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Communications

Occlusion of the posterior ciliary artery

I. Effects on choroidal circulation

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The literature contains no reference to the effects *in vivo* of occlusion of the posterior ciliary artery (PCA) on the choroidal circulation. It has simply been assumed that occlusion of one or more PCAs is not likely to produce any filling defect in the choroid because the choroidal vascular bed has been described as being one continuous bed, with no segmental distribution (Nicholls, 1938; Vilstrup, 1952; Wybar, 1954a, b; Correia, 1957; Scullica, 1957; Ruskell, 1961; Ring and Fujino, 1967). Our experimental studies involving occlusion of the PCAs in rhesus monkeys have, on the contrary, revealed that such an assumption is entirely incorrect and that the distribution of the PCAs is, in fact, segmental. These studies have also led to many interesting observations which are reported below.

There is a good deal of confusion as to the nomenclature, number, origin, and distribution of the PCAs. One of us has helped to clarify these subjects (Hayreh, 1962, 1964, 1970, 1971). Briefly, the ophthalmic artery in humans gives out one (in 3 per cent.), two (in 48 per cent.), or three (in 39 per cent.) PCAs. Each artery divides into multiple branches before piercing the sclera, medial (by the medial PCA) or lateral (by the lateral PCA) to the optic nerve. Of these branches, two small ones (one on the medial and the other on the lateral side) are called the long PCAs, while the rest are the short PCAs.

Material

The study was carried out in 85 rhesus monkey eyes.

Methods

By lateral orbitotomy, the PCAs were cauterized near their site of entry into the eyeball, leaving a small arterial stump close to the globe as follows:

Lateral PCAs (LPCAs) in 31 eyes
Medial PCAs (MPCAs) in 17 eyes
All PCAs (APCAs) in 37 eyes

The Table (overleaf) shows the follow-up period after PCA occlusion in 85 eyes of rhesus monkeys. The choroidal circulation in these eyes was assessed by repeated intravenous fluorescence angiography (IVFA).

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Table I *Follow-up period after PCA occlusion in 85 eyes of rhesus monkeys*

Period of follow-up	*Occlusion*		
	LPCA	*MPCA*	*APCA*
Up to 2 hrs	19	6	15
Up to 1 wk	4	1	2
2–3 wks	3	4	7
6–7 wks	1	2	6
About 3 mths	3	4	7
Over 1 yr	1	Nil	Nil
Total number of eyes	31	17	37

In thirty eyes (21 followed up for less than 2 hrs; 9 for up to 3 months), at the end of the experiment, the carotid vascular tree was irrigated with 2 per cent. gluteraldehyde *via* the left ventricle. This was followed by irrigation of the vascular tree with normal saline. Silicone rubber was then injected *via* the common carotid artery to perfuse the ocular vascular bed. The animal was stored in the deep freeze for 24 hrs or longer to "set" the silicone rubber, after which the eye and the optic nerve were removed and cleared, using the alcohol-methyl-salicylate clearing technique. The choroidal vasculature filling pattern was studied under the dissection microscope. Later, the choroidal pigment was bleached, by the potassium-permanganate-oxalic acid technique, for further studies.

Observations

(A) INTRAVENOUS FLUORESCENCE ANGIOGRAPHIC (IVFA) STUDIES

In all these studies of choroidal filling done by IVFA, only the posterior pole and the area up to a short distance temporal to the macula were investigated. It is not possible, therefore, to comment on the filling of more peripheral regions during the transit of the dye. The findings are described separately under three headings, as follows:

(1) After LPCA occlusion.

(2) After MPCA occlusion.

(3) After APCA occlusion.

(1) *After LPCA occlusion*

During the dye transit, the extent of filling of the choroid up to the end of the retinal venous phase was as follows at different intervals after occlusion:

(*a*) *About 1 hour after occlusion* (*Fig. 1a, b*) Only the area supplied by the MPCA filled, this varying from one-third to one-half of the visible fundus. The area was nasal to the optic disc and included the nasal peripapillary choroid (PPC). In about one-third of these eyes, the unfilled PPC usually filled during the retinal venous phase of circulation. In addition, isolated patches of choroidal filling were seen, as discussed below.

(*b*) *1 to 2 days after occlusion* (*Fig. 1c*) The filling extended temporally, but only for a short distance compared to (*a*) above.

