

RADIAL PERIPAPILLARY CAPILLARIES OF THE RETINA*†‡

II. POSSIBLE ROLE IN BJERRUM SCOTOMA

BY

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DESPITE the importance of the Bjerrum scotoma in the clinical management of glaucoma, there has been no generally acceptable explanation of its pathogenesis. Mechanical, toxic, and vascular theories have been proposed, and considerable evidence has accumulated in favour of the last:

(a) Reese and McGavie (1942) found that resistance to visual field loss in glaucoma was directly related to the ratio of systemic blood pressure to intra-ocular pressure;

(b) McLean (1957) and Harrington (1959) reported sudden progression of field loss in hypertensive glaucoma patients who were effectively treated with antihypertensive agents;

(c) Harrington (1965) reported a series of 45 patients who did not have glaucoma, but who did have field loss in the 10 to 20 degree isopters typical of the Bjerrum scotoma. Most of those which could not be explained by congenital, inflammatory, or space-occupying lesions, had a vascular basis;

(d) Gafner and Goldmann (1955), and later Drance (1962), showed that transient experimental elevation of intra-ocular pressure in normal persons could cause a transient Bjerrum scotoma;

(e) Cristini (1951) found decreased capillary vascularity in histological sections of the optic nerve and uvea in 45 cases of glaucoma;

(f) Gafner and Goldmann (1955) constructed an electronic model based on the anatomical findings of François and Neetens (1954), and suggested that increased intra-ocular pressure would result in anaemia of the optic nerve head;

(g) François, Neetens, and Collette (1959) showed by perfusion studies that elevation of intra-ocular pressure could reduce capillary filling in the area of the lamina cribrosa. Examination of a thorium dioxide injected human specimen showed that retinal capillary filling was decreased in the peripapillary region, particularly on the temporal side (François and Neetens, 1964).

(h) Kalvin, Hamasaki, and Gass (1966) showed that retinal vascular filling could vary with induced intra-ocular pressure in monkeys.

Most authors have postulated that the site of the lesions causing the Bjerrum scotoma is in the optic nerve head or behind the lamina cribrosa. In these areas, involved nerve fibre bundles are anatomically discrete. However, no anatomically discrete vascular supply to the bundles has ever been demonstrated.

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In the retina, the involved nerve fibres are distinct and arch from the superior and inferior temporal aspects of the nerve head. In this same distribution is a specific capillary layer, the radial peripapillary network, mentioned as occurring in the human retina by Michaelson (1954) and Toussaint, Kuwabara, and Cogan (1961), and subsequently described in monkey, pig, and cat by Henkind (1967).

When compared to the usual irregular mesh-like pattern of the retinal capillary tree, these radial capillaries are longer and straighter. They lie in a two-dimensional network in the nerve fibre layer, superficial to the three-dimensional interlacing network of the remainder of the retina. Communications do exist between the capillaries of this network, and with the underlying one, but these are few.

The pattern of these vessels suggested that they might be more vulnerable to pressure elevation than other retinal capillaries, and a study was initiated in cats to test this assumption. The radial peripapillary capillary network while present in the cat is less prominent than in man (Fig. 1).

