4 What Role Do Collector Channels Play in Determining Outcomes of Trabecular Bypass Surgery?

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Modern research into the pathogenesis of elevated intraocular pressure (IOP), which is characteristic of glaucoma, has focused on the deeper aspect of the trabecular meshwork and the endothelial lining of the inner wall of Schlemm's canal. Less attention has been paid to the collector channels and aqueous veins, as these vessels are not thought to be responsible for the altered hydrodynamics of the glaucomatous aqueous outflow pathway.[1,2] However, the flow resistance of these vessels may have an important role in determining the outcome of certain types of trabecular bypass surgery.

The bulk of aqueous outflow resistance is generated in the trabecular meshwork and inner wall of Schlemm's canal. It has been anticipated that procedures that bypass the trabecular meshwork (e.g., ab interno trabeculectomy and trabecular bypass procedures) would lower the IOP to levels not much higher than that of episcleral venous pressure; however, such procedures do not lower the IOP to the extent that was expected. This is likely due to the flow resistance of the collector channels and aqueous veins.

As aqueous humor flows out of Schlemm's canal, it enters into the collecting channels that connect the canal with the aqueous and episcleral veins, thus establishing venous return. The collector channels and aqueous veins have diameters that are many micrometers across, and the use of Poiseuille's law leads to the conclusion that these vessels should have negligible flow resistance.[3] However, experimental support for this conclusion is mixed. Mäepea and Bill[4,5] measured pressures in Schlemm's canal of primate eyes and found that the pressures were little different from the episcleral venous pressures, in agreement with Poiseuille's law. However, several other investigators have perfused enucleated primate and human eyes before and after a 360-degree trabeculotomy that would be expected to eliminate all flow resistance proximal to the collector channels and aqueous veins.[6–11] All of these studies have shown that at least 25% of outflow resistance remains after this procedure, in contrast to what would have been predicted theoretically. This has been demonstrated with sinusotomy as well.[12] It is possible that contractile cells surrounding these vessels[13] may locally constrict vessel size and increase their flow resistance.

The effect of this collector channel resistance is magnified when a partial trabeculotomy is done or a stent is inserted into Schlemm's canal. In such procedures, most aqueous flow bypasses the trabecular meshwork and enters into Schlemm's canal, or is exposed directly to the collector channel ostia themselves. The flow passing through those segments of Schlemm's canal nearest these openings is much higher than occurs normally. Furthermore, because there is substantial flow resistance in the collector channels, all of the flow cannot travel through just one or two collector channels, and thus flow may have to travel a significant extent through Schlemm's canal before exiting through the collector channels. These effects can generate a significant pressure drop in Schlemm's canal, in contrast to the normal physiological situation[14] in which the flow resistance of Schlemm's canal is negligible. This resistance is in addition to that generated by the collector channels, and thus it is not surprising that use of the trabectome or stents in Schlemm's canal leads to higher than expected postsurgical IOP.

Thus, when looking to lower aqueous outflow resistance by removing or bypassing the trabecular meshwork, it might be advisable to consider measures that also reduce the distal flow resistance in the collector channels and aqueous veins.

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