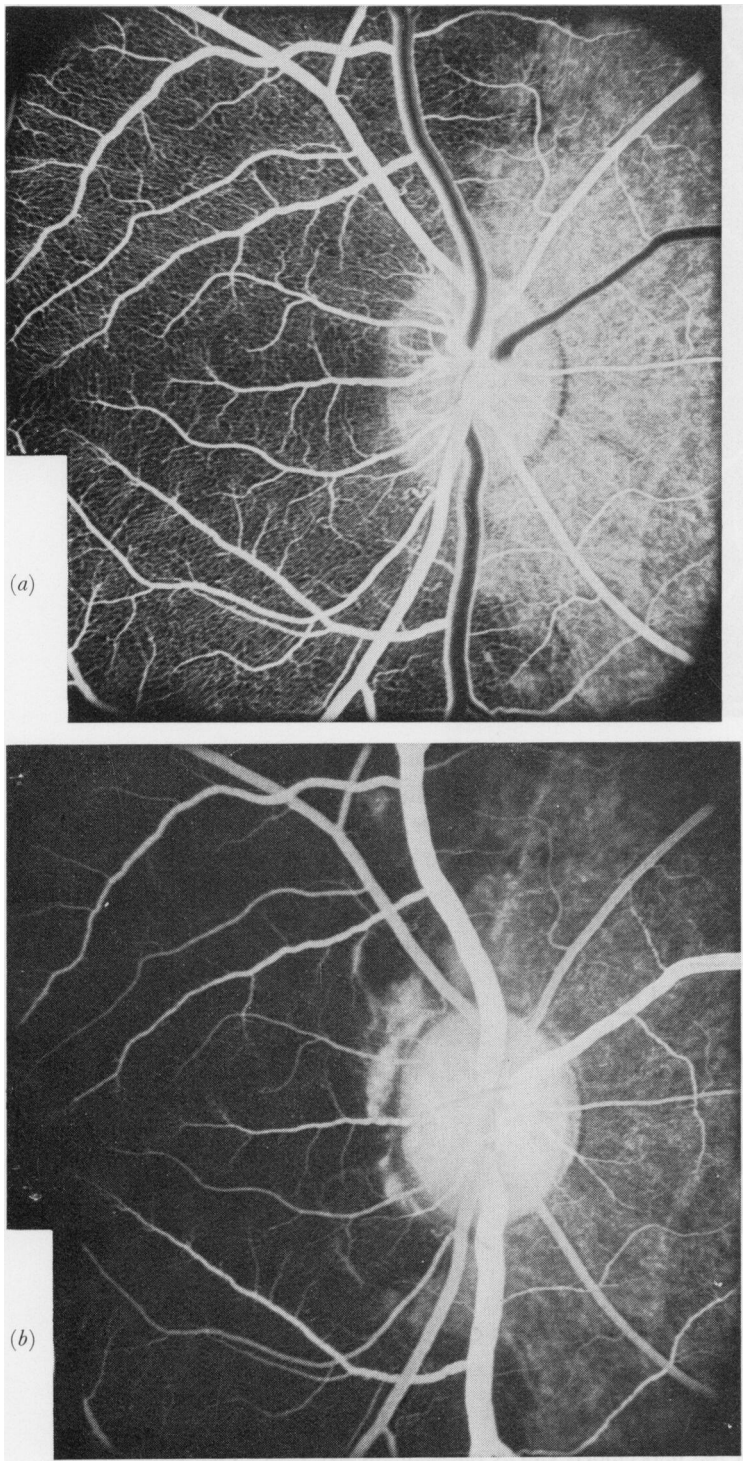


FIG. 1 *Angiograms after LPCA occlusion:*
(*a*) and (*b*) 1 hr after occlusion during retinal (*a*) arterio-venous and (*b*) venous phases

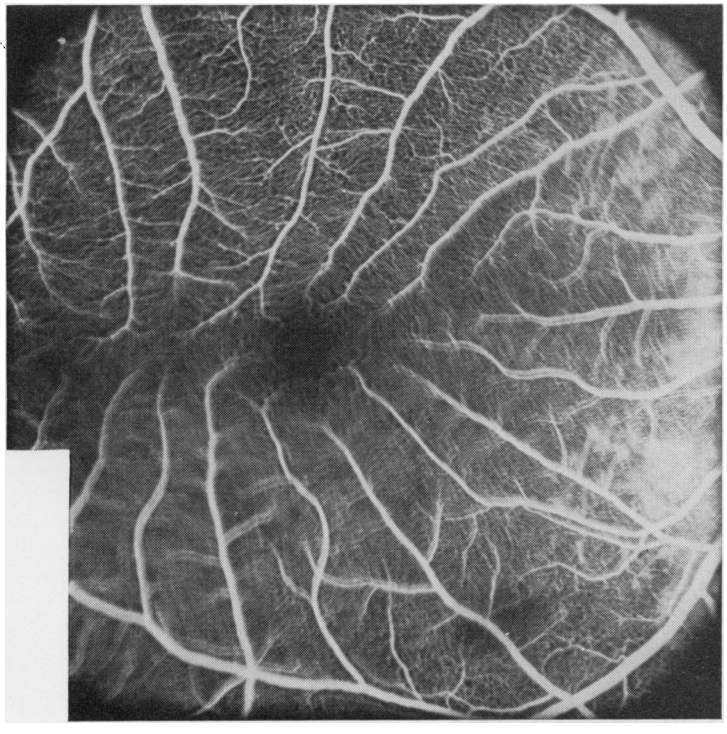


FIG. 1 (*c*) During retinal post-venous phase after 2 days of occlusion

(*c*) *After* 1 *week* (*Fig.* 2*a*) IVFA indicated choroidal filling up to the macular region.

(*d*) *After* 2 *to* 3 *weeks* Most of the temporal choroid started to fill with fluorescein (Fig. 2*a*), though this circulation was delayed as it was traced away from the posterior pole.

After this period, the extent of choroidal filling improved very slowly; some delay in circulation was evident even after 3 months.

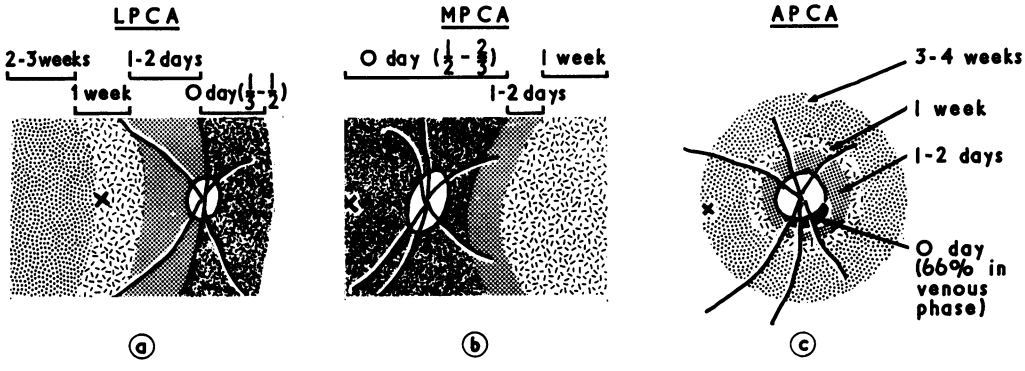


FIG. 2 *Diagrammatic representation of choroidal filling after occlusion of* (a) *LPCA,* (b) *MPCA, and* (c) *APCA.* X = *position of macula*

(2) *After MPCA occlusion*

(*a*) *About* 1 *hour after occlusion* Through the unoccluded LPCA one-half (in five eyes—Fig. 3) to two-thirds (in twelve eyes) of the choroid filled with dye. This included the whole area temporal to the optic disc. In about one-third of the eyes, the lower half of the choroid filled shortly before the

upper half. The upper border of this choroidal filling was fairly well-defined. Additional isolated patches of the choroid were seen to fill, as discussed below.

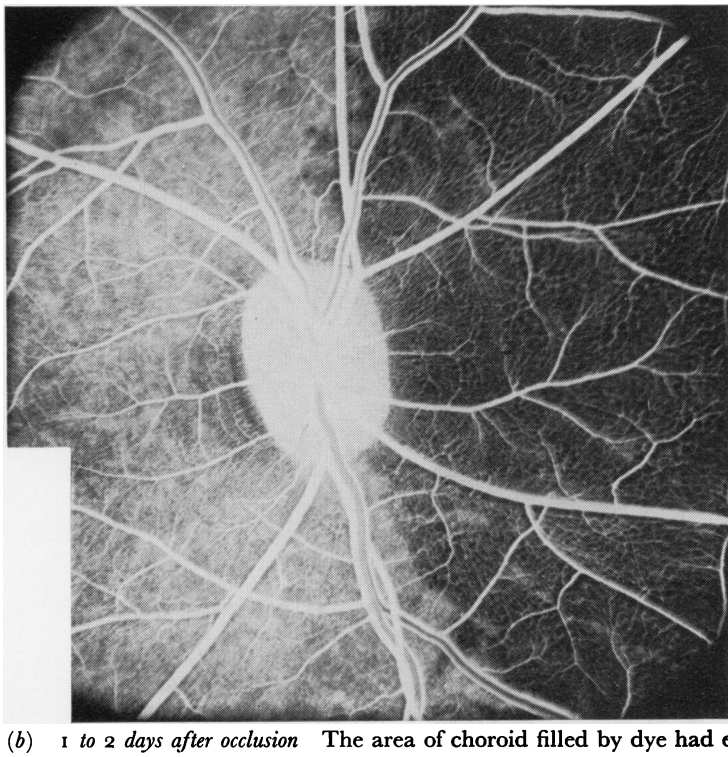


FIG. 3 *Angiogram after MPCA occlusion during retinal arterio-venous phase*

(*b*) 1 *to* 2 *days after occlusion* The area of choroid filled by dye had extended slightly nasally.

