PATHOLOGY OF HENLE'S FIBRE LAYER*†‡ AFTER OCCLUSION OF THE CENTRAL RETINAL ARTERY

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THE outer fibre layer of Henle is the foveal portion of the outer plexiform layer of the retina. This layer is composed of the elongated inner processes of the foveal cones and rods which radiate with a peculiar slant away from the foveal pit. These inner processes, thus, represent short neurites connecting the visual cells with their bipolar cells. These latter neurons are shifted towards the retinal periphery and arranged in a circle around the fovea due to its special structure in man.

Nutrition of the outer 120 microns of the human retina is believed to come from the choriocapillaris (Michaelson, 1965). In the foveal region even more of the retina is thought to be supplied from the choroidal side—since the fovea has no retinal blood vessels. Eye pathologists are, therefore, accustomed to see well-preserved outer retinal layers in routine stains even in late stages of complete occlusion of the central retinal artery.

The present study is to present evidence of direct and irreversible damage to human Henle's fibre layer after occlusion of the central retinal artery, using a nerve stain.

Case Report

A 69-year-old white male came to the Veterans Administration Hospital on June 3, 1963, after he had experienced total loss of vision in his left eye the same morning. His history revealed an upper respiratory infection 6 weeks before that had been treated with intramuscular penicillin. Vision in both eyes had been normal until the night before.

Examination.—He was obese with a blood pressure of 176/104 and a corrected erythrocyte sedimentation rate of 21 mm. in 1 hour. Laboratory tests included a negative Bentonite flocculation, total protein 7.0 g. per cent., muric acid 3.7 mg. per cent., white blood count 7,000, haematocrit 46 per cent., haemoglobin 14.1 g.

Urinalysis revealed a 2+ albuminuria, specific gravity 1.024, and creatinine 1.07 mg. per cent.

Blood urea nitrogen 20 mg. per cent., fasting blood sugar 78 mg. per cent.

Chest x rays revealed mild pulmonary emphysema and x rays of the hands showed hypertrophic arthritis.

The patient's right eye was entirely normal and had 20/20 vision. The left eye exhibited superficial as well as deep pericorneal injection. The pupil was irregular in shape and there was no direct reaction to light. Movements of the eye were painful and especially the upper sclera was tender to touch. Slit-lamp examination revealed slight corneal oedema, wrinkling of Descemet's membrane, 3+ flare, inferior hyphaema, and a posterior synechia at 1 o'clock. Gonioscopy showed the angle to be open and filled with blood. Fundus examination showed complete occlusion of the central retinal artery, retinal oedema, and a cherry red spot in the foveal region.

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Treatment.—Systemic prednisolone 10 mg. four times a day, with local atropine 1 per cent. four times a day, and local steroid eye drops four times a day, was started. However, the eye became increasingly painful.

On June 7, 4 days after admission a complete hyphaema developed spontaneously in the left eye and the pain became even more severe. The patient asked for removal of the painful blind eye, and enucleation was carried out on June 13. The eye was fixed immediately in neutral formalin.

Histopathological Findings.—The eye was of normal size and the anterior chamber was filled with blood. The main branches of the occluded central retinal artery were seen as white branching streaks on the swollen optic disc (Fig. 1).

FIG. 1.—Opened eye with white arterial branches on optic disc and cherry red spot in the fovea (arrow).

Microscopic study of routine paraffin sections showed a normal cornea and confirmed the presence of blood in the anterior chamber. The iris exhibited diffuse infiltration with lymphocytes and plasma cells. Extensive rubeosis iridis was seen on the anterior iris surface. The lens was normal and the ciliary body contained diffuse infiltration with mononuclear inflammatory cells. Routine sections of the retina showed total atrophy of the nerve fibre and ganglion cell layers. Special staining of the neurons with the silver carbonate techniques of Hortege (Scharenberg and Zeman, 1952) confirmed the necrosis of all inner retinal neurons. Centrifugal nerve stumps were found surviving on the optic disc (compare Wolter, 1965). The outer retinal layers appeared normal in routine sections. The pigment epithelium was normal except for some peripheral senile changes. Diffuse infiltration with few lymphocytes was found in the choroid. The sclera itself was normal. However, lymphocytic infiltration was observed in the perilimbal region and in a more diffuse arrangement in the episclera of its anterior portion.

