Vascular perfusion pressure gradients in the eye*

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The intraocular pressures required to terminate blood flow in the retina, choroid, and anterior uvea were compared. The retinal and choroidal circulations were studied by ophthalmodynamometry while the choroidal and anterior uveal circulations were compared by direct cannulation experiments. The results indicated that as intraocular pressure is gradually elevated toward ophthalmic artery pressure, blood flow ceases in a sequential pattern in the anterior uveal, choroidal, and retinal circulations. Systolic and diastolic blood pressures were 14 and 13 mm Hg lower, respectively, in the choroid than in the retina. Circulation ceased in the anterior uvea at levels of intraocular pressure 5 to 20 mm Hg lower than that required to terminate blood flow in the choroid. The relationship of these results to vascular perfusion pressure in various segments of the eye is discussed.

Key words: perfusion pressure, iris artery pressure, choroidal venous pressure, ophthalmodynamometry, ophthalmic artery pressure.

Recently, a technique has been described for performing fluorescein angiography at acutely elevated levels of intraocular pressure.1,2 Studies of this nature in man demonstrate that at levels of intraocular pressure just below systolic ophthalmic artery pressure, retinal arterioles and capillaries fluoresce while no dye appears in the choroidal vasculature.2,3 At lower intraocular pressures, choroidal fluorescence occurs but fluorescence of the iris vasculature may be prevented.4 This differential susceptibility of the retinal, choroidal, and iris vasculature to elevated intraocular pressure may indicate that there is a progressively lower perfusion pressure to each of these three circulations. In the presence of such a perfusion pressure gradient, blood flow would be terminated in a progressive sequence in the three vascular beds when intraocular pressure is gradually elevated toward systolic ophthalmic artery blood pressure. This hypothesis was tested in the present investigation by comparing the in-
traocular pressures at which blood flow ceased in the retina, choroid, and anterior uvea in albino rabbits. The retinal and choroidal circulations were studied by ophthalmodynamometry, while the choroidal and anterior uveal circulations were compared by direct cannulation experiments.

Materials and methods

Albino rabbits weighing 3 to 4 kilograms were anesthetized with intravenously administered pentobarbital sodium, 30 mg. per kilogram. Topical anesthesia was effected with proparacaine hydrochloride.

Ophthalmodynamometric studies were performed in 14 rabbits after dilating the pupil of one eye with 10 per cent phenylephrine hydrochloride and 1 per cent cyclopentolate hydrochloride. The anterior chamber was cannulated with a 23 gauge needle connected to a reservoir of heparinized saline and intraocular pressure was monitored with a pressure transducer and recorded on a dynograph. The central retinal artery and juxtapapillary choroidal vessels were observed with an indirect ophthalmoscope and +14D lens as intraocular pressure was gradually increased in a stepwise fashion.

In abino rabbits, the observer could easily visualize large choroidal arteries adjacent to the disc. Distinct and repeatable end-points for systole and diastole could be obtained despite the rabbits' rapid heart rate. The diastolic end-point was considered to be the first frank pulsation of the central retinal artery at the disc for the retinal circulation. Intraocular pressure was then rapidly elevated until all pulsations ceased, following which intraocular pressure was gradually decreased until the first frank pulsations reappeared. This was considered the systolic end-point. Three determinations of systolic and diastolic end-points were performed in each eye and the mean value recorded.

The choroidal and anterior uveal circulations were compared by cannulating the temporal long posterior ciliary artery and one vortex vein in 10 rabbits (Fig. 1). After removing the lateral orbital wall and disinserting the lateral rectus muscle, a tapered polyethylene PE-10 cannula was inserted into the temporal long posterior ciliary artery just posterior to its entrance into the sclera. The cannula was threaded anteriorly within the artery under microscopic observation so that its tip rested over the ciliary body in proximity to the greater arterial circle of the iris. The intraluminal position of the cannula tip was ascertained by injecting saline through the cannula and blanching the iris arteries. The superotemporal vortex vein was then isolated and a polyethylene PE-10 cannula was inserted with its tip pointing toward and situated just outside the globe. After successful cannulation was ascertained by steady reflux of blood from both cannulas, polymethylacrylate adhesive was applied to the sites of cannulation and the cannulas were connected to pressure transducers and a dynograph. The ciliary artery cannula reflected pressure in the greater arterial circle of the iris which was supplied by the medial long posterior ciliary and anterior ciliary arteries.5 The capability of these vessels to adequately maintain perfusion to the iris arterial circle was shown by the steady reflux of blood that occurred from the ciliary artery cannula when it was disconnected from the transducer. The vortex vein cannula measured effluent choroidal venous pressure.