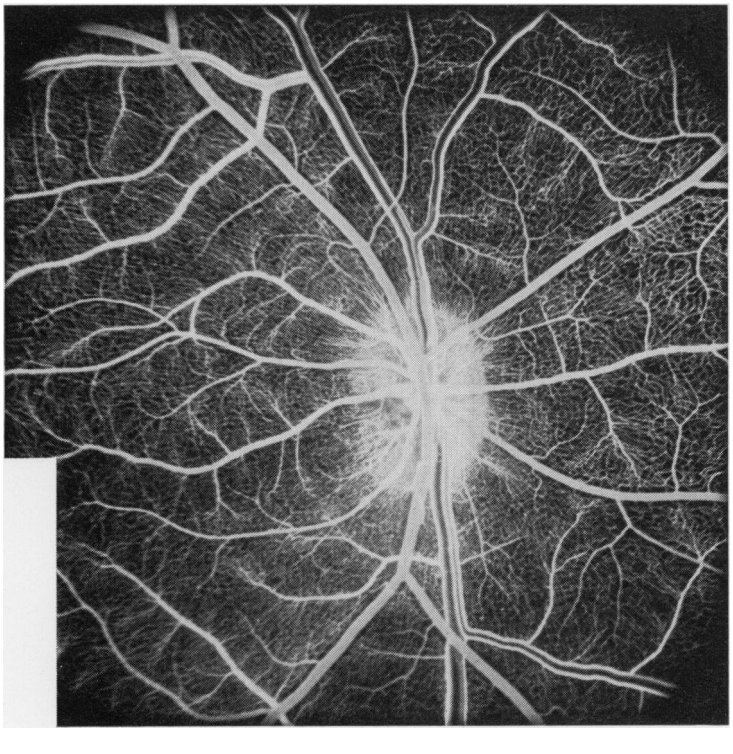
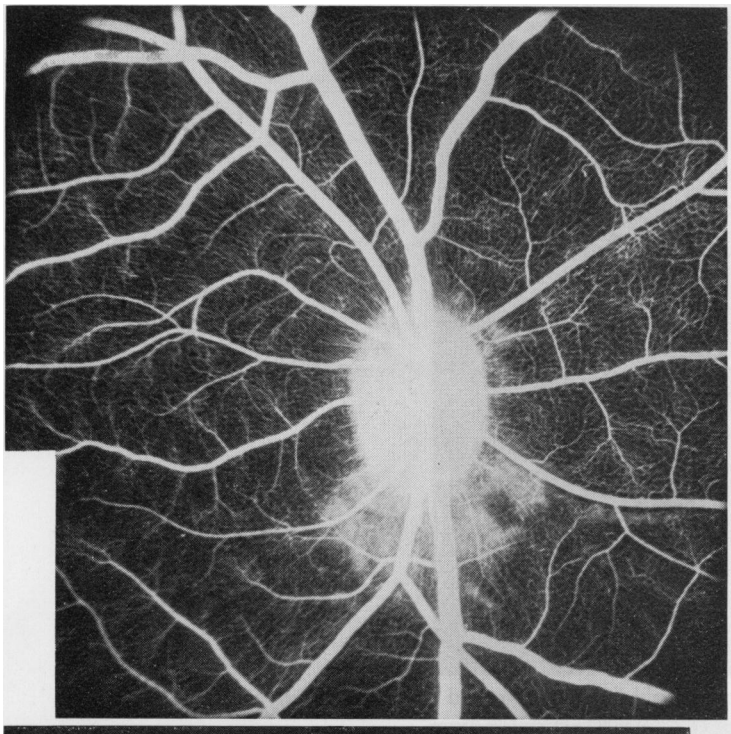
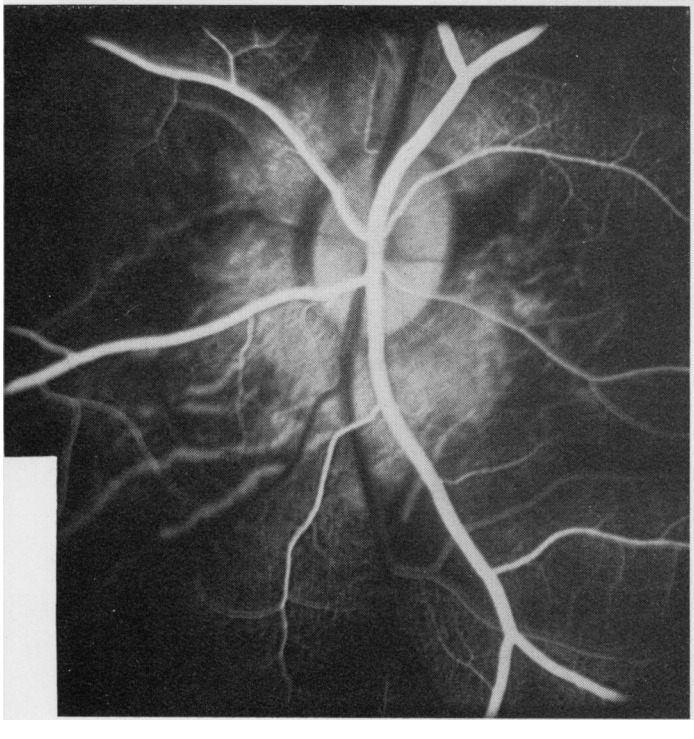


FIG. 4 (*a*) (See p. 724)

(*c*) *In about* 1 *week* Delayed filling by dye was seen in most of the nasal choroid (Fig. 2*b*).
(*d*) *After* 2 *to* 3 *weeks* The whole of the visible choroid was seen to be filled with dye, there being some delay in filling of the more nasal parts.



(*b*)



(*c*)

FIG. 4 *Angiograms after APCA occlusion:*

(*a*) and (*b*)—1 hr after occlusion during retinal (*a*) arterio-venous and (*b*) late venous phases. Note no filling of choroid in (*a*), and filling of lower PPC in (*b*)

(*c*) 29 hrs after occlusion during retinal arterial phase. Most of PPC has filled and some main choroidal vessels fill from the PPC

(3) *After APCA occlusion*

(*a*) *About 1 hour after occlusion* No filling of the choroid was seen in the transit of the dye (Fig. 4*a*). In about two-thirds of the eyes in this group, a sector of the PPC filled during the retinal venous phase, the dye appearing first at the margin of the optic disc (Fig. 4*b*). In the remaining one-third of the eyes, no filling of the PPC by dye was seen. Additional isolated areas of the choroid were, however, seen to fill with dye, as discussed below.