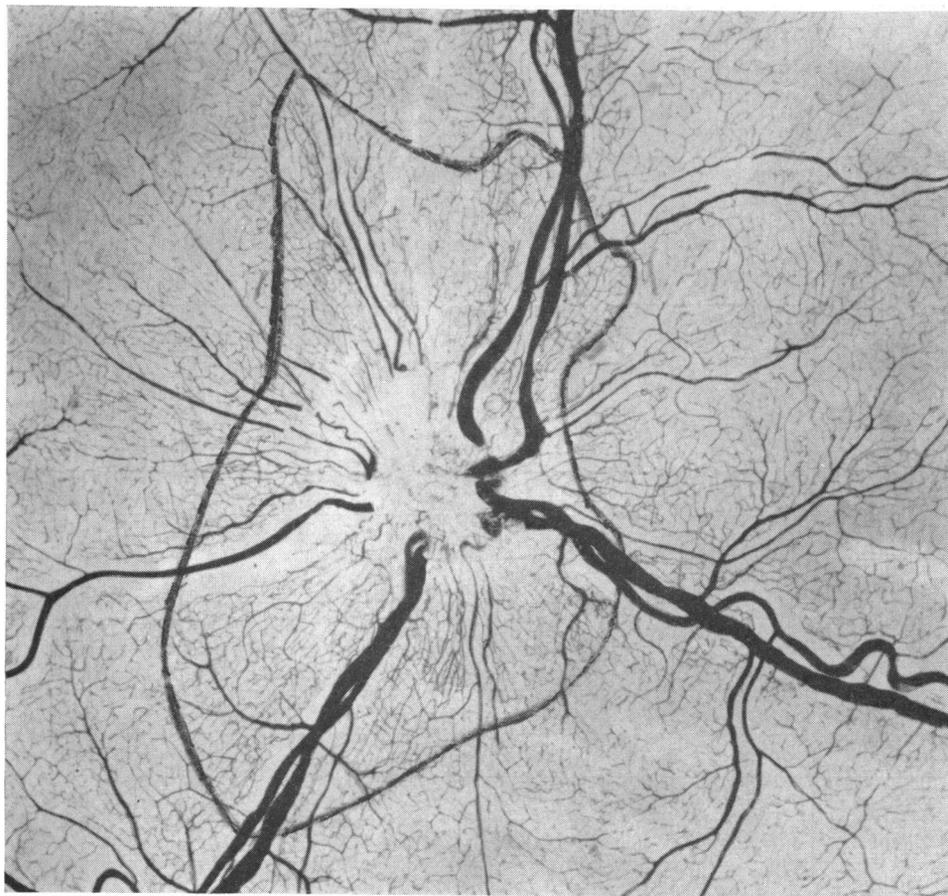


FIG. 1.—Peripapillary region of the right eye of a cat. The radial capillaries are prominent around the superior and inferior temporal vessels (outlined). India ink. $\times 20$.

Material and Methods

Ten adult cats were anaesthetized with intramuscular or intravenous Nembutal. In nine, the anterior chamber of one eye was cannulated with a No. 25 needle attached to a saline reservoir of variable height so that induced intra-ocular pressure could be regulated. Intra-ocular pressure (ranging from 30–50 mm. Hg)* was monitored with a mercury manometer either *via* a sidearm from the reservoir, or with a second intracameral cannula. The intra-ocular pressure elevation was maintained for 20 to 30 minutes and India ink injection was then carried out, in two cases *via* left ventricular puncture and in the rest *via* bilateral carotid cannulae. In all but one instance the perfusion reservoir was kept at 160 cm. saline (120 mm. Hg).

After inking the eyes were removed and fixed in formalin for at least 24 hours before being opened. The retinae were then removed, mounted flat on microscope slides with glycerine jelly, and examined for completeness of filling. The normal eye was used as a control in each case inasmuch as inking could be variable from one animal to the next.

Results (Table)

In three animals (Nos 2, 3, 9) there was markedly less filling in the treated eye. One of these was perfused after anaesthetic death (No. 9).

In six animals, a minimal but definite decrease in filling of the treated eye was observed. In five of these, the difference was most marked around the disc.

* The mean value of normal intra-ocular pressure in the cat is 16.5 mm. Hg: Davson, 1962.

TABLE
RESULTS OF INKING RETINAL VESSELS IN CATS WITH EXPERIMENTALLY ELEVATED OCULAR TENSION

Animal No.	Intra-ocular Pressure in Treated Eye (mm. Hg)	Difference in Inking between Two Eyes		
		Minimal	Marked	Peripapillary Selectivity
1	35*	x		x
2	40		x	
3	35		x	
4	38	x		x
5	30	x		x
6	40	x		x
7	50	x		x
8	30	x		x
9†	40		x	
10	40	x		x

* Idiopathic raised ocular tension.

† Animal perfused after anaesthetic death. Raised intra-ocular pressure maintained.

In one animal (No. 10) the only significant filling difference was in the area around the disc (Figs 2 and 3).



FIG. 2.—Animal No.10 : control eye. Disc and superior temporal retina. Radial capillaries are easily seen. India ink. $\times 12$.



FIG. 3.—Animal No. 10: treated eye. Disc and superior temporal retina. Almost no filling of radial capillaries. India ink. $\times 12$.

Of particular interest was the perfusion of animal No. 1. Intra-ocular surgery had been performed on the right eye 2 weeks previously. In preparation for perfusion, Schiötz tonometry was done for the first time. By a stroke of uncommon good fortune, pressure in the unoperated left eye was 35 mm. Hg, and in the right 16 mm. Hg. The animal was perfused with India ink without further treatment. Decrease in filling of the peripapillary capillaries of the eye with raised pressure was grossly evident (Figs 4 and 5, overleaf).

Comment

The results of these India ink perfusions lend support to the idea that the radial peripapillary network may be more vulnerable to elevated intra-ocular pressure than other retinal capillaries. In animal No. 1, intra-ocular pressure had been raised for an unknown period of time in the left eye. The reduction in capillary filling is sufficiently marked to suggest that anatomical as well as functional changes had taken place.

