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OUTFLOW

IV

A Model of Aqueous Outflow

Unlike the vessels that drain the retinal and the choroidal vascular beds which, at least to a degree, can be observed with the ophthalmoscope, the outflow channels for the aqueous humor are obscure. Consequently our understanding of aqueous drainage, such as it may be, is a synthesis of inferences: from the behavior of the eye in various pathological states, from gross and microscopic anatomy, and from theories of solid and fluid mechanics. Such inferences entail uncertainty, and any account that one gives of aqueous drainage is therefore unavoidably conjectural.

We may formulate our conjectures by constructing a model. A model has the advantage of permitting us, on the one hand, to be specific and affirmative in our descriptions, while, on the other hand, preserving the hypothetical quality of the construct. The model protects us, while we investigate the most obscure of topics, from the embarrassment of claiming knowledge which we in fact do not possess. The value of the model does not consist in its replicating reality. The model is valuable because it defines uncertainties and thereby provides a foundation for meaningful research. Indeed the imperfections of the model reflect those problems that would otherwise elude us. Paradoxically the most serious error into which any model might lead us is the belief that the model, persuasive from a particular perspective, could take the place of the reality that it purports at best only to mimic.

We begin with the obvious. Given the circumstance that the contents of the globe are under elevated pressure, and that there is flow of fluid from within the globe to the outside, from a region of elevated pressure to one of low pressure, it is axiomatic that there should be a mechanical resistance to this flow, some manner of reducing valve, without which the intraocular fluid, be it the blood of the retinal or choroidal circulations or the extravascular aqueous humor, would be entirely expressed from the eye, until the driving pressure collapsed to nothing.

In earlier issues of the Glaucoma Letter we considered how the pressures of the retinal and choroidal circulations are reduced. It is helpful to begin by looking for analogous mechanisms in the case of aqueous outflow. In each of the former circulations veins are distended by blood forced through a capillary network by the arterial pressure. As the vein approaches the low pressure extra-ocular circulation, it is compressed by the intra-ocular pressure and its cross-section is decreased, thereby increasing the resistance to flow and creating the pressure gradient that we have already described in some detail. It will be remembered that in the case of each of these circulations, the intravascular pressure is maintained by a segment of collapsed vein with increased resistance. The amount of the resistance is a function of the length and degree of venous collapse.

The Canal of Schlemm occupies a position in the aqueous circulation that is anatomically analogous to that of the efferent retinal and vortex veins in the retinal and choroidal circulations respectively. Each of these structures is subject to compression by the intraocular pressure, and each must remain to some degree distended, if it is to serve as a conduit for the drainage of the respective circulation. In the case of the retinal and choroidal circulation the forces that maintain the patency of the veins are readily identified: they are the pressures of the circulating blood transmitted through a capillary bed. In the case of the aqueous circulation, however, there is no capillary bed to deliver its flow to the lumen of Schlemm's canal, because aqueous is extravascular. The Canal of Schlemm is filled by pressure-driven transudation through its walls. The same pressure which fills the canal tends also to collapse and occlude it. It is inescapable that forces other than those exerted by the aqueous humor are required to secure the patency of the Canal of Schlemm.

any inherent in vivo rigidity of the trabecular meshwork. Almost surely, the stability of outflow resistance with varying intraocular pressure reflects the effect of other pressure-induced forces acting on the anterior chamber angle.

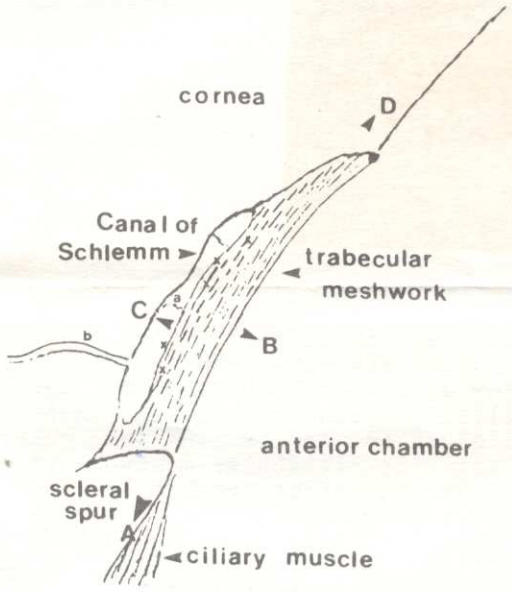

Figure 1. Model of the anterior chamber angle. The trabecular meshwork fills a sulcus in the sclera. When intraocular pressure rises, the sulcus gapes and the distance A<>D increases because the meshwork is more elastic than the sclera. Contraction of the ciliary muscle as from cholinergic stimulation and peripheral displacement of the choroid as from increased intraocular pressure also tend to increase A<>D. As A<>D increases, the meshwork is drawn toward B. Since the meshwork is anchored by the epithelial tubule (a), the result is a distention of the meshwork C<>B, with consequent decrease of its flow resistance. Aqueous flowing through the meshwork tends to displace the inner wall of Schlemm's canal outward toward C. The epithelial tubule (a) may prevent the collapse of the Canal of Schlemm. (b) is a collector channel. (x) is the location of the juxtacanalicular meshwork, a term used interchangeably with the inner wall of the Canal of Schlemm. Compression and expansion of the juxtacanalicular meshwork in the dimension C<>B is responsible for compensatory changes in outflow resistance.