(*b*) *1 to 2 days after occlusion* The PPC and the immediately adjacent choroid started to fill; the filling of part of the PPC usually started in the arterial (Fig. 4*c*) or arterio-venous phase, and the rest of the circulation was delayed.

(*c*) *1 week after occlusion* The choroid, extending for a considerable distance around the PPC, filled well from the PPC during the transit of the dye (Fig. 2*c*).

(*d*) *After 2 to 3 weeks* The choroidal filling by dye improved with time and, after about three weeks, most of the choroid filled (Figs 2*c*, 5), though this filling was delayed, particularly in peripheral parts.

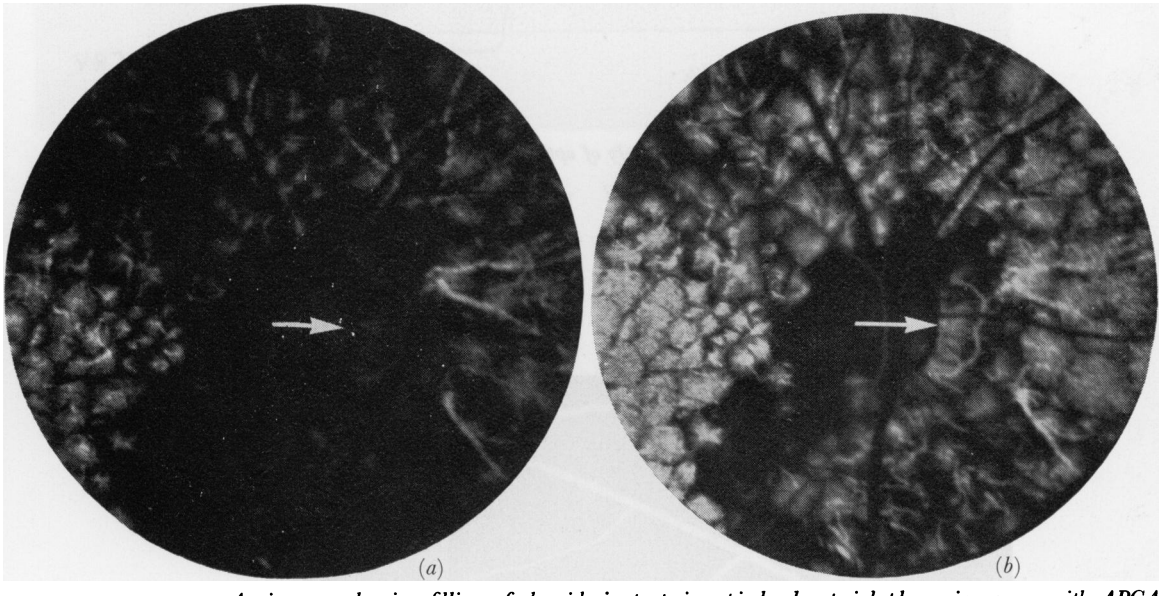


FIG. 5 *Angiograms showing filling of choroid* via *posterior episcleral arterial plexus in an eye with APCA occlusion. 44 days after occlusion*

(*a*) Filling of PCAs starts during the pre-retinal-arterial phase with a small area of PPC filling nasally (arrow)
(*b*) Extension of choroidal filling with onset of retinal arterial phase

(*e*) *After 3 months* The choroidal dye filling was complete, though still somewhat delayed in the more peripheral regions.

The filling pattern of the choroid extending from the PPC region peripherally indicates that, in these cases, the choroid fills from the PPC in addition to other collaterals. The PPC itself fills from pial branches, which have anastomosed with other arteries which supply the optic nerve and sheath (Fig. 6, overleaf). This was demonstrated also by silicone rubber injection specimens.

Isolated patches of dye filling in choroid in area of supply of the occluded PCAs (lateral, medial, or all).

At the first examination after occlusion of the PCAs, an isolated patch of filling appeared in the unfilled area of the choroid during the late venous phase of the retinal transit of

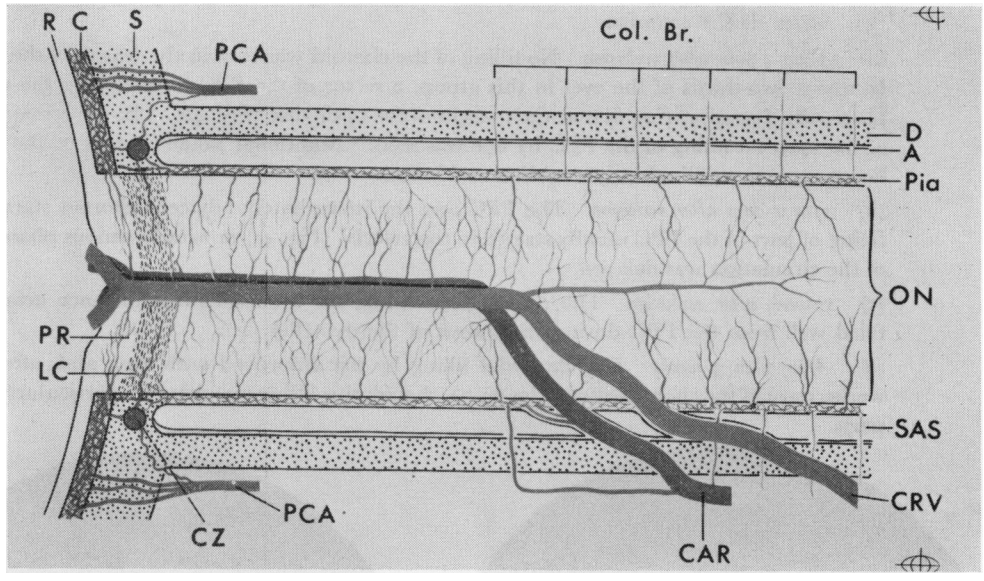


FIG. 6 *Schematic diagram of blood supply of optic nerve in man*

A	— Arachnoid mater	CRV	— Central retinal vein	Pia	— Pia mater
C	— Choroid	CZ	— Circle of Zinn and Haller	PCA	— Posterior ciliary artery
CAR	— Central artery of the retina	D	— Dura mater	PR	— Pre-laminar region
Col.Br.	— Collateral branches from orbital arteries to optic nerve and its sheath	LC	— Lamina cribrosa	R	— Retina
		ON	— Optic nerve	S	— Sclera