Wolff and Penman (1951) demonstrated that nerve fibres from peripheral ganglion cells lie deeply in the nerve fibre layer, while those nearer the nerve head occupy an ever more superficial position. The most superficial nerve fibres, those in the area of the radial peripapillary vascular network, would subserve the pericentral field of vision (corresponding to the Bjerrum scotoma). If this be the case, the function of the radial peripapillary capillaries would most likely be to supply the nerve fibre layer in its distribution. If in man these capillaries are indeed compromised by a rise in intra-ocular pressure, the clinical reflection of the physiological abnormality

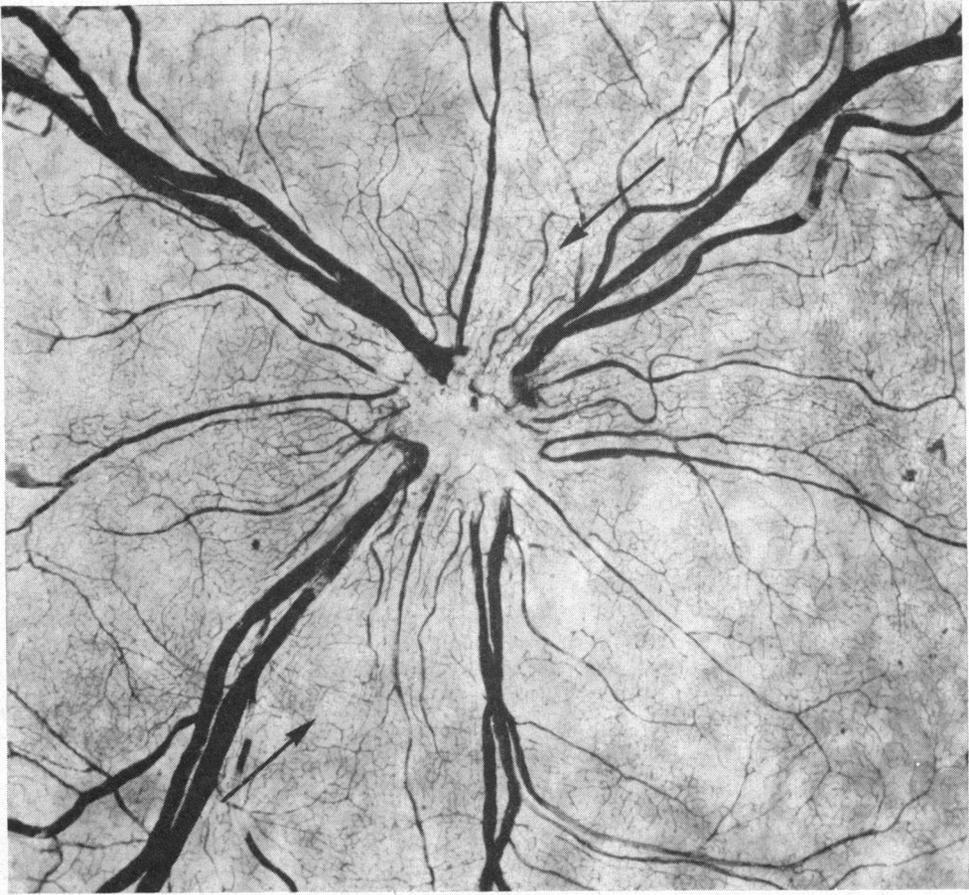


FIG. 4.—Animal No. 1: peripapillary region of right (normotensive) eye. Peripapillary vessels are richly filled (arrows). India ink. $\times 20$.

would be the Bjerrum scotoma. While functional and anatomical changes in capillaries of the optic nerve head or in the optic nerve behind the lamina cribrosa have been shown previously in glaucoma, this does not argue against the idea that Bjerrum scotoma is retinal in origin.

Summary

A distinct capillary structure, the radial peripapillary network, exists in the retina, its apparent function being to supply the peripapillary superficial nerve fibre layer. Its pattern and distribution suggest that underfilling of constituent capillaries in glaucoma may cause the Bjerrum scotoma. India ink perfusion after induced intraocular pressure elevation in cats showed selective underfilling of these peripapillary capillaries.

REFERENCES

- CRISTINI, G. (1951). "XVI Conc. Ophthal. 1950 Britannia Acta", vol. 2, p. 865. B.M.A., London.
 DAVSON, H. (1962). "The Eye", vol. 1, p. 160. Academic Press, New York.

