Communications

Pathogenesis of visual field defects

Role of the ciliary circulation

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Clinically the presence of nerve fibre bundle defects in glaucoma and non-glaucomatous lesions, and altitudinal or vertical hemianopias in pre-chiasmal lesions has been known for a long time. These suggest a sectoral lesion in the optic nerve or retina. In the majority of such cases, no retinal lesion is seen to account for these visual field defects. It has been presumed that the lesion lies in the optic nerve. Such a visual field defect can be produced only by a sectoral lesion. Most of these field defects are considered to be vascular in origin, but studies of the blood supply of the optic nerve have so far failed to demonstrate any segmental blood supply to the nerve which could explain them. The present study demonstrates such a sectoral blood supply in the optic nerve head and choroid and is of considerable significance in explaining the site of the lesion and the pathogenesis of these defects.

Material and methods

Fluorescence fundus angiography was performed in 24 eyes of rhesus or cynomolgus monkeys after an injection of 0.15 ml. of 5 per cent. fluorescein solution into the common carotid artery on the same side. The fundus was photographed serially every 0.8 second, by a Zeiss fundus camera adapted for fluorescence angiography, from the time of injection to the end of the transit of the dye. Several fluorescence angiographies were done on the same eye.

In eight of these 24 eyes fluorescence angiography was done after an experimental occlusion of the central retinal artery, in order to make the choroidal circulation more clearly visible (Hayreh and Perkins, 1969; Hayreh, 1969).

Observations

The pattern of the choroidal filling was recorded in all these eyes. Normally the choroidal bed fills up about a second or so before the retinal circulation. Once the retinal circulation fills up, it completely masks the underlying choroidal bed. In sixteen eyes the pattern of filling of the choroidal vascular bed was studied during this very short time interval between the retinal and choroidal fillings. In the remaining eight eyes, the retinal circulation was completely eliminated by an experimental occlusion of the central retinal artery in the orbit before fluorescence angiography, and this allowed a detailed study of the choroidal vascular bed without any masking effect by the retinal circulation (Hayreh, 1969; Hayreh and Perkins, 1969).

In ten eyes the whole of the choroidal vascular bed did not fill simultaneously; a part of the bed filled before the rest. This was due to the difference in the circulation time of the different posterior ciliary arteries. This difference in the circulation time made it possible to outline the distribution of the different posterior ciliary arteries in the choroid

and the optic disc. The distribution of these arteries showed the following patterns in the present study:

(a) In 80 per cent. the temporal and nasal parts of the choroid filled separately—more often the temporal part before the nasal part. The ciliary supply to the optic disc filled with the respective parts of the choroid, so that it was possible to outline the territory of the optic disc supplied by the posterior ciliary artery. This is illustrated in Figs 1, 2, 3, 4, and 5a.

FIG. 1 Fluorescence fundus angiogram of right eye of a cynomolgus monkey during pre-retinal-arterial phase, showing a well-demarcated filling of nasal half of choroid and prelaminar vessels in nasal part of optic disc

FIG. 2 Fluorescence fundus angiogram of right eye of a cynomolgus monkey during the pre-retinal-arterial phase, showing filling of temporal half of choroid and prelaminar vessels in temporal part of optic disc

FIG. 3 Fluorescence fundus angiogram of left eye of a cynomolgus monkey during the pre-retinal-arterial phase, showing filling of the temporal half of the choroid with an early filling of some of the prelaminar vessels in the optic disc from the choroid. Note an early filling of the inferior nasal part of the choroid with the peripapillary zone not yet filled

FIG. 4 Fluorescence fundus angiogram of right eye of a cynomolgus monkey after experimental occlusion of the central retinal artery. The lateral posterior ciliary artery supplies the temporal half and the superior nasal part of the choroid. Early filling of the medial posterior ciliary artery in the inferior nasal sector of the choroid is seen. The border zone between the two and the nasal peripapillary region is still not filled. Part of the optic disc supplied by the lateral posterior ciliary artery is filled

The medial posterior ciliary arteries usually supplied about half of the choroid and the lateral posterior ciliary arteries supplied the other half (Figs 1, 2, 3, and 5a), but sometimes one more than the other (Fig. 4). The areas of distribution by the two posterior ciliary arteries remained separate for some time with little communication between the two (Fig. 4). One received the impression that the choroidal vascular bed was not a freely communicating bed but tended to have a sectoral distribution. The same applied to the distribution of the arteries in the optic disc.

FIG. 5. Fluorescence fundus angiograms of left eye of a cynomolgus monkey after experimental occlusion of the central retinal artery:

- (a) Filling of the temporal half of the choroid,
- (b) Another angiogram during the early stages showing filling of the superior nasal quadrant only. Marked fluorescence of the optic disc in (a) and (b) is due to previous injections of fluorescein.

(b) In 20 per cent. the upper and lower parts of the choroid and optic disc filled separately, with a sharp horizontal line of demarcation (Fig. 6, and Fig. 7, overleaf). Here again no free communication could be detected in their distribution.

FIG. 6. Fluorescence fundus angiograms of right eye of a cynomolgus monkey after experimental occlusion of the central retinal artery:

- (a) First angiogram, showing filling of upper half of choroid and optic disc with a well-demarcated horizontal border
- (b) Second angiogram, showing filling of superior nasal quadrant at first. The marked fluorescence of the optic disc in (b) is due to the previous injection of fluorescein.

FIG. 7 Fluorescence fundus angiograms of right eye of a cynomolgus monkey after experimental occlusion of the central retinal artery:

- (a) Filling of upper half of choroid at first.
- (b) Another angiogram showing filling of lower half of choroid at first. The marked fluorescence of the optic disc in (a) and (b) is due to previous injections of fluorescein

(c) On repeated fluorescence angiography in the eyes seen in (a) and (b) above, it was possible to see in some a further sectoral filling of the choroid and the optic disc. This was mainly quadrantic in pattern, as shown in Figs 5b and 6b.

Discussion

The observations of the present study *in vivo*, that the posterior ciliary arteries have a segmental distribution in the choroid and optic disc, would help to explain the mechanism of some of the visual field defects which have been ill-understood so far. Before discussing these, it is essential to describe briefly the pattern of the posterior ciliary arteries in man, and the blood supply of the optic disc and optic nerve, in order to make the discussion explicit.

POSTERIOR CILIARY ARTERIES

A detailed account of the pattern of the posterior ciliary arteries in man has been given elsewhere (Hayreh, 1962a). Briefly, the posterior ciliary arteries can be designated medial, lateral, or superior, according to their relationship to the optic nerve near the eyeball (Fig. 8). The ophthalmic artery may give out one (in 3 per cent.), two (in 48 per cent.), three (in 39 per cent.), four (in 8 per cent.), or five (in 2 per cent.) posterior ciliary arteries of varying sizes.

