Brit. J. Ophthal. (1967) **51**, 688

RETINAL ARTERIOLAR COLLATERALS IN MAN*†‡

BY

C. J. BALL AND P. HENKIND

From the Department of Ophthalmology, New York University School of Medicine, New York, N.Y.

ARTERIOLAR collaterals can form within the retinal vascular bed of man and animals although, in contrast with their venous counterpart, they are rare. Only some twenty reports of their occurrence are extant, as well as three papers dealing with their production experimentally. The following case is presented as an example of unusually extensive retinal arteriolar collaterals in which anastomotic vessels attained the size and appearance of normal arterioles. These vessels appeared not to be influenced by the patient's severe hypertension, which had caused marked sclerotic changes and irregular narrowing in the normally distributed arterioles.

Case Report

A 31-year-old white male had severe hypertension of 16 years' duration and resulting renal insufficiency and congestive heart failure. Blood pressures at the time of the ocular examinations were 194–240 mm. Hg systolic and 130–140 mm. Hg diastolic. The patient also suffered from grand mal seizures.

Visual symptoms began approximately at age 20 when the patient noted an absolute left hemianopic field defect in the right eye. Thereafter, at intervals of 1 or 2 months, the scotoma abruptly enlarged to his right, momentarily reducing the visual field to a small area in the far temporal periphery. At similar intervals a dense scotoma appeared centrally before the left eye, enlarging slightly at first, then gradually disappearing. The visual symptoms of the two eyes were not related, nor was there any relation to the seizures.

The corrected visual acuity was 20/20–3 in the right eye and 20/20–2 in the left. The intra-ocular pressure and ophthalmodynamometry readings were normal.

The findings of major interest were in the right fundus. The optic nerve head showed loss of substance temporally. The reflex from the walls of the nasal arterioles was increased, giving a burnished appearance. On one occasion slowing of the blood column with "box-carring" was seen in the superior nasal arteriole with sludging of blood in the adjacent venule. This lasted only seconds and could not be correlated with subjective symptoms. Most remarkable were the temporal arterioles and their branches. Fig. 1 shows a composite fundus photograph and Fig. 2 a diagram of the arterioles, indicating their connexions and empty segments as determined by direct ophthalmoscopy, fundus photography, and fluorescein photography. Both the superior and inferior temporal arterioles arose from the respective main trunks of the central retinal artery as bloodless non-fluorescing translucent white bands, through which the edge of the optic disc and underlying veins could be easily seen. Distal to these empty vessels, numerous inter-arteriolar anastomoses were present. These, except for their tortuous course and short stretches of irregular calibre, appeared normal (in contrast with the nasal arterioles of this eye, and the arterioles of the

* Received for publication July 5, 1966.
† Address for reprints: 550 First Avenue, New York City, 10016.
‡ This work was supported in part by Grant NB 05059–01 of the United States Public Health Service.

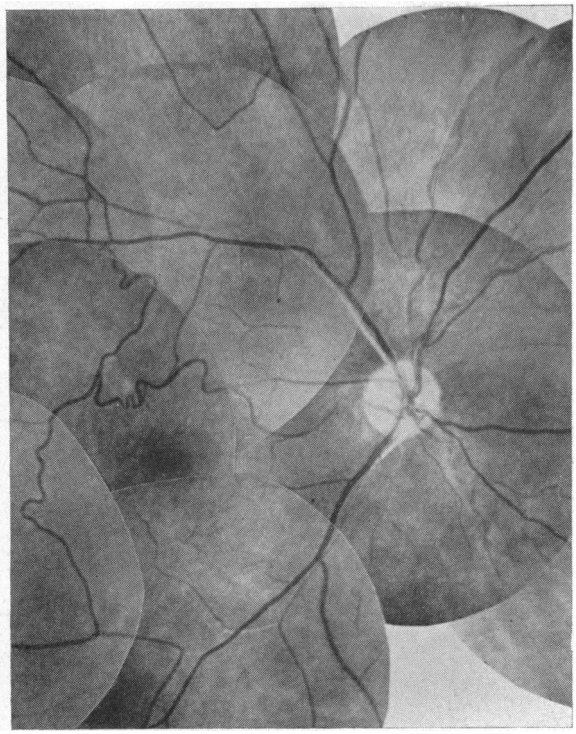

FIG. 1.—Composite photograph of right fundus, showing empty (white) segments of superior and inferior temporal arterioles and the arteriolar collaterals.