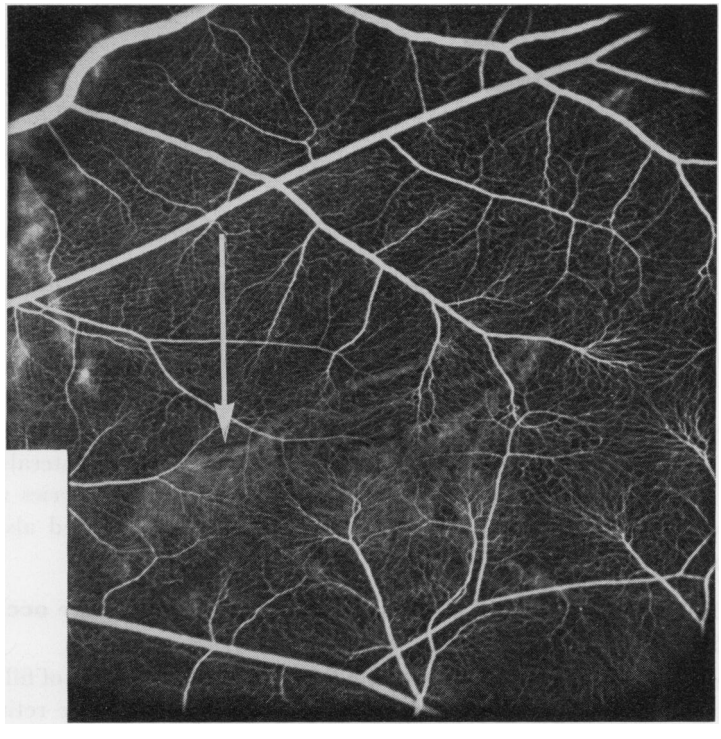
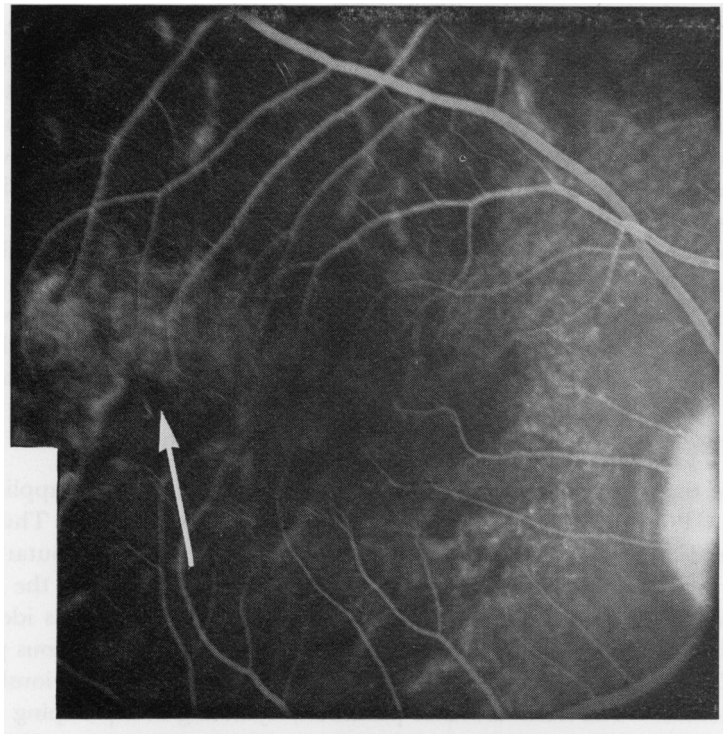
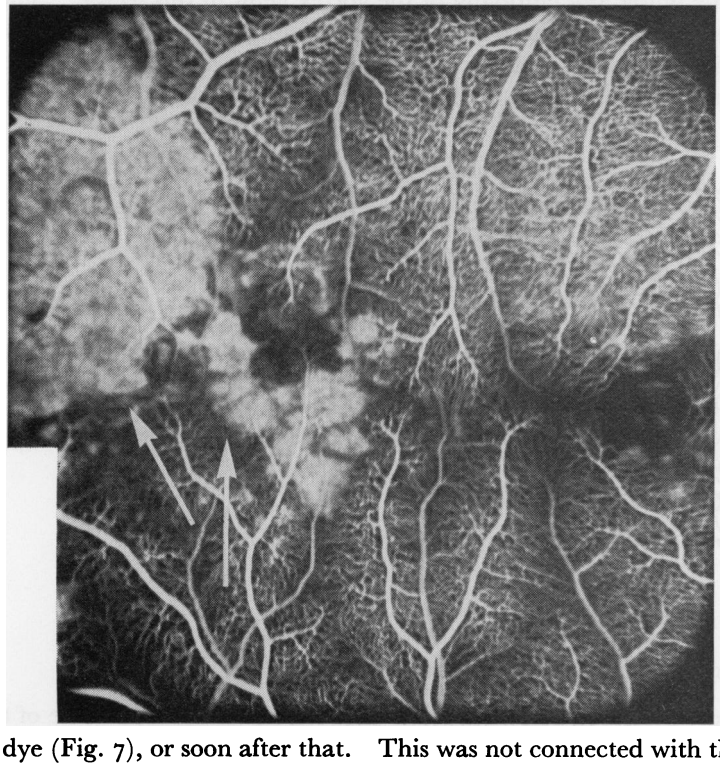


FIG. 7 *Angiograms showing filling of isolated patches in choroid supplied by occluded PCA*

(*a*) After MPCA occlusion, patch nasal to disc appeared during retinal venous phase



(*b*) After LPCA occlusion, patch temporal to macula appeared during retinal post-venous phase



(*c*) After LPCA occlusion, patches temporal to macula appeared during retinal arterial phase

dye (Fig. 7), or soon after that. This was not connected with the already filled part of the choroid. From this patch the filling extended all around and joined the already filled choroid.

In LPCA occlusion, in the region photographed, a patch was seen in two-thirds of the eyes. The patch appeared either in the supero-temporal or the infero-temporal quadrant or both, and/or in the macular region (Fig. 7*b*, *c*). In MPCA occlusion, a patch was seen in the superior, inferior, or central nasal regions in about three-quarters of the eyes (Fig. 7*a*). In APCA occlusion, these patches were seen in four-fifths of the eyes, twice as often on the temporal as on the nasal side, and twice as frequently in the upper as in the lower choroid. Thus, the superior temporal segment filled more frequently than any other sector. The macular area showed patchy filling in about half the eyes. The fact that these patches were not observed in the rest may simply be the result of the appropriate area not being included, or may be due to the variability in the collaterals available.