- (a) *Medial posterior ciliary arteries* may be one (in about 70 per cent.) or two (in about 30 per cent.).
- (b) *Lateral posterior ciliary arteries* may be one (in about 75 per cent.), two (in about 20 per cent.), three (in less than 2 per cent.), or none (in about 3 per cent.).
- (c) *Superior posterior ciliary arteries* are seen in only 9 per cent. and may be one (in 7 per cent.) or two (in 2 per cent.). These are usually small in size.

The mode of origin of the posterior ciliary arteries from the ophthalmic artery varies. The medial posterior ciliary arises in common with the central retinal artery in 44 per

FIG. 8 Photograph of inferior surface of optic nerve (ON) and eyeball (EB), showing ophthalmic artery (OA), central retinal artery (CRA), and medial (MPCA) and lateral (LPCA) posterior ciliary arteries

cent. of cases (Fig. 8). In the remainder it usually arises alone and occasionally with other branches of the ophthalmic artery. The lateral posterior ciliary artery arises with the central retinal artery in only 12 per cent. of cases; in the remainder usually as an independent branch, and very rarely with any other branch. When the ophthalmic artery crosses over the optic nerve in the second part of its course in the orbit, the medial posterior ciliary artery is the first branch of the artery in 53 per cent. of cases (Fig. 8), and the lateral posterior ciliary in 22 per cent. On the other hand, when the ophthalmic artery crosses under the optic nerve in the orbit (in 17.4 per cent.), the lateral posterior ciliary artery is the first branch of the artery in 91.3 per cent. and the medial posterior ciliary is never the first branch.

The posterior ciliary arteries run forwards, divide into ten to twenty small branches (Wolff, 1948; Duke-Elder, 1961) and pierce the sclera near the optic nerve. Out of these large numbers of branches of the posterior ciliary arteries, one on the medial and another one on the lateral side form the long posterior ciliary arteries, while the rest are called the short posterior ciliary arteries. The two long posterior ciliary arteries pierce the sclera on the medial and lateral sides slightly further away from the optic nerve than the short ones. A retrospective study of the photographs of about a dozen specimens, given by Hayreh (1958), indicates that the lateral posterior ciliary arteries usually pierce the sclera lateral or supero-lateral to the optic nerve, while the medial posterior ciliary arteries pierce the sclera medial or infero-medial to the nerve (Fig. 8). This information, however, needs further confirmation. The posterior ciliary arteries are usually markedly tortuous.

BLOOD SUPPLY OF THE OPTIC DISC

This is given in detail elsewhere (Hayreh and Perkins, 1968, 1969; Hayreh, 1969). Briefly, the lamina cribrosa region is supplied by centripetal branches from the short posterior ciliary arteries, either by direct branches or, less frequently, through the so-called arterial circle of Zinn and Haller (Fig. 9).

FIG. 9 Pattern of blood supply of optic nerve in man

A	Arachnoid	CRV	Central retinal vein	PCA	Posterior ciliary arteries
C	Choroid	CZ	Circle of Zinn	R	Retina
CAR	Central artery of the retina	D	Dura	S	Sclera
Col. Br.	Collateral branches of ophthalmic artery	ON	Optic nerve		

The prelaminar region is supplied by centripetal branches from the adjacent peripapillary choroid (Figs 1, 2, 3, 4, 6a, 9, and 11). The part containing the papillomacular bundle is much more vascular than the rest (Figs 2 and 4).

The surface nerve fibre layer of the disc is supplied by the retinal arterioles; sometimes the temporal sector of this receives a contribution from the underlying ciliary vessels of the prelaminar region.

Thus, the *main source of blood supply to the optic disc is derived from the ciliary circulation*. As far as one can judge, the ciliary contribution to the disc by the various short posterior ciliary arteries tends to be sectoral.

BLOOD SUPPLY OF THE INTRAORBITAL PART OF THE OPTIC NERVE

A detailed account of this is given elsewhere (Hayreh, 1958, 1962b, 1963a, b, 1964; Singh and Dass, 1960). Briefly, the intraorbital part of the optic nerve can be divided into two parts when considering its blood supply:

- (a) *The anterior segment* containing the central retinal vessels in its centre.
- (b) *The posterior segment* which lies behind (a).
- (a) *The anterior segment*

This contains axial and peripheral vascular systems (Fig. 9).

- (1) *Axial centrifugal vascular systems*: This is seen in 75 per cent. of the cases and is always formed by the intraneural branches of the central retinal artery and *not* by the so-called "central artery of

the optic nerve". The axial system supplies the centre of the optic nerve to a variable extent in this part of the nerve.

(2) *Peripheral centripetal vascular system*: This is present in 100 per cent. of the cases and is formed by (i) multiple recurrent pial branches all round the retrolaminar part of the optic nerve from the peripapillary choroidal vessels and some from the circle of Zinn and Haller; (ii) pial branches from the central retinal artery on the inferior (in 100 per cent.), lateral (in 50 per cent.), medial (in 39 per cent.) and superior (in 18 per cent.) surfaces of the optic nerve; and (iii) pial branches from the ophthalmic artery or its various intra-orbital branches. The distributions of (ii) and (iii) are reciprocal.

(b) *The posterior segment*

In this part of the optic nerve no axial centrifugal vascular system exists except for a very rare small recurrent branch from the central retinal artery, which may run back in the centre of the nerve in the posterior segment for about 2 to 3 mm. only. The whole of this segment is supplied by the peripheral centripetal vascular system (Fig. 9). This is formed by the pial vessels which usually arise direct from the ophthalmic artery and, less commonly, from the central retinal, medial muscular, medial posterior ciliary, posterior ethmoid, supraorbital, or other muscular arteries. These usually number one to three, rarely more. They are much less commonly situated on the lateral surface of the optic nerve.

I want to stress here that the "central artery of the optic nerve", as described by François and Neetens (1954, 1956) and François, Neetens, and Collette (1955), has never been seen by me in over a hundred human specimens, and that this has been further confirmed by a large number of workers.