FIG. 2.—Diagram of arterioles seen in Fig. 1. The dotted lines indicate empty segments. (Veins are omitted.)

left eye). A third bloodless segment arose at a bifurcation of the inferior temporal arteriole and continued only to the next bifurcation, beyond which the arteriole was filled *via* collaterals.

The blood supply of these temporal vessels apparently came largely from anastomoses with a branch of the superior nasal arteriole, and *via* small branches from the upper and lower trunks of the central artery, arising proximal to the onset of the occluded segments. Apart from these vascular changes, the fundus appeared normal save for some mild pigmentary mottling at the macula.

In the visual field of the right eye was an absolute temporal hemianopic defect, sparing the macula (Fig. 3, overleaf). The nasal boundary could not be defined clearly, because of unsteady fixation on the part of the patient and an apparent variability in its location. A similarly variable scotoma extended from the hemianopic defect temporally below the macula, and a small fixed sloping-edged scotoma was present above the blind spot. The spatial correlation of the hemianopic defect with the abnormal retinal circulation is obvious, but that of the tongue-like extension and the isolated scotoma is not. The retinal area generally corresponding to the isolated scotoma is, however, supplied by a branch connected with the empty inferior temporal arteriole. Thus, this scotoma may be related to barely adequate flow in this region.

Discussion

The first report of retinal arteriolar collaterals in man was that of Hock (1869), and some twenty cases have since been recorded (Henkind, 1966). Recently, similar

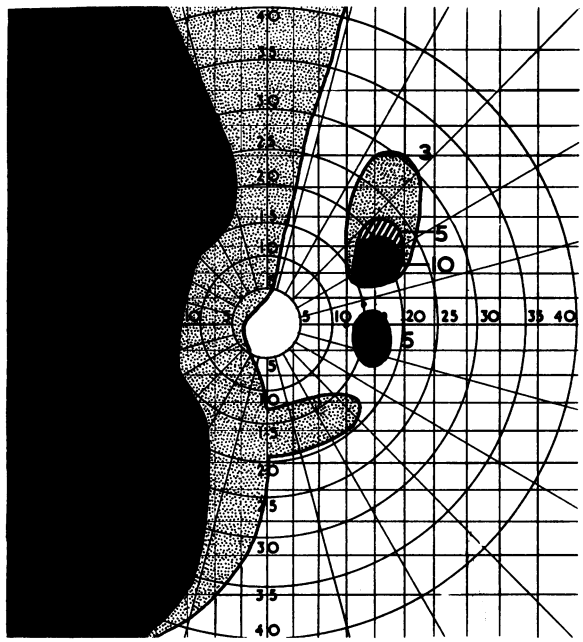

FIG. 3.—Central field of right eye. Temporal border of hemianopic scotoma varied over the dotted area. Numbers refer to 3, 5, and 10/1000 white test objects.

channels have been produced experimentally by embolism of the retinal arteriolar tree (Ashton and Henkind, 1965). These collaterals, in man and animals, are almost exclusively inter-arteriolar; arteriovenous communications are rare sequelae of arteriolar occlusion although they may be fairly common in other disorders, such as diabetes (Cogan and Kuwabara, 1963) and retinal vein occlusion (Klien, 1966). This finding is consistent with observations made on collateral circulation in other organs after arterial occlusion: in most instances collateral artery joins artery, and vein links vein, and arterio-venous communications are uncommon (Liebow, 1963). In the only pathological study of human material, arterio-arteriolar channels were found in a retinal digest preparation from an elderly patient with a long-standing branch artery occlusion (Kornzweig, Eliasoph, and Feldstein, 1964).