Special nerve stains (Scharenberg and Zeman, 1952) of flat sections of the central retina revealed advanced pathological changes in Henle's fibre layer. The inner processes of the central visual cells had lost their normal regular radial arrangement (compare Wolter, Goldsmith, and Phillips, 1957). These processes were in this case of an irregular wavy course and there were many loops, interruptions, thickenings, and dark-staining terminal bodies (Figs 2 to 6, opposite and overleaf). The neurites also exhibited irregular size and club-shaped swellings within their course as well as next to their interruptions (Figs 2 and 3). Well-developed terminal bodies of neurite stumps were seen in some areas (Figs 4 and 6). More complicated structures of distorted and swollen neurites were seen everywhere (Figs 4, 5, and 6). Occasional microglia in the process of phagocytosis was found in Henle's fibre layer (Fig. 2). The cell bodies of the visual cells in the foveal region were normal as far as we could see in our flat sections. The pigment epithelium, Bruch's membrane, choriocapillaris and choroid of the central region also appeared normal.

Comment

The outer layers of the retina—including Henle's fibre layer—are believed to derive their nutrition from the choriocapillaris (Michaelson, 1965). Advanced degenerative changes

FIG. 2.—Flat section through Henle's fibre layer in the fovea, showing neurites of irregular course and with interruptions as well as round terminal bodies. Arrow indicates phagocytosing microglia.—Frozen section, Hortega stain, photomicrograph $\times 750$.

FIG. 3.—Club-shaped stump of a neurite in Henle's fibre layer (arrow) as well as other abnormal neurites.—Frozen flat section, Hortega stain, photomicrograph $\times 750$.

FIG. 4.—Round terminal bulbs of interrupted neurites (arrows) and some more bizarre formations of degenerating neurites in Henle's fibre layer.—Frozen flat section, Hortega stain, photomicrograph $\times 750$.

FIG. 5.—Swollen neurites with bizarre degenerative formations and interruptions in Henle's fibre layer.—Frozen flat section, Hortege stain, photomicrograph $\times 750$.

FIG. 6.—Bizarre formation of swollen degenerating neurites in Henle's fibre layer (arrow).—Frozen flat section, Hortege stain, photomicrograph $\times 750$.

and interruption of the neurites of Henle's fibre layer, observed in the present case 10 days after complete occlusion of the central retinal artery, thus represent a most interesting finding. It has to be emphasized that vision had been normal before the arterial occlusion, and that the choriocapillaris, Bruch's membrane, and pigment epithelium were normal in this eye. The presence of painful episcleritis and uveitis as well as rubeosis iridis and hyphaema was an unusual feature of the condition in the present case.

Little is known of the reactions of Henle's fibres in retinal disease. The question arises, however, whether the retinal degeneration in this case could be a direct result of ischaemia in Henle's fibre layer caused by interruption of the retinal blood flow.

Summary

Degeneration and interruption of neurites in Henle's fibre layer were observed 10 days after occlusion of the central retinal artery.

REFERENCES

- MICHAELSON, I. C. (1965). Address of invited foreign guest, Section on Ophthalmology, A.M.A. Annual Convention, New York, June, 1965.
 SCHARENBERG, K., and ZEMAN, W. (1952). *Arch. Psychiat. Nervenkr.*, **188**, 430.
 WOLTER, J. R. (1965). *Trans. Amer. ophthal. Soc.*, **63**, 678.
 ———, GOLDSMITH, R. I., and PHILLIPS, R. L. (1957). *A.M.A. Arch. Ophthal.*, **57**, 376.