SEGMENTAL DISTRIBUTION OF THE POSTERIOR CILIARY ARTERIES

Wagenmann (1890) thought, from his studies in rabbits, that the distribution of the short posterior ciliary arteries in the choroid was strictly segmental and that these arteries were functional end-arteries. Leber (1903) was of a similar opinion and emphasised the lack of anastomoses between the different branches of these arteries except in the peripapillary region and in the extreme periphery of the choroid. Coats (1907) and Hepburn (1912), from dubious clinical observations, subscribed to the idea of the segmental pattern of the choroidal vascular supply. Wybar (1954a,b), basing his opinion on his injection studies of the short posterior ciliary arteries, concluded that these arteries were segmentally arranged and that each branch supplied a localized zone of the choroid with an arteriolar-capillary network, but he denied any anatomical evidence for the concept that these arteries were true end-arteries. He injected seven eyes through the ophthalmic artery, after occlusion of a single short posterior ciliary artery. In four eyes the choroid was completely filled but, in the other three eyes, a partial sectoral filling defect occurred in the choroid which corresponded in situation to the blocked short posterior ciliary artery. On injection of a single short posterior ciliary artery immediately behind the eyeball in two eyes, he was unable to inject the entire choroid. He, however, regarded the filling defects in the last five eyes as artefacts.

In the present study *in vivo*, a well demarcated segmental distribution by the posterior ciliary arteries in the choroid and the optic disc was seen in ten of 24 eyes. In 80 per cent. of these eyes the temporal or nasal half of the choroid and the optic disc filled with the dye completely, while the other half showed a complete absence of filling; the areas of filling and non-filling were sharply outlined (Figs 1, 2, 3, and 5). In the remaining 20

per cent. of eyes, a similar phenomenon was seen in the upper or lower half of the choroid and optic disc (Figs 6 and 7) instead of the temporal or nasal half. On repeated fluorescence angiography in these ten eyes, in some only a quadrantic filling of the choroid and optic disc was seen, the rest being completely empty (Figs 5*b* and 6*b*). The fact that such a segmental distribution could not be outlined in all the 24 eyes does not necessarily mean that it was absent in the rest. This is because the ability to outline areas of supply by different posterior ciliary arteries by means of fluorescence angiography depends upon there being some difference in the circulation time in the different posterior ciliary arteries. When there is no such difference in the circulation time, the areas supplied by the various posterior ciliary arteries fill simultaneously, and it is impossible to outline the distribution by the individual posterior ciliary artery or the short posterior ciliary arteries. The other difficulty with the present technique is that it is impossible to take fluorescence angiographic pictures faster than 0.8 second with the Zeiss equipment, and this is too long a time interval to outline individual arteries with difference in circulation time of less than 0.8 second. This is illustrated by the fact that in some cases in which one transit of the dye showed a filling of half of the choroid and the disc, in another transit it was possible to outline only a quadrantic filling (Figs 5*b* and 6*b*). These studies *in vivo* thus suggest a *well-demarcated segmental distribution by the posterior ciliary arteries in the choroid and the optic disc*. This is further suggested by the selective localization of pathological lesions in the choroid and the disc in relation to its vascular anatomy. Some of the optic disc lesions in this category are discussed in this paper.

NERVE FIBRE BUNDLE DEFECTS

The term "nerve fibre bundle" refers to any small group of nerve fibres which lie together as they enter the optic disc (Traquair, 1944). The most classical visual field defect produced by nerve fibre bundle defects is the arcuate scotoma (Bjerrum scotoma, comet scotoma, scimitar scotoma). Though it is classically described in glaucoma, it is also seen in a large variety of lesions of the retina, optic nerve, optic disc, juxtapapillary choroiditis, myopia, etc. Cases showing the nerve fibre bundle defects due to a large number of causes have been reported by Harrington (1964a). It can assume a great variety of shapes and positions.

Nerve fibre bundle defects have been extensively investigated (Meisling, 1900; Friedenwald, 1902; Sinclair, 1905; Rønne, 1909, 1915, 1927; Traquair, 1944; Dubois-Poulsen and Magis, 1955, 1956; Dubois-Poulsen, 1952, 1956; Harrington, 1959, 1960, 1964a, b; and Hoyt, 1962; and others). Hoyt (1962), on tracing degeneration of the nerve fibres in the optic nerve after experimental retinal lesions in the Bjerrum area, found the distribution of degeneration from the upper and lower arcuate bundles located in the temporal half of the optic nerve, in a wedge-shaped area behind the lamina cribrosa, extending up to the central retinal vessels. These findings agreed in general with topographic fibre organization of the optic nerve described by Parsons (1902), Brouwer and Zeeman (1926), and Polyak (1957), though none of these described the arcuate bundles specifically.

The pathogenesis of nerve fibre bundle defects in glaucoma is described elsewhere. These defects may also be produced by inflammatory, traumatic, and vascular lesions of the optic disc and optic nerve, vascular lesions being considered by the majority to be by far the commonest cause. The site of the vascular lesion and the vessels involved has, however, proved a highly controversial matter.

Dubois-Poulsen and Magis (1954) found that the Bjerrum scotoma was not pathognomonic of glaucoma and might be found in many lesions affecting the visual pathways

anterior to the lateral geniculate body. They added that it corresponded to a well-separated bundle of nerve fibres and that its cause must be vascular and extraocular. They thought that the actual lesion might be located immediately posterior to the eyeball in the arterial circle of Zinn and Haller or further back in the optic nerve. They did not think that raised intraocular pressure had any role to play in the pathogenesis of the lesion in glaucoma. Harrington (1964a,b) stated that interference with the vascular supply of the anterior optic nerve in the region of the circle of Zinn and Haller might also give rise to nerve fibre bundle defects which could be indistinguishable from the field defects of glaucoma.

The vascular lesions responsible for nerve fibre bundle defects lie either in the optic disc or in the optic nerve. Since the centripetal vessels in the optic disc and optic nerve are radial and have a segmental supply, these would involve the nerve fibres in a sectoral fashion. In the optic disc there is no centrifugal vascular system and blood supply by the individual subdivisions of the posterior ciliary arteries to the nerve head is of a better defined sectoral type than that in the retrobulbar optic nerve. Moreover, the arrangement of nerve fibres in the optic disc topographically is presumably more representative of the retinal arrangement than that in the intraorbital part of the optic nerve. A typical nerve fibre bundle defect is, therefore, far more likely to arise from an optic disc lesion than from a posterior optic nerve lesion.

In the past, attention has been mainly focused on the lamina cribrosa region as the site of vascular occlusion by involvement of branches of the circle of Zinn or of the retrobulbar part of the optic nerve. Since the circle of Zinn is not commonly found, its distribution is generally taken over by direct branches from the posterior ciliary arteries themselves. These have a sectoral distribution in the region of the lamina cribrosa with little anastomoses between the adjacent arteries. Similarly, in the prelaminar region, it seems that each branch of the posterior ciliary arteries supplies a well-defined sector. The involvement of one of these arteries thus produces a sectoral nerve fibre bundle defect. The retrolaminar part of the optic nerve obtains its centripetal peripheral blood supply from the recurrent pial branches from the choroidal vessels and the circle of Zinn (Fig. 9). Occlusion of one of the posterior ciliary arteries or its smaller subdivisions, therefore, would involve a sector of the lamina cribrosa, the prelaminar part of the optic disc, and the retrolaminar part of the optic nerve (Fig. 9). When a sector of the peripapillary choroid only is involved, the region of the lamina cribrosa may be spared.

