oral nicotinic acid and priscol simultaneously. The only contraindications are peptic ulcer and coronary disease.

Summary

Three cases of occlusion of the retinal artery treated with retrobulbar priscol and amyl nitrite are reported, with a brief review of the relevant literature.

A case is made for an initial retrobulbar injection of the drug, followed by prolonged oral treatment with a combination of vasodilator drugs.

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