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OUTFLOW

III

The Choroidal Circulation

In contrast to the retinal vessels which are readily observed through the ophthalmoscope and with the slit lamp, the arteries and veins of the choroid are concealed behind the retinal pigment epithelium, and our knowledge of the choroid is derived largely from laboratory investigation. Nonetheless there are a few observations that we may make ourselves. With the ophthalmoscope we see coursing anteriorly along the temporal and nasal meridian of the eye the long posterior ciliary arteries each accompanied by a long posterior ciliary nerve. The choroidal veins are outlined in broad silhouettes as they converge in stellate or perhaps pinwheel configuration onto the outflow channel, hence the name "vortex". The ocular pulse, which is thought to be caused by the choroid, is reflected in the "spontaneous" pulsation of the retinal veins on the disc that we discussed in previous issues of the Glaucoma Letter, and in the oscillating intraocular pressure that is so easily demonstrated with tonometry or tonography. At the time of retinal detachment surgery the emissary channels from the vortex veins are readily identified on the surface of the sclera. One searches for them then to protect them from accidental injury. In eyes with atrophic retinal pigment epithelium the large vessels of the choroid are clearly visible. In that situation one may study individual vessels with contact lens and slit lamp. However, where the choroid is seen distinctly, the retina must be atrophic. The same insult that injured the retina is likely to have damaged also the choroid; and even if this were not the case, it is likely that atrophy of the retina itself would induce changes in the overlying tissue. We have no opportunity, therefore to observe the intact choroidal circulation directly.

Blood from the choroidal capillaries drains into the vortex veins and these pass through the sclera to empty into branches of the ophthalmic vein. From its location inside the scleral envelope, we infer that the entire intra-ocular choroidal circulation is potentially subject to the intraocular pressure. We know also, that on the outside of the globe, the pressure in the ophthalmic vein into which the vortex circulation drains is relatively much lower than the intravascular pressure of the choriocapillaris. As in the case of the retinal circulation, we infer the existence of a pressure gradient. But while we can directly observe the collapse of the veins on the disc that accounts for the pressure gradient in the retinal venous drainage, the location of the pressure gradient of choroidal outflow is not immediately apparent.

The retinal vein leaves the globe in a direction perpendicular to the scleral surface, The vortex veins, on the other hand, take an oblique course through the sclera. This anatomic configuration has led to the theory that with increasing intraocular pressure there is progressive compression of the vortex veins, producing an increased resistance to flow and thereby preventing the pressure-induced collapse of the choriocapillaris. Intrascleral pressure on these obliquely coursing veins was thought to create the necessary resistance to prevent the emptying of the choroidal vascular bed. It is an elementary postulate of physics that the degree to which the vortex vein coursing through the sclera will be compressed by any given intraocular pressure is a function of the fluid pressure within the vein, of the orientation of the vein, of the depth at which the vein is located within the sclera, and of the elastic properties of the sclera.

An analogous situation obtains with respect to the collector channels from the Canal of Schlemm that traverse the sclera to drain into the aqueous veins. From time to time the question is raised whether compression of these vessels might not account for at least part of the resistance to aqueous outflow. This problem has recently been in investigated by John L. Battaglioli at the Massachusetts Institute of Technology in a thesis, "The Role of Vessel Collapse on the Flow of Aqueous Humor," published in June 1981. Although

Battaglioli studied outflow resistance from the Canal of Schlemm, his results are directly applicable to the dynamics of vortex vein drainage, provided one admits the plausible assumption that there is no great difference in the elastic moduli of the sclera of the posterior pole from the elastic moduli of the sclera adjacent to the limbus.

One can readily visualize that with an increase in intraocular pressure the sclera stretches. The amount of stretching for any given increase in pressure is governed by the tensile modulus of the sclera. As the sclera stretches, it also becomes thinner. This thinning is considered identical with the compression which might occur if, for instance, the sclera were compressed between two rigid plates. The amount of thinning for any given increase in pressure is governed by a physical characteristic of the sclera which is called its compressive modulus. Battaglioli found that although a number of measurements of the tensile modulus of sclera have been published, its compressive modulus was unknown. He built a small hydraulic press with which he measured the compressive modulus of elasticity of the sclera. Using this instrumentation Battaglioli found the compressive modulus to be 3.3×10^5 dynes/cm^2 compared with a tensile modulus of 3.3×10^7 dynes/cm^2. These figures indicate that it requires one hundred times as much force to stretch as to compress the sclera by a given fraction, a ratio which on first thought is surprising, but then, on second thought seems not incompatible with the clinical experience of those of us who have had occasion to dissect and suture sclera in the operating room.