An occlusion of one of the pial nutrient vessels from the ophthalmic artery or of one of its intraorbital branches behind the retrobulbar part of the optic nerve (Fig. 9) produces a localized infarct and a scotoma in the region of the nerve fibres involved, but the choroidal circulation has no role to play in these lesions situated in the orbit behind the retrobulbar part.

In arteriosclerotic optic atrophy, it is usually thought that the lesion lies in the nutrient vessels of the optic nerve. I feel that it could equally well be due to arteriosclerotic changes in the posterior ciliary vessels which involve the optic nerve head and the area immediately behind the lamina cribrosa, producing nerve fibre bundle defects, optic atrophy, and cupping of the disc. This may simulate glaucoma, the mechanism of which is discussed on p. 301. In these cases there may even be sector defects and peripheral constriction (Parin, 1951; Hughes, 1958; Peters, 1958).

A posterior ciliary artery occlusion may also result from any form of arteritis (*e.g.* temporal arteritis) or embolism or arteriosclerosis. This would produce infarction of the optic disc and the adjacent optic nerve; the area involved depends upon the size of the

artery occluded, *i.e.* the occlusion of the main posterior ciliary artery involves a larger area of the optic nerve head, but involvement of one of the smaller subdivisions leads to a sectoral lesion (Hayreh, 1969). The size and distribution of the nerve fibre bundle defects depend upon the area of the optic nerve head involved. In these cases the retinal vessels are normal. Fig. 10 shows an example of a nerve fibre bundle defect, indistinguishable from glaucoma, seen in a patient with temporal arteritis and sectoral involvement of the optic disc with a normal retina.

FIG. 10 Visual field defect in the left eye of a 66-year-old woman with temporal arteritis (reproduced by courtesy of Dr. J. F. Cullen from one of his cases). Target 5/2000 white. Visual acuity 6/9

In myopia, nerve fibre bundle defects may be due either to chorioretinal atrophy near the optic disc, or to a marked peripapillary choroidal atrophy in one sector which interferes with the blood supply to the corresponding sector of the disc. These result in a Bjerrum scotoma, etc. Similarly, juxtapapillary choroiditis may produce nerve fibre bundle defects by either of the two mechanisms mentioned in myopia.

Hence any lesion destroying the peripapillary choroid in a sector would interfere with the blood supply of the optic disc and result in the nerve fibre bundle defect in the corresponding sector. Also any lesion blocking a posterior ciliary artery would produce ischaemia of a sector of the optic disc and maybe of the adjacent optic nerve and produce the nerve fibre bundle defect; in these cases a secondary peripapillary choroidal atrophy is also produced by the vascular occlusion. Hoyt (1962) described two cases of the latter variety with arcuate scotoma, peripapillary choroidal atrophy, and sectoral optic atrophy in the inferior temporal sector, which he rightly attributed to occlusion of the posterior ciliary arteries.

In incomplete occlusion of the posterior ciliary artery, the imbalance induced between the normal intraocular pressure and the low blood pressure in the disc vessels of choroidal origin would produce a classical picture of glaucoma (*see p. 301*). These cases in the past have been called "low tension glaucoma" or pseudoglaucoma (*see p. 301*). Thus, a complete occlusion of the posterior ciliary arteries produces ischaemic optic neuropathy, while an incomplete occlusion produces glaucomatous changes. In both cases the nerve fibre bundle defects are identical.

VISUAL FIELD DEFECTS IN GLAUCOMA

Classically the diagnostic visual field defect in glaucoma is the nerve fibre bundle defect. In addition to this there may be a general depression with peripheral contraction, especially

in the superior nasal quadrant, and enlargement, elongation, and barring of the blind spot. Central vision is retained until a late stage.

The subject of the pathogenesis of visual field defects, especially the nerve fibre bundle defects in glaucoma has, since their first description a century ago by Landesberg (1869) and later by Bjerrum (1889), been a matter of great interest and controversy. It is discussed in detail elsewhere (Hayreh, 1969; Hayreh, Revie, and Edwards, 1970), and only a brief résumé will be given here. Various views on the subject can be classified in two main categories:

(A) *Neurogenic origin*

This has been considered by many authors, the latest contribution being that of Smith (1965), according to whom the field defects are due to direct damage to the optic nerve fibres on the optic disc by the raised intraocular pressure and distortion occasioned by partial collapse outwards of the lamina cribrosa. Smith thought that the most important single factor is the loss of the normal plane of the lamina cribrosa, producing a sharp kinking of the nerve fibres.

(B) *Vasogenic origin*

This has been suggested by many workers*. Harrington (1964a, b) concluded that, in glaucoma, the nerve fibre bundle defects are produced by anything that upsets the normal balance between the intraocular pressure and the retinal or ophthalmic arterial pressure, resulting in decreased blood flow in the anterior optic nerve in the region of the lamina cribrosa and the arterial circle of Zinn and Haller. More or less similar was the opinion of Drance and others (1968). Hayreh (1969) concluded from experimental studies (Hayreh and Perkins, 1968, 1969) that the relationship between the blood pressure in the prelaminar centripetal vessels in the optic disc (derived from the ciliary circulation) and the intraocular pressure determines the state of patency of these vessels and that there exists a critical balance; lowering of the blood pressure or elevation of the intraocular pressure has a similar effect. This was further confirmed in subsequent studies by altering one of these two pressures and keeping the other constant (Hayreh and others, 1970). The retinal circulation did not play any role in this, which contradicts the views of Henkind (1967a, b) and those of Kornzweig and others (1968).

Fig. 11 (overleaf) shows schematically the arrangement of blood vessels in the prelaminar part of the optic disc. Their distribution is segmental in the disc, so that obliteration of these, or of the adjacent peripapillary choroid (Fig. 12) from which they arise, could produce nerve fibre bundle defects. The studies revealed that the choroidal contribution to the blood supply of the optic disc is the most susceptible to obliteration by raised intraocular pressure; next in susceptibility is the peripapillary choroid (Fig. 12). The rest of the choroid shows less susceptibility to obliteration and the retinal circulation showed no susceptibility at all. A much higher incidence of peripapillary choroidal atrophy in patients with glaucoma than is seen ordinarily in persons in the same age group seems to offer further evidence in support of this view. There is a suggestion that certain sectors of the vascular supply to the optic disc from the choroid are more susceptible than the others (Hayreh, 1969; Hayreh and Perkins, 1969).