We feel that this isolated patchy filling in the area of choroid supplied by the occluded artery could be due to one or more of the following routes of collateral supply to the region:

(1) Via *retrograde circulation in the vortex veins*

The territory of the uveal tract drained by one vortex vein is usually supplied by three sets of arteries, MPCA, LPCA, and the anterior ciliary arteries (Fig. 8). Thus, occlusion of the MPCA and/or LPCA reduces the blood pressure in the venous tributaries draining the non-filling sector. Blood could regurgitate from the main stem of the vortex vein into these tributaries and lead to retrograde filling of the choroid. This idea is further supported by the fact that these isolated areas appear during the late venous phase of the dye transit. This may be one of the factors in the late filling of the previously non-filled sector of the choroid. The normal ocular pulsation, by acting as a pumping mechanism, would help in the filling and emptying of the vortex veins in this sector. Silicone rubber injection studies certainly demonstrate vortex vein filling where there is no corresponding choroidal arterial supply. Experimental occlusion of one or two of the vortex veins seems to have had no influence on the filling pattern of the isolated patches in any of these studies.

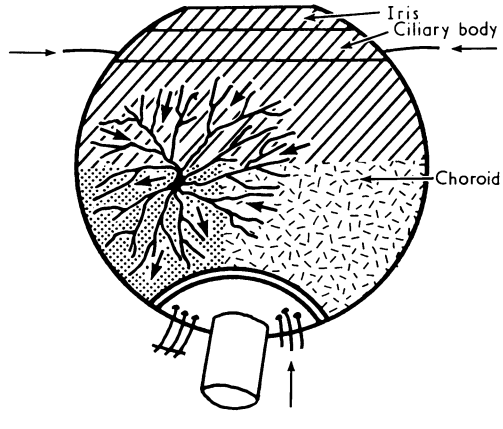


FIG. 8 *Diagram showing route of retrograde flow of blood* via *the vortex vein into the part of choroid supplied by an occluded PCA. Arrows indicate direction of flow of blood in various vessels*

(2) Via *the posterior episcleral arterial plexus*

This plexus is fully described below (p.731). The collaterals on the surface of the posterior sclera between the LPCA, MPCA, and optic nerve sheath and other vessels would enable dye or blood to reach the occluded vessel distal to the occlusion. Such communications

were demonstrated in the silicone rubber injection studies. The cutting of the PCAs some distance posteriorly from the sclera would leave these collaterals intact. In LPCA occlusion, isolated filling of the macular region or the adjacent area during the late venous or post-venous phase could be due to such communications. The choroidal filling through these collaterals increased with the passage of time (Fig. 5).

(3) Via *the pial plexus*

Isolated small patchy filling of the PPC in the occluded sector was seen (Figs 1*b*,*c*; 4*b*,*c*; 5*a*,*b*; 9). This usually appeared about the venous phase. Fluorescence spread from these to adjacent areas. This filling was thought to be due to the filling of the PPC *via* its pial branches anastomosing with pial branches of other origins (Fig. 6). This again was confirmed by silicone rubber injection studies.

(4) *In two eyes an area of choroid, temporal to the macular region, filled with dye during the retinal arterial phase of the circulation*

From this area, filling extended nasally (Fig. 7*c*). This was noticed in the eyes where the lateral long PCA was not occluded.

During the late phases, about 10 to 15 min. after the fluorescein injection, faint fluorescence was seen in most of the choroid very shortly after occluding the vessels, indicating a very slow perfusion of fluorescein into the previously non-filled choroid *via* the various collaterals. Areas which did not show this fluorescence at the review about 1 to 2 hrs after occlusion of the PCAs subsequently developed fundus lesions (Hayreh and Baines, 1972).

During the initial period after the PCA occlusion, particularly during the first week, a marked leakage of fluorescein into the eye was noticed on IVFA. This may be due to the following factors:

(*i*) Unlike the normal pigment epithelial cells, the infarcted pigment epithelium is unable to prevent the diffusion of fluorescein from the choroidal vascular bed into the overlying retina and vitreous. Moreover, the permeability of the choroidal vascular bed would be increased by its ischaemia.

(*ii*) The involvement of the long PCAs (which are branches of the PCAs) in these eyes might produce ischaemia of a part of the anterior uvea, resulting in increased vascular permeability in the involved vessels.

(*iii*) The marked ocular hypotony seen in these eyes may also increase vascular permeability.

Peripapillary choroid (PPC)

After PCA occlusion the PPC showed filling defects in the IVFA in every eye (Fig. 9, overleaf).

(1) *After LPCA occlusion*

One hour after the occlusion, there was no filling of the temporal PPC during the transit of the dye in two-thirds of the eyes, and in the remainder this area filled only during the retinal venous phase of the transit (Figs 1*b*; 9*a*). Filling of the temporal PPC had not improved noticeably even after two days (Fig. 1*c*). Filling of this region during the transit of the dye started towards the latter part of the second week, but it was delayed as compared to the nasal side (Fig. 9*a*). This delay was evident in some eyes even 10 to 12 weeks after occlusion.

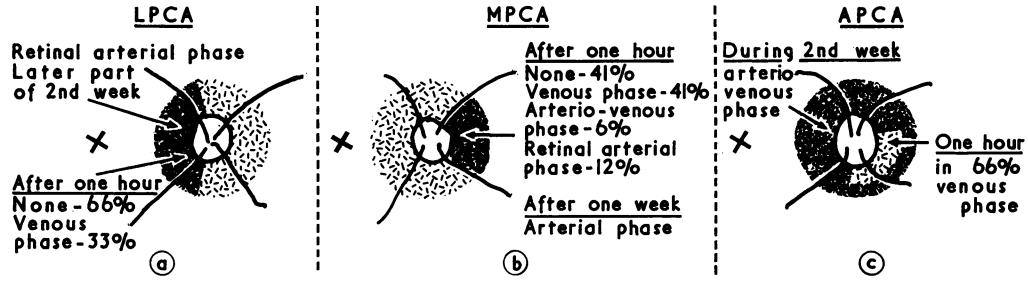


FIG. 9 *Diagrammatic representation of filling pattern of the peripapillary choroid (PPC) as seen after occlusion of:* (*a*) LPCA, (*b*) MPCA, and (*c*) APCA

X = position of macula during retinal arterial (A), arterio-venous (AV), and venous (V) phases.

(2) *After MPCA occlusion*

1 hour after the occlusion the PPC did not fill completely on the nasal side during the transit of dye in 41 per cent. of the eyes. However, the filling defect in the MPCA occlusion was usually much smaller than that seen in the temporal PPC with LPCA occlusions (Fig. 9*b*). The nasal part of the PPC filled only during the retinal venous phase of the transit of the dye in 41 per cent., during the arterio-venous phase in 6 per cent., and in the arterial phase in 12 per cent.

After about 1 week, the nasal PPC started to fill during the late arterial phase, slowly recovering to normal filling later on. A slight delay was still evident in some eyes even after about three months.