To estimate the magnitude of the stresses induced in the sclera by the intraocular pressure, Battaglioli relied on equations published in 1934 by Timoshenko. (S.P. Timoshenko and J.N. Goodier, "Theory of Elasticity", N.Y., 1934) For an eye 24 mm in diameter, the tangential stress ranges from 5.5 times the intraocular pressure at the inner surface of the sclera to 5 times the intraocular pressure at the outer surface. The radial stress, on the other hand, ranges from a value equal to the intraocular pressure on the inner surface to zero at the outer surface, this last calculation coinciding nicely with untrained intuition.

Battaglioli then made a mathematical analysis of the forces governing the behavior of a vessel coursing tangentially in the sclera. He determined that the deformation of the vessel within the sclera could be expressed in terms of seven parameters: tangential stress, radial stress, tangential modulus, compressive modulus, Poisson's ratio, pressure within the vessel, and its undeformed cross-section. These parameters he then grouped into four dimensionless ratios that could be matched with a large scale experimental model in all respects except one. He was unable to find a substance with the same disparity between compressive and tensile moduli as sclera. He compromised by using a closed cell foam made of vinyl chloride rubber, which resembled the sclera in that the ratio of its tensile modulus to its compressive modulus was much greater than 1. For the foam, this ratio was 22.5; for the sclera, as mentioned earlier, it is 100. He then fashioned an 8" x 8" x 12" rectangular block of the foam and drilled a 3/8" diameter hole at right angles to the 8" x 8" surface. Into the hole was inserted a thin plastic tube filled with electrically conductive fluid, making it possible at one and the same time to control the pressure within the simulated vein and to measure its cross-section. Forces were applied to the foam through rigid plates glued to its surfaces. Thus it was possible to study how external pressures on the foam would change the shape of the tube that passed through it. When the externally applied forces were scaled to correspond to the calculated stresses that various intraocular pressures would induce in the sclera, it became possible to make inferences concerning their effect on trans-scleral outflow channels.

With this experimental set-up, Battaglioli was able to show that for a tangentially oriented vessel, depending on its length and location, scaled pressures of 35 to 55 mm Hg would be required before the induced scleral deformation led to a significant pressure drop. For vessels that course not tangentially but obliquely, the external pressure required to collapse the tube becomes much higher. If the vessel traverses the sclera in a direction radial to the globe, then increasing the intraocular pressure will cause the vessel not to collapse, but to dilate.

Battaglioli was concerned with the dynamics of aqueous outflow. He concluded that resistance in the collector channels did not contribute to the total outflow resistance of the eye except possibly at pathologically elevated pressures. His results, however, are immediately applicable to the question of whether there may be a pressure drop in the vortex veins as they traverse the sclera. Battaglioli's findings strongly suggest that this is not the case, and that under physiologic conditions, the scleral tunnel is not the locus of resistance to vortex vein outflow.

Battaglioli's thesis suggests that it may be worthwhile to take another look at the choroidal circulation, obscured though it is by the pigment epithelial barrier.

When one looks at the fundus with the ophthalmoscope or with the slit lamp and the contact lens, one sees broad bands of choroidal vein like the curved spokes of a wheel converging onto the exit foramen. On first thought one accepts the profile of these vessels as indicative of a large volume, consistent with the well understood vascularity of the choroid. Yet the observation that in the case of the retina, broad venous profiles represent not engorged but partially collapsed vessels, raises the question whether or not the breadth of the choroidal veins might not reflect partial collapse rather than engorgement. This suspicion appears to be confirmed in the occasional situation when, because of atrophy of the retinal pigment epithelium one has an opportunity to study choroidal veins stereoscopically with the slit lamp. One sees then that at least some, and perhaps most of the choroidal veins whose contours are visible with the ophthalmoscope far from being cylindrical, are actually flat ribbons. Only occasionally does one see a choroidal vein that is even partially distended. Fluorescein angiography of areas in the fundus that have been denuded of pigment epithelium shows that the choroid's broad veins often fill with only a thin layer of blood.