* Troncoso (1902), Morax (1916), Elschning (1928), Magitot (1929), Lauber (1936), Evans (1939), Reese and McGavie (1942), Weekers (1942), Traquair (1944), Sjögren (1946), McGuire (1948), Beauvieux (1948), Sanna (1950), Cristini (1951), Duke-Elder (1953, 1955, 1957), Dubois-Poulsen and Magis (1955, 1956), Redsløb (1955, 1957), Gafner and Goldmann (1955), Leydhecker (1955), Dubois-Poulsen (1956), Nordmann (1956), McLean (1957), Niedermeier (1957), Harrington (1959, 1964a, b), Drance (1962a, b), Francois and Neetens (1964, 1966), Kalvin, Hamasaki, and Gass (1966), Hamasaki and Fujino (1967), Henkind (1967a, b), Drance, Wheeler, and Pattullo (1968), Ernest and Potts (1968, 1969), Alterman and Henkind (1968), Kornzweig, Eliasoph, and Feldstein (1968), Hayreh and Perkins (1968, 1969), Hayreh (1969), and others.

FIG. 11 Schematic representation of the choroidal vessels and their centripetal contribution to the prelaminar part of the optic disc

FIG. 12 Fluorescence fundus angiogram of right eye of a cynomolgus monkey after experimental occlusion of central retinal artery and 70 mm. Hg intraocular pressure. It shows obliteration of the peripapillary choroid all round the optic disc. Fluorescence of the disc is due to previous injections of fluorescein

The involvement of the peripapillary choroid in glaucoma (Fig. 12) would produce not only changes in the prelaminar part of the optic disc (Fig. 11), but also ischaemic changes in the retrolaminar part of the optic nerve, because of the recurrent pial branches arising from this part of the choroid supplying the retrolaminar part of the optic nerve (Fig. 9). Since the pial branches in this part of the optic nerve contribute to the peripheral centripetal vascular system, their involvement could produce contraction of the peripheral visual fields and ultimately the cavernous degeneration of this part of the optic nerve, both due to ischaemia of the peripheral part of the retrolaminar part of the optic nerve.

From these studies, it is postulated that (a) the site of the lesion in the nerve fibre bundle defects in glaucoma lies in the optic disc and, for the peripheral contraction of the fields, possibly in the peripheral part of the retrolaminar part of the optic nerve; and that (b) this is due to the involvement of the choroidal supply to the disc and the retrolaminar part of the optic nerve as a result of disturbed balance between the arterial blood pressure in these choroidal vessels and the intraocular pressure.

LOW TENSION GLAUCOMA OR PSEUDOGLAUCOMA

This is a type of glaucoma in which, instead of raised intraocular pressure, there is a fall in blood pressure in the ciliary vessels leading to the same end-results as glaucoma. One variety of this type of glaucoma is arteriosclerotic optic atrophy, in which the fall in blood pressure in the optic disc vessels, caused by arteriosclerotic changes in the posterior ciliary vessels, upsets the normal balance between the intraocular pressure and the blood pressure in the disc vessels. This produces glaucoma. Arteriosclerotic changes in the posterior ciliary arteries may, therefore, be a cause of glaucoma and all the associated changes and clinical findings of glaucoma, thus making arteriosclerotic optic atrophy just another variety of glaucomatous optic atrophy. Carotid artery stenosis, which produces low ophthalmic artery blood pressure resulting in low ciliary artery blood pressure, has the same effect. In these cases even emboli from the carotid plaques may occlude the posterior ciliary artery or its finer branches to the optic disc and thus produce the nerve fibre bundle defects.

From these observations one could define glaucoma as a disease in which the normal balance between the intraocular pressure and the blood pressure in the choroidal vessels supplying the optic disc and the retrolaminar part of the optic nerve is disturbed, resulting in vascular insufficiency in the optic disc and the retrolaminar part of the optic nerve, which produces visual field defects and optic disc and optic nerve changes.

PRECHIASMAL ALTITUDINAL HEMIANOPIA

In this discussion we are concerned only with those altitudinal hemianopias which are of prechiasmal origin with no abnormality in the retina. These are seen in:

Arteriosclerotic optic atrophy (Hughes, 1954; Harrington, 1964b),

Arterial hypertension.

Following massive haemorrhages (*see* p. 305),

Temporal arteritis (Schmidt, 1930; Wagener, 1946; Benedict, Wagener, and Horton, 1957; Piper and Unger, 1957; Wagener and Hollenhorst, 1958; Cullen, 1967),

Ischaemic optic neuropathy (also called arteriosclerotic papillitis, opticomalacia, vascular pseudopapillitis or papillary apoplexy) (Piper and Unger, 1957; Cullen, 1967),

Papilloedema (Walker and Cushing, 1916; Cogan, 1966),

Angioma of the optic nerve head (Hughes, 1958),

Tabes with primary optic atrophy (Walsh, 1957; Harrington, 1964b),

Arteriosclerosis of the internal carotid artery (Knapp, 1940),

Indirect injuries of the optic nerve (Hughes, 1958),

Other causes involving the optic nerve.

Hemianopia usually occurs in the lower visual field and less commonly in the upper half. The demarcation line lies horizontally and may not be absolutely straight. A generalized depression of the preserved field may also be seen.

The pathogenesis of these defects has evoked many ingenious hypotheses. The views of Wolff (1935) and Hughes (1958) are mentioned in the discussion on post-haemorrhagic amaurosis (p. 306). Piper and Unger (1957) thought that they were due to acute circula-

tory disorders of the optic nerve in post-haemorrhagic amaurosis and ischaemic optic neuropathy. They were of the opinion that occlusion of the distal part of the anterior branch of the central artery of the optic nerve caused an anaemic infarct in the lamina cribrosa region where the fibres from the upper half of the retina run. However, it has now been fairly conclusively proved that the central artery of the optic nerve is rarely if ever present in man (Hayreh, 1963a, b).