A full discussion of collateral vascular channels is provided by Liebow (1963), and we can only add some data pertinent to the retinal circulation. In the majority of affected patients emboli, which blocked retinal arterioles and led to subsequent formation of collateral channels, apparently arose secondary to valvular heart disease. In a case reported by Jensen (1938), emboli probably came from the carotid artery. In our patient, who had severe hypertensive cardiovascular disease, it was unknown whether the retinal vascular occlusion was due to embolic or thrombotic phenomena, the episode having occurred more than a decade before examination.

It has been shown experimentally that acute occlusion of branch retinal arterioles leads to the formation of large collaterals which develop without exception from the pre-existing capillary bed (Dollery, Henkind, Paterson, Ramalho, and Hill, 1965; Henkind, 1966). Arteriolar collaterals have appeared hours to days after glass bead embolism of the retinal vessels in cats, and after slightly longer periods in pigs (Dollery, Henkind, Paterson, Ramalho, and Hill, 1966; Henkind, 1966; Shakib and

Ashton, 1966). In man the earliest reported arterial anastomosis was seen 7 days after a branch arterial occlusion (Barkan, 1902).

The collaterals in both animals and man may enlarge to the size of the occluded parent vessel, and in retinal digest preparations appear identical in cellular structure with normal arterioles of comparable size (Kornzweig and others, 1964; Henkind, 1966). We feel that the collaterals found in our patient developed from pre-existing retinal capillary channels which, after blockage of the patent vessels, were modified by altered flow and pressure and possibly by other unknown factors, so that arterialized thoroughfare channels were formed.

Although their paths were more tortuous than normal, these arterioles had the diameter, colour, and light reflex usually associated with normal retinal arterioles.

Some questions of great theoretical interest are raised by the fact that the collateral arterioles appeared normal, whereas the rest of the retinal arteries in both eyes were sclerosed and irregular because of hypertension. Are these collaterals incapable of reacting to the same stimulus as that which causes other arterioles to contract, or are they not being subjected to the stimuli producing such changes in the "normal" channels?

In the retina it appears that vascular constriction may result more from intrinsic mechanisms, *i.e.* vascular tonus due to intraluminal pressure, than from extrinsic innervation. Possibly the pressure within the collaterals is lower than in other arterioles. The collaterals' connexion with sclerosed narrowed arterioles, probably carrying reduced blood flow, is suggestive evidence for this point. Also, the fact that the temporal arteries distal to their points of occlusion are of essentially normal calibre strengthens the view that narrowing of the arterioles depends to some extent on intravascular pressure. However, until more is known about retinal collaterals, and indeed about retinal vessels, this is in the realm of speculation.

Summary

A case of extensive arteriolar collateral formation in the retina of a young man with severe hypertensive cardiovascular disease is presented. A brief review of retinal arteriolar collaterals is provided.

REFERENCES

Ashton, N., and Henkind, P. (1965). *Brit. J. Ophthal.*, **49**, 225.
Barkan, A. (1902). *Arch. Ophthal. (N.Y.)*, **31**, 1.
Cogan, D. G., and Kuwabara, T. (1963). *Diabetes*, **12**, 293.
Dollery, C. T., Henkind, P., Paterson, J. W., Ramalho, P. S., and Hill, D. W. (1966). *Brit. J. Ophthal.*, **50**, 285.
———, Ramalho, P. S., Paterson, J. W., Hill, D. W., Henkind, P., Shakib, M., and Ashton, N. (1965). *Lancet*, **1**, 1303.
Henkind, P. (1966). *Brit. J. Ophthal.*, **50**, 482.
Hock (1869). *Wien. med. Presse*, **10**, 1036 (*Quoted by* Jensen, 1938).
Jensen, V. A. (1938). *Acta ophthal. (Kbh.)*, **16**, 485.
Klien, B. A. (1966). *Amer. J. Ophthal.*, **61**, 25.
Kornzweig, A. L., Eliasoph, I., and Feldstein, M. (1964). *Arch. Ophthal. (Chicago)*, **71**, 542.
Liebow, A. A. (1963). "Handbook of Physiology", vol. 2, sect. 2, ch. 37. American Physiological Society, Washington.