The anterior chamber was cannulated with two 23 gauge needles, one connected to a reservoir of heparinized saline to alter intraocular pressure, the other to a pressure transducer and recorder to monitor intraocular pressure. Records were then made of iris artery pressure (IAP) and effluent choroidal venous pressure (ECVP) as intraocular pressure was increased from physiologic levels in a stepwise fashion.
Table I. Systolic and diastolic measurements in central retinal and juxtapapillary choroidal artery*

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<th>Retina</th>
<th>Choroid</th>
<th>Differencef</th>
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<tr>
<td>Systole</td>
<td>105</td>
<td>92</td>
<td>14 ± 2.8</td>
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<tr>
<td>Diastole</td>
<td>82</td>
<td>69</td>
<td>13 ± 3.1</td>
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*Mean values from 14 experiments.
†Mean difference ± S.E. between retinal and choroidal values.

Results

Table I lists the mean systolic and diastolic blood pressures in the central retinal artery and in a juxtapapillary choroidal artery, determined by ophthalmodynamometry. In all animals, systolic and diastolic blood pressure were significantly higher in the retinal circulation, the mean difference being 14 mm. Hg for systole and 13 mm. Hg for diastole.

Fig. 2 shows the typical response of iris artery pressure (IAP) to stepwise elevations of intraocular pressure. During steady state physiologic conditions, IAP was an average of 7.8 ± 2.8 (S.D.) mm. Hg above intraocular pressure. When intraocular pressure was elevated, IAP increased simultaneously but transmural iris artery pressure (IAP – IOP) decreased progressively. When transmural pressure approached zero, cessation of blood flow in the anterior uveal circulation occurred and was indicated by an abrupt decline in IAP. Lowering intraocular pressure slightly at this point permitted circulation to re-establish itself with a consequent recovery of IAP to its previous level (Fig. 3). The IOP at which cessation of blood flow occurred was termed the collapse pressure. Careful observation of the tip of the ciliary artery cannula through the operating microscope demonstrated that pulsation of the blood column in the cannula ceased abruptly when IOP was elevated to the collapse pressure for the iris arterial circle. There was no visible deformation or compression of the cannula tip to account for the sudden reduction of IAP.

Fig. 4 compares the response of IAP and effluent choroidal venous pressure (ECVP) to stepwise elevations of intraocular pressure. At all levels of intraocular pressure, ECVP exceeded IAP. When intraocular pressure was elevated to levels that reduced transmural iris artery pressure to the vicinity of zero, IAP abruptly declined while ECVP was maintained at an elevated level that exceeded intraocular pressure. These results indicate that blood flow in the anterior uveal circulation ceased at levels of intraocular pressure that did not occlude the choroidal circulation. In order to terminate blood flow in the choroidal vasculature and cause ECVP to abruptly decline, IOP had to be elevated 5 to 20 mm. Hg above that at which IAP was first noted to abruptly decline. The mean difference in collapse pressure between the anterior uveal and choroidal circulations was 9.8 ± 6.9 (S.D.) mm. Hg.

Fig. 5 depicts the relationship between intraocular pressure, mean IAP and mean ECVP in 10 experiments.

Discussion

Blood flow through a vascular bed is modified by the balance between intraluminal blood pressure and extravascular tissue pressure. When this balance, termed the transmural pressure, falls below a criti-
IOP
mm Hg

IAP
mm Hg

Fig. 3. Record of IOP and IAP. After IOP was elevated to 70 mm. Hg, abrupt decline of IAP at A indicates cessation of blood flow in the anterior uveal circulation. When IOP was then reduced to 60 mm. Hg at B, circulation was re-established and IAP recovered to its previous level. This sequence of events was repeated at C and D.