Such forces may, in the first place, arise within the meshwork. We mentioned that an increase in the intraocular pressure, other factors remaining equal, would lead to an outward displacement of the meshwork with resultant compression of the Canal of Schlemm. However, since the inner wall of the meshwork is tethered to Schwalbe's line anteriorly and to the scleral spur in back, outward displacement of the inner wall of Schlemm's canal will increase the space between the trabecular plates and lower their resistance to outflow. This flow-induced distention of the meshwork constitutes a simple mechanism for maintaining a relatively constant intraocular ressure.

Other forces act on the meshwork through its external attachments. If, as seems likely from microscopic considerations, the modulus of elasticity of the meshwork is smaller than that of the adjacent sclera, then an increase in the intraocular pressure will, purely as a consequence of the local geometry, cause the trabecular sulcus to gape, disproportionately increasing the meridional stresses within the meshwork. As a result the meshwork will be drawn centripetally, widening the juxta-canalicular intratrabecular spaces, an effect that will be greatly enhanced if counter-traction is exerted by the epithelial tubules.

That mechanisms such as these are of more than theoretical interest is suggested by experimental work performed some years ago by Dr. David Campbell, then at the Massachusetts Eye and Ear Infirmary. Campbell showed that the placing of nonperforating sutures into the sclera in the vicinity of the limbus results in a significant increase in the resistance to aqueous outflow. The only effect of these sutures was to distort the sclera of the anterior chamber angle. Apparently therefore, maintenance of normal outflow facility requires not only an unobstructed meshwork, but requires also that the adjacent sclera remain undeformed. If distortion of the sclera in one direction increases resistance to outflow, it is conceivable that distortion of the sclera in the opposite direction might diminish that resistance, and there appears on the horizon the as yet unexplored possibility of ameliorating open angle glaucoma by nonpenetrating plastic surgery to the limbus.

Of particular interest in this context is neovascular glaucoma, a disease in which abnormal vessels proliferate onto the iris, into the anterior chamber angle, and onto the trabecular meshwork. The iris loses its resilience and becomes a rigid plate. What happens to the elastic properties of the meshwork is not known. It has always been assumed that the new vessels cause glaucoma by growing into and obstructing the pores of the trabecular meshwork. But this is probably not the

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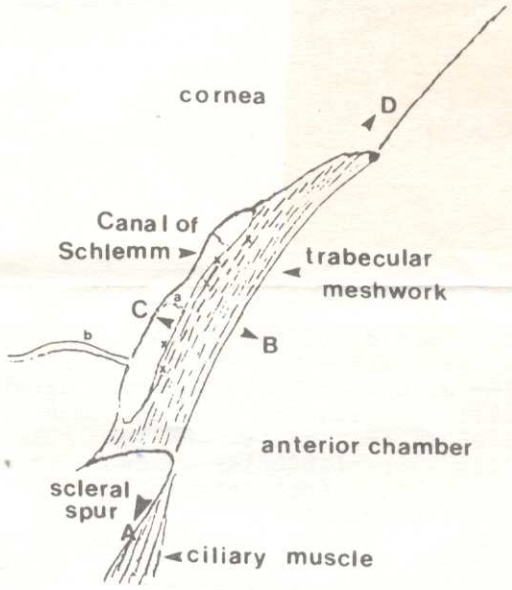

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case. More likely the impairment of outflow comes about when the new vessels stiffen the meshwork, thereby reducing those radial strains that are responsible for the spaces between the trabecular plates. Whether similar mechanisms obtain in congenital glaucoma and in the glaucoma of essential iris atrophy, remains to be considered.

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Contraction of the ciliary and iris musculatures are the most prominent intraocular effects of cholinergic agents. The ciliary muscle inserts into the scleral spur, and it is to the scleral spur that the trabecular meshwork is anchored. The fact that cholinergic drugs lower the intraocular pressure in open angle glaucoma strongly suggests that traction by the ciliary muscle may in some circumstances contribute to the distention of the meshwork. One assumes that the pull of the ciliary muscle on the scleral spur widens the trabecular sulcus and distends the trabecular meshwork in just the same way as does the pressure-induced widening of the scleral sulcus mentioned above. The fact that in the non-glaucomatous eye cycloplegia only rarely causes an increase in intra-ocular pressure, suggests that ciliary muscle tone does not affect the configuration of the healthy meshwork.

Posteriorly, the ciliary body fuses with the choroid. Therefore changes in the meridional tension exerted by the choroid must be expected to have an effect on outflow facility qualitatively if not quantitatively comparable to that of miotics. The meridional tension of the choroid is clearly a function of the intraocular pressure. It will also be affected by volume changes in the choriocapillaris and in the choroidal veins. Possibly vasoactive drugs such as epinephrine and timolol influence the intraocular pressure by altering the caliber of the choroidal vessels, the volume of the choroid, and hence the meridional tension the choroid exerts through the ciliary body on the scleral spur. The volume of the choroid in turn is a function of the intraocular pressure, as was explained in a recent issue of the Glaucoma Letter.

Ernst J. Meyer, M.D.