Harrington (1964b) suggested that injury to the blood supply of the optic nerve produced altitudinal hemianopia, usually inferior. He proposed a sharp division of the optic nerve into three areas based on its blood supply—superior peripheral, inferior peripheral, and central. He further stated that the vascular supply to the optic nerve came through a network of vessels in the arachnoid membrane, which passed through the subarachnoid space and entered the optic nerve at right angles. In its upper part the space is narrow and the vessels that traverse it from the membrane to the nerve are short and easily damaged by torsion, oedema, and other injury. This, according to Harrington, gives rise to a unilateral altitudinal field defect with horizontal border, steep edges, and great density. My detailed anatomical studies on the subject of the blood supply of the optic nerve (Hayreh, 1958, 1962b, 1963a,b, 1964) mentioned above, and also those of the other workers on the subject, in no way support any of Harrington's speculations. Hughes (1958) rightly pointed out that there was no evidence of a horizontal or vertical division of the blood supply of the optic nerve, apart from the central-peripheral separation. He stated, however, that these field defects strongly suggested some segmental arterial supply in the optic nerve whereby the blood supply of one horizontal half of an optic nerve may be cut off by a single lesion. In many such cases the central area of the field is spared, but in some this also is affected. He stressed that none of the anatomical work reported could provide any explanation for the clinical experience.

Walker and Cushing (1916) suggested that altitudinal hemianopia in papilloedema was due to the intracranial lesion pressing against the upper rim of the optic foramen.

The present studies have clearly demonstrated a hitherto unknown well-demarcated line of division between the upper and lower parts of the choroid and the optic disc with a horizontal boundary line (Fig. 6a and 7). Occlusion of one of the two main posterior ciliary arteries, which supplies half of the optic disc and the corresponding choroid, produces an altitudinal field defect with no retinal changes at all. In acute occlusive disorders the corresponding half of the disc will be oedematous at first, with evidence of ischaemic neuropathy in that part of the disc. Later, all cases show optic atrophy of the corresponding half of the disc. *Such a well-defined horizontal localization of blood supply exists only in the optic disc and that too in its ciliary supply and not anywhere else in the optic nerve. Thus, altitudinal hemianopias of prechiasmal origin and without any retinal lesion are caused by the occlusion of the main posterior ciliary artery which supplies that half of the disc and the choroid.* Choroidal vascular occlusion also produces death of the visual cells in the retina in that area.

It is difficult to give a definite explanation why the upper part of the choroid and optic disc circulation should be far more commonly involved than the inferior part. One can only surmise. When the distribution by the posterior ciliary artery is horizontal, probably the lower half of the choroid and optic disc are supplied by the medial posterior ciliary artery and the upper half by the lateral posterior ciliary artery, as judged from a very limited study of the sites of penetration of the sclera by these arteries (above, p. 293). As mentioned above, the medial posterior ciliary artery usually arises as the first branch of the ophthalmic artery (Fig. 8) in 53 per cent. of cases when the latter crosses over the optic nerve. Because of this the medial posterior ciliary artery probably receives more blood

and at a higher pressure than the lateral posterior ciliary artery. The fact that the central retinal artery circulation is intact in these cases would also tend to indicate intact circulation in the medial posterior ciliary artery—both having a common origin from the ophthalmic artery in 44 per cent. of cases (Fig. 8). Also, in about 30 per cent. of cases, another medial posterior ciliary artery arises from the ophthalmic artery medial to the optic nerve, both being of prominent size. It is not essential for the lateral posterior ciliary artery to be completely occluded in such cases to give rise to an inferior altitudinal hemianopia. The normal intraocular pressure would obliterate in the optic disc any vessel of choroidal origin which had a low blood pressure. In this way the vessels in half of the optic disc could be obliterated, either by complete occlusion of the main posterior ciliary artery supplying that area, or by the oblitative effect of a normal intraocular pressure on a very low blood pressure in the prelaminar vessels of that area.

PRECHIASMAL VERTICAL HEMIANOPIA

Hemianopias are always considered to be due to chiasmal or suprachiasmal lesions and are expected to be mostly bilateral. A unilateral hemianopia, when seen, is usually considered to be a precursor of the ultimate bilateral hemianopias.

Unilateral vertical hemianopia has been reported in cases with post-haemorrhagic amaurosis (Scott, 1957; Harrington, 1964b); the line of hemianopia may be irregular (Scott, 1957).

The medial and lateral posterior ciliary arteries usually supply the medial and lateral halves respectively of the choroid and the optic disc with a sharp line of demarcation between the two (Figs 1, 2, 3, and 5). The various lesions mentioned in the altitudinal hemianopias which produce occlusion of the main posterior ciliary arteries may produce a vertical instead of an altitudinal hemianopia, which may be nasal or temporal. If bilateral, it may result in homonymous or heteronymous hemianopias and may be confused with chiasmal or suprachiasmal lesions. The border of the hemianopic field, instead of passing through the fixation point, as in chiasmal and suprachiasmal hemianopias, will pass through the blind spot in the optic disc hemianopias. The nasal hemianopia, therefore, in these cases is most likely to be accompanied by a central scotoma due to the involvement of the papillo-macular bundle (Fig. 13a-c, overleaf). In a case of acute onset, the temporal or nasal half of the disc may be oedematous at first with normal retinal vessels and fundus. Later, optic atrophy of the corresponding half of the disc will be seen in all these cases.

QUADRANTIC FIELD DEFECTS

It has already been seen that the main posterior ciliary artery frequently supplies half of the choroid and optic disc, and this may be the superior or inferior half (Figs 6a and 7), or the temporal or nasal half (Figs 1, 2, 3, and 5a). Each posterior ciliary divides at some distance from the eyeball into further subdivisions. If it first divides into two, each of these subdivisions may supply a quadrant of the choroid and the disc. Such a quadrant filling of the choroid by the dye was seen in the present study (Figs 5b and 6b). Occlusion or stenosis of such a subdivision due to any cause would lead to a quadrant field defect. During acute vascular occlusion of such a subdivision with resultant infarction, that quadrant of the disc may be oedematous, but later it will be atrophic. The possibility of bilateral quadrant defects due to this cause has to be borne in mind.

FIG. 13 A 67-year-old woman developed a sudden loss of central vision in the right eye one afternoon 18 months ago, which has since remained unaltered. Visual acuity in that eye is now counting fingers in the temporal field; the right disc shows atrophy of the temporal half with a normal nasal half and the retinal vessels show a generalized narrowing—particularly the arteries (a). The right peripheral visual fields show a nasal hemianopia with the border of the hemianopic field passing through the blind spot (b). The patient was unaware of this right nasal hemianopia. The left eye is normal (c).

The involvement of still smaller subdivisions of the posterior ciliary artery, supplying a sector of the optic disc, would produce a sectoral field defect.

PERIPHERAL CONTRACTION OF VISUAL FIELDS

The centripetal and centrifugal vascular systems of the optic nerve explain the presence of central and peripheral visual field defects in vascular lesions (Hughes, 1958). The commonest types of field loss in arteriosclerotic optic atrophy are the multiple indentations in the peripheral field, and these most commonly affect the lower field (Hughes, 1958). In tabes the peripheral contraction proceeds towards the centre (Walsh, 1957).