Fig. 4. Record of IOP, IAP, and ECVP. Both IAP and ECVP increase progressively after each stepwise elevation of intraocular pressure. When intraocular pressure was elevated to 90 mm. Hg, abrupt decline of IAP at A indicates cessation of flow in the anterior uveal circulation while choroidal circulation continued to maintain the elevated level of ECVP. Following saline injection into the ciliary artery cannula at B to ascertain that clotting had not occurred, anterior uveal circulation was re-established temporarily but cessation of flow reoccurred at C. 1 and 2 indicate injection of saline into the vortex vein cannula to ascertain that clotting had not occurred. At an intraocular pressure of 100 mm. Hg, choroidal circulation was able to maintain an ECVP of 105 mm. Hg.

cal value, the blood vessels will collapse and blood flow will cease. Intraluminal blood pressure generally rises in response to elevations of extravascular tissue pressure and the maximum level to which it can increase is determined by the perfusion pressure to the vascular bed. In the eye, this is equal to systolic ophthalmic artery blood pressure and it has been demonstrated in the cat that ocular circulation
ceases when intraocular pressure is elevated to within 6 mm. Hg of ophthalmic artery pressure. The present study demonstrated, in addition, that as intraocular pressure is gradually elevated toward ophthalmic artery pressure, blood flow ceases in a sequential pattern in the anterior uveal, choroidal, and retinal circulations.

In the present study the retinal and choroidal circulations were compared by ophthalmodynamometry which revealed that systolic and diastolic end-points were visualized at significantly lower intraocular pressures in the choroid. The choroidal and anterior uveal circulations were compared by inserting a cannula into a vortex vein to measure ECVP and into the temporal long posterior ciliary artery to measure pressure in the greater arterial circle of the iris (IAP). Previous neoprene injection studies of the anterior uvea in cats have demonstrated that the arterial circle of the iris is a continuous ring vessel of large diameter which proceeds uninterrupted circumferentially within the iris from one long posterior ciliary artery to the other. India ink injection studies from this laboratory have demonstrated that the anatomy of the rabbit anterior uveal circulation is similar. Because of the large size and the continuity of the iris arterial circle, it would appear that the methods used to measure IAP in this study are truly representative of the pressure exerted by the opposite long posterior ciliary artery on the iris arteries.

Both ECVP and IAP increased in response to elevations of intraocular pressure as long as blood flow continued in the choroid and anterior uvea. When intraocular pressure was elevated sufficiently, however, a level was reached at which ECVP or IAP abruptly declined. This level of intraocular pressure was termed the collapse pressure for the choroidal or iris arterial circulation, respectively. The present study indicates that the collapse pressure for the iris arterial circulation is 5 to 20 mm. Hg less than that for the choroidal circulation.

These results indicate that the maximum perfusion pressure available to these three intraocular circulations differ. If systolic ophthalmic artery blood pressure is indicated by ophthalmodynamometry of the central retinal artery as pointed out by other investigators, then vascular perfusion pressure to the choroid is 14 mm. Hg less than ophthalmic artery pressure in the rabbit and the anterior uveal circulation is perfused at a still lower level. Similar findings have been demonstrated in human eyes by performing fluorescein angiography during induced ocular hypertension. Iris fluorescence can be prevented by intraocular pressures slightly above diastolic ophthalmic artery blood pressure in normotensive subjects, whereas choroidal circulation is maintained until intraocular pressure is elevated to within 6 mm. Hg of systolic ophthalmic artery blood pressure. It appears, therefore, that the full force generated by blood flow in the ophthalmic artery is not available to drive intraocular uveal blood flow. One explanation for this finding might be that the passage of ciliary blood vessels through the sclera creates sites of high resistance that
cause a significant fall off of blood pressure. The longer intrascleral course of the long as compared to the short posterior ciliary arteries would cause a larger decline in perfusion pressure to the anterior uveal circulation than the choroid. This would account for the lower collapse pressure in the anterior uveal than the choroidal circulation and would explain the remarkably low transmural iris artery pressure found in this study.

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REFERENCES