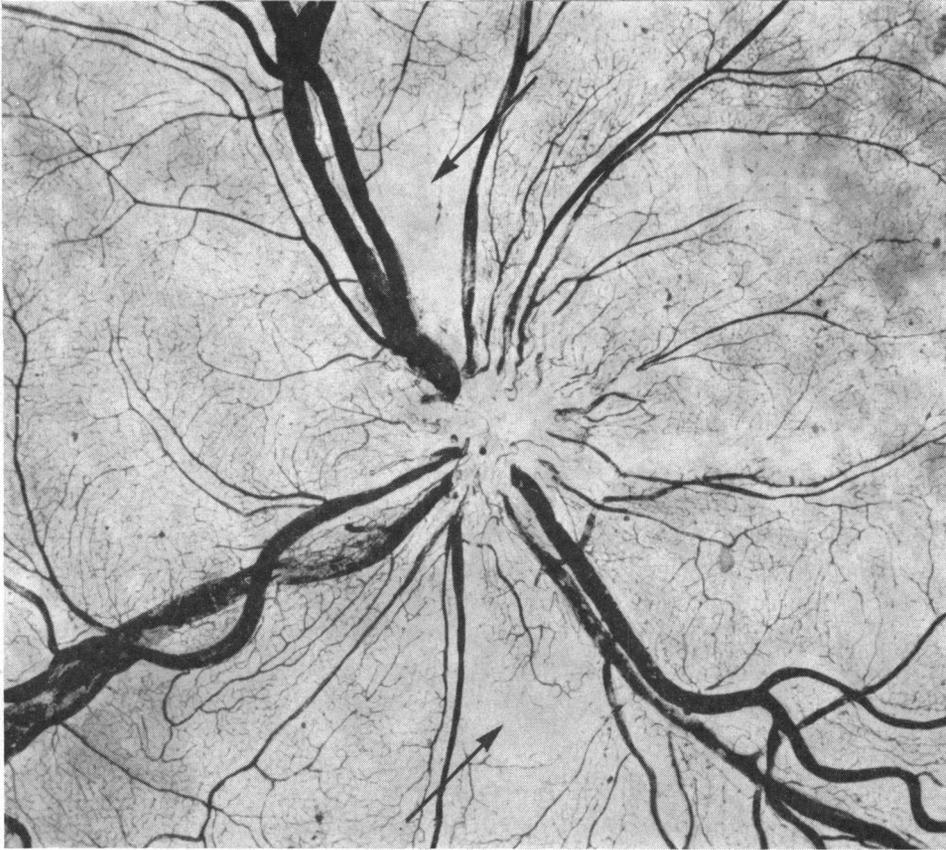


FIG. 5.—Animal No. 1: peripapillary region of hypertensive eye. Capillary filling is diminished inferiorly and absent superiorly (arrows). India ink. $\times 20$.

- DRANCE, S. M. (1962). *Arch. Ophthalm. (Chicago)*, **68**, 478.
- FRANÇOIS, J., and NEETENS, A. (1954). *Brit. J. Ophthalm.*, **38**, 472.
- (1964). *Arch. Ophthalm. (Chicago)*, **71**, 219.
- , ———, and COLLETTE, J. M. (1959). *Acta ophthalm. (Kbh.)*, **37**, 395.
- GAFNER, F., and GOLDMANN, H. (1955). *Ophthalmologica (Basel)*, **130**, 357.
- HARRINGTON, D. O. (1959). *Amer. J. Ophthalm.*, **47**, no. 5, pt 2, p. 177.
- (1965). *Ibid.*, **59**, 646.
- HENKIND, P. (1967). *Brit. J. Ophthalm.*, **51**, 115.
- KALVIN, N. H., HAMASAKI, D. I., and GASS, J. D. M. (1966). *Arch. Ophthalm. (Chicago)*, **76**, 94.
- MCLEAN, J. M. (1957). *Amer. J. Ophthalm.*, **44**, 323.
- MICHAELSON, I. (1954). "Retinal Circulation in Man and Animals". Thomas, Springfield, Ill.
- REESE, A. B., and MCGAVIC, J. S. (1942). *Arch. Ophthalm. (Chicago)*, **27**, 845.
- TOUSSAINT, D., KUWABARA, T., and COGAN, D. G. (1961). *Ibid.*, **65**, 575.
- WOLFF, E., and PENMAN, G. G. (1951). "XVI Conc. Ophthalm. 1950 Britannia Acta", vol. 1, p. 625. B.M.A., London.