(3) *After APCA occlusion*

1 hour after the occlusion a sector of the PPC filled in two-thirds of the eyes, frequently in the lower nasal part (Figs 4*b*; 9*c*). This filling occurred towards the latter part of the venous phase in most of these eyes.

Two days after the occlusion, the filling of the PPC had improved so that the filling defect was smaller and filled in earlier in the course of the transit of the dye than before (Figs 4*c*; 9*c*). During the second week, the whole of the PPC started to fill, usually before the end of the arterio-venous phase (Fig. 9*c*). With the passage of time more and more of the PPC and the surrounding choroid started to fill during the retinal arterial and, in some, even during the pre-arterial phase (Fig. 5*a*,*b*).

Retinal circulation after PCA occlusion

No abnormality was detected in the retinal circulation. In two eyes with a cilio-retinal artery, occlusion of the LPCA produced a retinal capillary filling defect in the region supplied by the cilio-retinal artery (Fig. 10, opposite). The cilio-retinal artery slowly filled later on from the optic disc.

(B) SILICONE RUBBER PERFUSION STUDIES

These studies showed the presence of episcleral vessels on the posterior aspect of the globe and the area around the optic nerve. These vessels anastomosed with one another to form

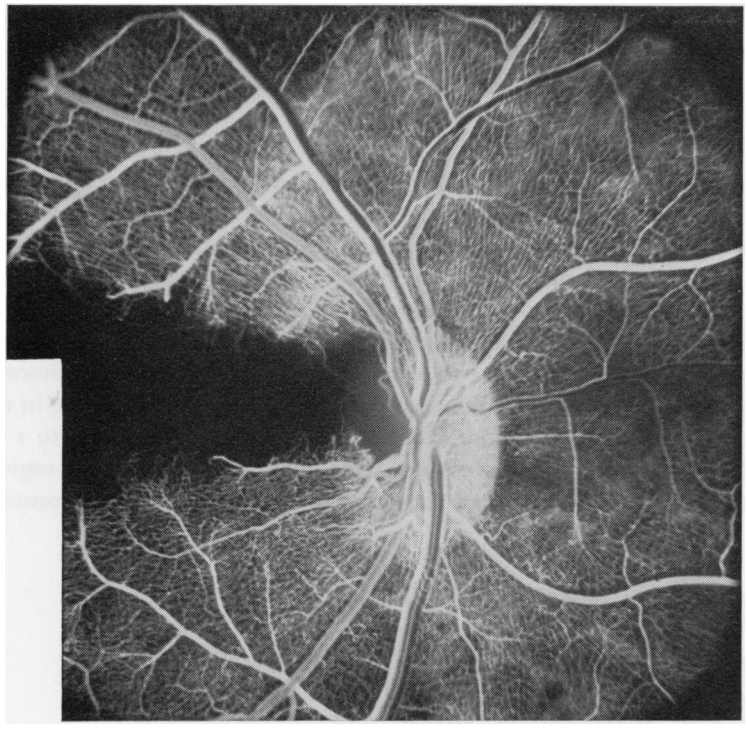


FIG. 10 *Angiogram showing filling defect in retinal vascular bed after LPCA occlusion in an eye with a cilio-retinal artery supplying the unfilled part of the retina*

the *posterior episcleral arterial plexus*. Branches from the following arteries were usually seen to participate in this network:

(*i*) Arteries on the dural sheath of the optic nerve, which in turn are derived from the various orbital arteries (Hayreh, 1962, 1963, 1964).

(*ii*) The PCAs just before their site of penetration into the sclera.

(*iii*) Arteries from the inferior oblique, superior oblique, and deep head of the lateral rectus at their insertion.

(*iv*) Small vessels accompanying the short ciliary nerves.

(*v*) Multiple small vessels from the surrounding loose areolar tissue.

In occlusion of the different PCAs, the various channels contributing to the episcleral arterial plexus played an important part in filling the occluded PCAs, as is evident from the following account.

(1) *After LPCA occlusion*

Nine eyes in this group were studied by silicone injection; seven eyes were followed for up to 2 hrs and two eyes for 7 and 14 days. The distal stumps of the cut LPCA filled *via* the episcleral plexus, mostly from the MPCA around the base of the optic nerve and also by vessels on the sheath of the optic nerve. The chorio-capillaris filled locally in the choroid around the site of their entry into the sclera. The vortex veins were filled completely with silicone in both their anterior and posterior parts.

(2) *After MPCA occlusion*

There were five eyes in this group which were followed for up to 2 hrs. The distal stumps of the MPCA filled *via* the episcleral plexus, mostly from the LPCA and, less frequently

via the anastomoses with the vessels on the sheath of the optic nerve. There was no filling of the MPCA in one eye. In all the eyes, the vortex veins filled in the posterior part, in addition to the normal filling of their anterior parts.

(3) *After APCA occlusion*

There were sixteen eyes in this group, of which nine were followed for up to 2 hrs, three up to 2 wks, and four for 3 mths. Among the eyes followed for up to 2 wks the stumps of the cut LPCAs filled much more frequently than the stumps of the MPCAs. This filling occurred *via* the episcleral arterial plexus, mostly by the vessels from the sheath of the optic nerve. The vortex veins were normally filled over the whole of the uveal tract, including the posterior part. The arterial filling was normal in the anterior part of the choroid from the equator forwards, and also in the posterior part of the choroid extending for a variable distance from the optic nerve (patchy and inconstant filling in eyes followed for up to 2 hrs but uniform and more extensive in those followed for up to 2 wks), but no such choroidal arterial filling was seen in a wide zone in the equatorial region extending around the eye and situated between the anterior and posterior filled zones in all these eyes (Fig. 11).

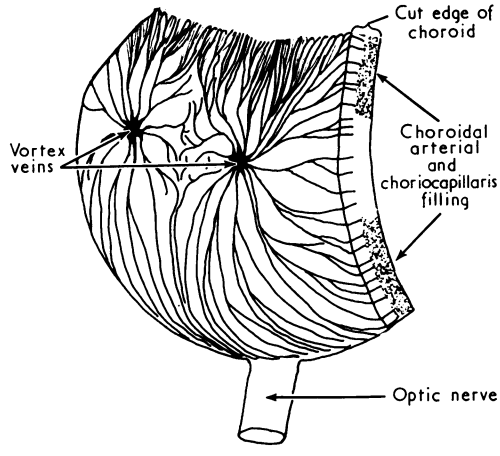


FIG. 11 *Schematic drawing showing venous and arterial filling of choroid by silicone after PCA occlusion*

A large number of pial vessels on the retrobulbar part of the optic nerve were seen to extend into the PPC. These were also seen in eyes with LPCA and MPCA occlusion.