Depression of the peripheral fields in glaucoma is a well-known phenomenon and its pathogenesis is discussed above (p. 300). As mentioned on p. 301, arteriosclerotic optic atrophy seems to be a variety of glaucomatous optic atrophy, with nerve fibre bundle defects and peripheral depression of the fields. The additional factor of involvement of the pial vessels by the arteriosclerotic process may aggravate and complicate the picture. In tabes the peripheral contraction is most probably due to involvement of the pial vessels by a syphilitic process.

POST-HAEMORRHAGIC AMAUROSIS

A large number of cases has been reported in the literature in which visual disturbances, even complete blindness, followed a marked loss of blood. It is interesting to note that Hippocrates was also aware of this when he wrote: "After the vomiting of blackish sometimes bloody matter, the patient complains of headache and eyes do not see". The mechanism of the visual disturbances and the field defects, particularly the altitudinal hemianopias, is still unknown.

Amaurosis is mostly known to follow haemorrhages from the gastrointestinal tract and uterus and less often from the nose, lungs, kidneys, wounds, etc. (Singer, 1904; Pröll, 1907; Bistis, 1908; Alt, 1912; Hegner, 1912; Calhoun, 1913; Pincus, 1919; Görlitz, 1920; Terrien, 1921; Puppel, 1924; Harbridge, 1924; Fink, 1924; Grimminger, 1925; Whiting, 1929; Pines, 1931; Wolff, 1935; Long, 1943; Levatin, 1947; Locket, 1949; Pears and Pickering, 1960; Waldeck, 1960; and others). Small recurrent haemorrhages rather than a single larger one, are mostly responsible for this process.

The visual disturbance varies from blurring to complete loss of vision. It is generally bilateral (in about 90 per cent., Coston, 1935; in 85 per cent., Duke-Elder, 1940). It may be permanent (in 54 per cent., Pergens, 1896; in 45.9 per cent., Singer, 1904; in 50 per cent., Terson, 1922; in 83 per cent., Tidy, 1941) or temporary. It usually starts between the third and seventh days after the haemorrhage (Terson, 1922; Wolff, 1935), but may develop within a few hours (within 24 hours in 30 per cent., Pergens, 1896; within 14 hours in 45 per cent., Groenouw, 1904; within 12 hours in 34.1 per cent., Terson, 1922). The literature has been reviewed by Singer (1904), Terson (1922), Harbridge (1924), and Locket (1949).

Clinically, the fundus is pale and anaemic with narrow retinal arteries. The optic disc may show blurred margins (Whiting, 1929; Locket, 1949; Walsh, 1957; and others) or oedema (Whiting, 1929; Goulden, 1929; Barr, 1934; Wolff, 1935; Ballantyne, 1935; Locket, 1949, and others) which may be marked and may involve only the upper half of the disc (Ballantyne, 1935). There may be small superficial haemorrhages near the optic disc. The retinal veins may be congested. Later all these cases show optic atrophy (Hirschberg, 1881; Ziegler, 1887; Raehlmann, 1889; Görlitz, 1920; and many others), which may involve only the upper half of the disc (Uhthoff, 1922; Ballantyne, 1935). There may, however, be pronounced loss of vision without any significant fundus changes during the early stages (Locket, 1949; Müller, 1953; Walsh, 1957; and Hughes, 1958), though later optic atrophy is almost always seen.

Altitudinal hemianopia with loss of the lower visual fields has been seen by von Graefe (1860), Grout (1914), Pincus (1919), Terrien (1921), Uhthoff (1922), Collins (1929), Wolff (1935), Locket (1949), Hughes (1954, 1958), Comberg (1956), Scott (1957), Piper and Unger (1957), Ferreira and Parreira (1957), Gálvez Montes (1959), Harrington (1964b), and others. Less commonly it may involve the loss of the upper visual fields (Hughes, 1958). In these cases, the hemianopia is irregular and the line of demarcation is along the horizontal meridian. The preserved field shows a generalized depression (Hughes, 1958). There may be vertical hemianopia (Scott, 1957; Harrington, 1964b), bitemporal hemianopia (Harrington, 1964b), concentric contraction of fields (Grout, 1914; Goulden, 1929; Harrington, 1964b), or central scotoma (Grout, 1914; Barr, 1934). Scott (1957) described wide sectoral field defects in these cases which may produce irregular hemianopias, either vertical or horizontal.

The pathological lesions described in these cases are not definite. Zeigler (1887), Seese (1914), and Görlitz (1920) reported them 22, 5, and 11 days respectively after the

onset of blindness. They found degeneration of ganglion cells, cytoid bodies in the nerve fibre layer, oedema of the retina, and fatty degeneration in the optic nerve. Görlitz (1920) found the last change in the optic nerve situated behind the lamina cribrosa.

The pathogenesis of post-haemorrhagic amaurosis, particularly that of the altitudinal and other field defects, has been the subject of much controversy. It has been considered to be due probably to primary ischaemia of the retina (Ziegler, 1889; Collins, 1929; Langdon, 1933; Cordes, 1958), focal anoxia of the retina (Pears and Pickering, 1960), vasospasm of the retinal arteries (Hartmann and Parfonry, 1934; Wolff, 1935), lack of oxygen similar to quinine amblyopia (Wolff, 1935), central retinal vein thrombosis (Cox, 1944), or ischaemic fatty degeneration of the optic nerve (Westhoff and Ziegler, 1899). Its prevalence after repeated haemorrhages and the lapse of time between the haemorrhage and the visual disorders suggest the possible role of some haemoclastic shock, anaphylactic crisis, or toxæmia due to profound haemolysis or other unknown causes (Holden, 1899; Groenouw, 1904; Pincus, 1919; Terrien, 1921; Terson, 1922; Kummel, 1932; Rønne, 1932; Duggan, 1943; and others). Duggan (1943) thought the toxin was adrenaline. Riser, Felgines, Gayral, Géraud, and Ribaut (1951) suggested that auto-intoxication was added to ischaemia to cause visual loss. Terrien (1921) thought the optic atrophy was due to the combined effect of haemorrhage and toxæmia. Other workers have suggested haemorrhage into the optic nerve sheath (von Graefe, 1860), oedema and multiple haemorrhages in the nerve (Leber, 1877), thrombotic foci causing degeneration and diffuse retinal oedema (Görlitz, 1920), endothelial damage due to blood loss and thrombosis on damaged endothelium (Uhthoff, 1922), endarteritis fibrosa (Raehlmann, 1889), thrombosis of the central retinal artery (Theobald, 1899), retrobulbar neuritis (Hoffman, 1899), a lesion in the optic nerve just behind the lamina cribrosa (Pears and Pickering, 1960), exudation into the sheath of the optic nerve with associated cerebral oedema sufficient to produce compression of the vascular supply of the optic nerve and retina (Fisher, 1929), changed consistency of the blood (Wilbrand and Saenger, 1906), and hypoproteinaemia leading to oedema of the optic nerve and vascular compression (Locket, 1949).