It follows that unlike the healthy retinal venous vascular bed whose volume remains practically unaffected by changes in the intraocular pressure, the volume of the choroidal venous vasculature is unstable. The collapse of a choroidal vein implies that its contents have been partially expressed, and the volume of the choroidal venous plexus therefore may be expected to vary inversely with the intraocular pressure. The fluctuation of choroidal volume with intraocular pressure will be reflected in fluctuating meridional stresses of the choriocapillaris and of Bruch's membrane as spherical shells. When the intraocular pressure decreases, the volume of blood in the choroidal veins increases, the inner layers of the choroid are displaced centrally, and the stress induced in them by the intraocular pressure diminishes. On the other hand, if the intraocular pressure increases, then the choroid flattens, and Bruch's membrane, displaced toward the periphery of the globe, becomes correspondingly stretched. This tension in the choroid is transmitted to the ciliary body to which the choroid is anchored, and through it to the scleral spur. With an increase in intraocular pressure then, the choroid tends to collapse, its inner layers are pushed outward by the intraocular pressure, there is increased meridional stress which makes posterior traction on the scleral spur, and which, analogous to the effect of a miotic, will tend to open the canal of Schlemm and restore the intraocular pressure to a more normal level. It is unknown what role, if any, this mechanism might have in the control of the intraocular pressure.

There is a second corollary to these considerations. Just as the collapsed segments of retinal vein provide a low pressure drainage system to the temporal and nasal portions of the disc, so the collapsed segments of choroidal vessel offer a low pressure sink for those venules and capillaries of the choroid that drain into them. We do not have sufficient anatomic or physiologic data to draw conclusions about the extent of this drainage, but it may constitute a substantial proportion of the choroidal circulation. In any event, the low pressure choroidal veins and such capillary tributaries as they may have constitute potentially a second outflow system for aqueous. It is held that as much as 20% of the aqueous drains from the anterior chamber angle into the ciliary body and choroid, whence it is eliminated from the eye by mechanisms unknown. The classical postulate that the site of choroidal outflow resistance is the compressed vortex vein within the sclera entails the inference that the entirety of the choroidal circulation within the globe should be above intraocular pressure. Aqueous, therefore, if it were to enter the choroidal circulation, could do so only by negotiating a pressure gradient. Thus one should also have to postulate the existence of an osmotic or of an active transport mechanism to convey aqueous into the choroidal capillaries, or alternatively one should have to assume some anatomically obscure extravascular pathway by which the uveo-scleral flow might escape from the globe, avoiding the choroidal capillaries and veins. The presence within the choroid of a network of collapsed vessels whose contents are under less than intraocular pressure may help to explain how aqueous leaves the eye by the uveo-scleral route.

The existence of such an intraocular network of collapsed vessels in the outflow segment of the choroidal circulation may also shed some light on that troublesome pathophysiologic phenomenon, the choroidal effusion. It is likely that the accumulation of fluid in the suprachoroidal space in many instances reflects the diminution or loss of the gradient between the intraocular pressure and the pressure in the choroidal veins. Choroidal effusions are seen most commonly after filtering surgery, when the intraocular pressure has been purposely reduced. In that situation the absence of significant intraocular pressure prevents the expression of blood from the choroidal veins through the vortex veins into the extraocular venous bed. The choroidal veins then become distended with blood under pressure from the capillaries. That por-

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tion of the capillary circulation which normally drains into the collapsed low pressure veins must now flow into veins whose contents are under significantly higher tension. The resultant stasis, whether from failure of reabsorption or from abnormal transudation, may well explain the consequent choroidal effusion.

Choroidal effusions are also observed when the pressure in the ophthalmic vein is elevated, as occurs, for example, in Sturge-Weber disease or with dural shunts. If outflow resistance in the choroidal circulation were the result of pressure induced scleral stresses, these elevations of ophthalmic venous pressure would be ineffective in raising intraocular venous pressure, and for this reason: Stress of the sclera is a function of the difference between intraocular and extraocular pressures. Increasing the extraocular pressure is just as effective as lowering the intraocular pressure in reducing that stress. An increase in ophthalmic venous pressure results in an increase in the interstitial pressure of the orbital tissues, i.e., an increase in extraocular pressure. The increased extraocular pressure brings about a corresponding decrease in scleral stress, and hence a decrease in the resistance to flow within the sclera. If the choroidal venous pressure rises nonetheless, this is the case because the elevated ophthalmic venous pressure is transmitted directly to the vortex veins within the globe, and we may assume that it causes congestion, transudation and edema there much as occurs at the optic disc in the presence of elevated intracranial pressure. A disruption of the vortex vein, as at retinal surgery, causes stasis and effusion by a similar mechanism.

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Finally, if intraocular pressure is markedly elevated, as occurs at times after retinal surgery, where placement of an encircling band suddenly raises the tension, then, as predicted by Battaglioli, pressures may indeed rise high enough to compress the vortex vein in its scleral tunnel and thereby to obstruct venous drainage with ensuing choroidal detachment. It is also possible that scleral compression with secondary engorgement of the ciliary body and or choroidal effusion contributes to the anterior displacement of the lens-iris diaphragm in the angle closure glaucomas, especially those that occur in the presence of patent iridectomies and in the absence, therefore, of pupillary block.

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