In the eyes with APCA occlusion which were followed for 3 mths, arteries resembling the normal PCAs and lying on the sheath of the optic nerve and some accompanying the short ciliary nerves were seen to have replaced the PCAs. The episcleral plexus was fairly prominent in these eyes. Thus, the main channels, which seemed ultimately to establish the full circulation in these eyes on a long follow-up, were the vessels on the sheath of the optic nerve and the ones accompanying the short ciliary nerves. Large numbers of prominent pial vessels were also seen to extend into the PPC and the whole of the choroid was filled completely.

In none of our specimens seen soon after the occlusion of the PCAs did the occluded choroidal vascular bed fill from the adjacent unoccluded choroid.

Discussion

The segmental distribution of the PCAs has excited considerable controversy. This is

because some workers have found the PCAs to have a segmental supply to the choroid so that they behave as functional end-arteries (Wagenmann, 1890; Siegrist, 1895; Leber, 1903; Gonin, 1903; Studer, 1906; Coats, 1907; Hepburn, 1912, 1935; Archer, Krill, and Newell, 1970; Amalric, 1971; Foulds, Lee, and Taylor, 1971). This has been denied by others (Nicholls, 1938; Vilstrup, 1952; Wybar, 1954*a*,*b*; Correia, 1957; Scullica, 1957; Ruskell, 1961; Ring and Fujino, 1967). Studer (1906) was of the opinion that the PCAs are end-arteries in the rabbit but not in man. Wybar (1954*a*,*b*) and Ring and Fujino (1967), in their latex injection studies of the human PCAs, however, concluded that these arteries were segmentally arranged and that each branch supplied a localized zone of the choroid. A review of the literature shows that a segmental distribution has been postulated by workers whose observations are based on *in vivo* occlusion in animals (mostly rabbits) and man, while the non-segmental view has been based mainly on *post mortem* injection studies in animals (again mostly rabbits) and man. One of us, during *in vivo* studies in the rhesus monkey by IVFA, found a well-demarcated segmental distribution by the PCAs (Hayreh, 1970). This has been further confirmed by our present study of experimental occlusion of the PCAs. In view of this, it seems that the PCAs act physiologically as end-arteries *in vivo*, but are not so anatomically. There is, however, a possibility that the absence of segmental distribution after *post mortem* injection studies may be an artefact. This could be due to the filling of the arterial and venous choroidal vascular bed, including the chorio-capillaris, by the injection material which would mask the segmental nature of the PCA distribution in the choroid. Hayreh (1970) found that the main LPCA/MPCA usually supplies half of the choroid, the distribution division being either vertical or horizontal. If there are two MPCAs, each may supply a quadrant nasally.

The LPCA normally fills one-half to two-thirds of the temporal choroid, while the MPCA fills one-third to one-half of the nasal choroid. Immediately after occlusion of the LPCA or MPCA, the area of the choroid supplied by that artery does not fill during the transit of the dye on IVFA (Figs 1, 2, 3). After the venous phase of the retinal circulation, siolated choroidal patches fill in the occluded area (Fig. 7); these are probably mainly due to a retrograde flow of blood in the vortex veins (p. 728) and also to their filling *via* the episcleral collaterals of the PCAs (p. 728) and the pial collaterals of the PPC (p. 729), as demonstrated by IVFA and silicone rubber injections. After occlusion of all PCAs, there is no filling of the choroid during the retinal transit of dye; in the late venous phase, or soon after that, sectoral filling of the PPC starts *via* the pial collaterals (Figs 2*c*; 4; 9), and that of the periphery by retrograde flow of blood in the vortex veins and the other collaterals mentioned above.

In occlusion of either the MPCA or the LPCA, on follow-up, the part of the choroid supplied by the occluded artery fills progressively more rapidly and more completely as time passes. Eventually, practically a normal circulation is established in the occluded choroid, with only a minor delay in the circulation compared to normal. In establishing this adequate circulation in the occluded choroid, the part played by the various collaterals is not yet definitely known. Possibly it is due to the following modes, singly or in combination:

(1) *Via* the posterior episcleral arterial plexus (p. 728, 731).

(2) *Via* the pial collaterals of the PPC (p. 729). This seems to be the major mode during the early stages of occlusion of all the PCAs as discussed above (p. 725).

(3) A slow extension of the filling of the occluded choroid for a short distance from the

unoccluded choroid is seen, but this is not at all significant in re-establishing the circulation in the occluded sector. The exact mechanism of this spread is not known; presumably it is due to localized overlapping between the distribution of small arterioles and veins to the chorio-capillaris.

The presence of inter-arterial anastomotic channels (Wybar, 1954a; Correia, 1957; Ruskell, 1961; Ring and Fujino, 1967) and of arterio-venous anastomoses (Loewenstein, 1949; Kiss and Orbán, 1951; François, Neetens, and Collette, 1955) in the human choroid have been described. The existence of arterio-venous anastomoses has been contradicted by Ashton (1952), Greaves and Perkins (1952), Ring and Fujino (1967), and the present study.

The pattern of filling of the occluded choroid in these eyes indicates that the effects of occlusion of the MPCA differ from those of occlusion of the LPCA in the following ways:

(*a*) The former is likely to involve a smaller area of the choroid than the latter;

(*b*) The restoration of choroidal circulation in the occluded sector is comparatively faster in MPCA occlusion than in LPCA occlusion.

The present studies show that in the PPC the filling does not extend from one sector to its neighbour. The PPC, therefore, is not a continuous vascular bed all around the optic disc (OD); the blood vessels in it must have a sectoral distribution. The presence of such a sectoral pattern in the PPC is important because of its contribution to the supply of the adjacent OD and retrolaminar optic nerve. Therefore, obliteration of a sector of the PPC could, if the pial collaterals are not adequate, produce a sectoral lesion of the OD and optic nerve (Hayreh, 1970).

Summary

Choroidal circulation after occlusion of the lateral, medial, or all the posterior ciliary arteries (PCAs) was investigated in 85 rhesus monkey eyes by *in vivo* intravenous fluorescence angiography and in thirty of these by *post mortem* silicone rubber perfusion of the vascular bed. The segmental distribution of the PCAs is stressed.

In lateral and medial PCA occlusions, the filling of the choroid supplied by the occluded artery, immediately after the occlusion, was delayed till after the retinal venous phase, when sluggish, incomplete filling took place *via* collaterals from the posterior episcleral arterial plexus and by retrograde circulation through the vortex vein tributaries, but not from the normally filling choroid. One to two days later, the filling appeared earlier and was more extensive. Progressive improvement in filling took place until, at 2 to 3 weeks after the occlusion, the choroid filled in the affected area, although delayed. In occlusion of all PCAs, centrifugal arterial filling from the peripapillary choroid extended into the affected area of choroid, incompletely and sluggishly at first, but completely, though delayed, 3 to 4 weeks later. The filling was thought to take place *via* the pial plexus of the optic nerve and the posterior episcleral arterial plexus.

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