Inferior altitudinal hemianopia, according to Wolff (1935), is due to the effect of gravity, the lower part of the retina receiving more blood than the upper part after severe haemorrhage. There is accompanying atrophy of the upper half of the discs and diminution in calibre of the upper retinal arteries. Wolff postulated that the oedema of the optic disc in these cases was due to Starling's phenomenon, wherein the cutting off of the blood supply injures the endothelium of the capillaries, so that when the blood pressure is restored in them, an excessive amount of fluid is allowed to pass through the walls and produces oedema. Hughes (1958) commented that, in altitudinal hemianopia, the horizontal line of demarcation would suggest a retinal origin. In one example he observed the fundus during the episode and found the retinal vessels to be normal. In this case the horizontal line of demarcation did not exactly follow the horizontal meridian and it seemed probable that the lesion lay in the optic nerve. He reported another similar case. Harrington (1964b) thought it to be due to ischaemia of the anterior part of the optic nerve secondary to extreme hypotension or to stenosis or embolism of the optic nerve branches from the ophthalmic artery. Walsh (1957) postulated thrombosis affecting the visual cortex or optic radiation due to a fall in blood pressure in cases in which the fundus was normal.

The possibility that in some cases the visual disturbances, especially total blindness, are due to occlusion of the central retinal artery cannot be ruled out. In these, however, a normal fundus and normal retinal vessels have frequently been reported, and one can

hardly find a case report showing a fundus picture of classical central retinal artery occlusion. The presence of an extreme arterial hypotension has been reported in these cases. The altitudinal hemianopia seen frequently in these cases has, I feel, the same pathogenesis as that discussed above (p. 302), *i.e.* it is due to involvement of the choroidal circulation to one half of the disc and choroid (Figs 6a and 7). In some cases, this could be the result of thrombosis affecting the posterior ciliary vessels because of extreme arterial hypotension. *The presence of an isolated oedema of the optic disc without any retinal oedema can be produced only by occlusion of the ciliary supply to the disc* (Hayreh, 1969). The presence of vertical hemianopia in these cases can sometimes be explained on the same basis as altitudinal hemianopia, where the main posterior ciliary artery supplies the temporal and nasal halves (Figs 1, 2, 3, and 5) instead of the superior and inferior halves (Figs 6a and 7). Since smaller subdivisions of the posterior ciliary arteries supply small sectors of the disc, their individual involvement would lead to a sectoral defect, as discussed above.

Conclusions

- (1) The posterior ciliary arteries have a segmental supply in the choroid and optic disc. The main posterior ciliary artery usually supplies half of the choroid and optic disc, more commonly the nasal or temporal half, less often the superior or inferior half. The smaller short posterior ciliary arteries supply smaller sectors which may be quadrantic or smaller. The segments are usually sharply demarcated.
- (2) The main source of blood supply to the optic disc comes from the ciliary circulation. The prelaminar part of the disc is supplied by the peripapillary choroid.
- (3) The recurrent pial branches from the peripapillary choroidal vessels and the circle of Zinn and Haller contribute to the peripheral centripetal vascular system of the retrolubar part of the optic nerve. The axial centrifugal vascular system in this part of the nerve is usually formed by the central retinal artery.
- (4) Occlusion of one of the posterior ciliary arteries or one of its smaller subdivisions through any cause would involve a sector of the lamina cribrosa, the prelaminar part of the optic disc, and the retrolaminar part of the disc. This would lead to a nerve fibre bundle defect, the size and shape of the defect depending upon the artery involved.
- (5) A complete occlusion of the posterior ciliary artery produces ischaemic optic neuropathy, while an incomplete occlusion, *e.g.* in arteriosclerosis, produces glaucomatous changes in the optic disc and nerve.
- (6) Visual field defects in glaucoma are vasogenic in origin through involvement of the nerve fibres in the optic disc and nerve secondary to choroidal vascular obliteration.
- (7) Glaucoma may be defined as a disease in which the normal balance between the intraocular pressure and the blood pressure in the choroidal vessels supplying the optic disc and the retrolaminar part of the optic nerve is disturbed, resulting in vascular insufficiency in the optic disc and retrolaminar part of the optic nerve, which in turn produces the visual field defects and the optic disc and optic nerve changes.
- (8) Low-tension glaucoma is a type of glaucoma in which, instead of raised intraocular pressure, there is a fall in blood pressure in the ciliary vessels leading to the same end-results as in glaucoma.
- (9) Prechiasmal altitudinal hemianopia may be produced by occlusion of one of the main posterior ciliary arteries which supplies the upper or lower half of the choroid and optic disc.

(10) Prechiasmal vertical hemianopia may be produced by occlusion of one of the main posterior ciliary arteries which supplies the nasal or temporal half of the choroid and optic disc.

(11) Quadrantic or sectoral field defects may be produced by occlusion of one of the smaller subdivisions of the posterior ciliary artery. The size and shape of the defect depends upon the artery involved.

(12) Peripheral contraction of the visual fields is frequently produced by the involvement of the pial vessels of the optic nerve which constitute its centripetal peripheral vascular system. The pial vessels of choroidal origin in the retrolaminar part of the optic nerve may be involved in glaucoma secondary to the involvement of the peripapillary choroid and this may result in peripheral contraction of the field in glaucoma.

(13) In post-haemorrhagic amaurosis, the visual disturbances and visual field defects, including altitudinal hemianopia, may frequently be due to the involvement of the ciliary blood supply to the optic disc and choroid. The type of defect depends upon the ciliary artery involved.

Summary

In the present study the choroidal vascular pattern and its contribution to the optic disc was studied in 24 eyes of monkeys by rapid serial fluorescence fundus angiography. This revealed a segmental supply by the posterior ciliary arteries in the choroid and optic disc. The size of the segment depended upon the size of the artery. The main posterior ciliary artery usually supplies half of the choroid and optic disc, which is more often the temporal or nasal half and less commonly the upper or lower half. The smaller arteries supply smaller sectors of these segments, which may be quadrantic.

The ciliary circulation has a very important role to play both in the pathogenesis of different types of visual field defects, most of which have so far remained ill-understood, e.g. nerve fibre bundle defects of different origins, visual field defects in glaucoma, altitudinal and vertical hemianopias of prechiasmal origin, quadrantic and sectoral defects, and in post-haemorrhagic amaurosis.

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