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Technical Note

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# Flow Resistance in Glaucoma, POAG, is Poorly Understood

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It has come to be widely recognized that the "conventional" drainage pathway of aqueous humor is slowed by resistance of fluid outflow caused in part by anatomical/mechanical properties of the trabecular meshwork (TM). But what is less widely recognized is that the major part of TM has cellular mechanisms for REDUCING resistance. The first layer of TM demonstrates large inter-cellular spaces between loosely woven connective tissue and endothelial cells, while the second layer of TM has "sheets" or "layers of tissue" composed of collagen, elastin, and protein-glucose complexes [1-3].

These 2 component layers of TM are characterized by presence of macrophage or phagocyte cells that actively "swallow" cellular debris, so as to subdue outflow resistance: A) By reducing aqueous humor viscosity; and B) By removing physical obstacles such as cellular fragments [4].

More than a few techniques for Laser ablation, of TM have been introduced, tested, and deployed: with or without concomitant surgery for cataract [5-7]. Because Laser ablation of tissue produces mechanical cleavage, it is widely posited that a TM that is obstructed by cellular debris would be "opened-up."

This point of reference seems reasonable, at first, to adequately justify blue-green Argon Laser procedures that damage TM. But on greater scrutiny TM is found to be metabolically active and exhibits self-regulation so, therefore, it may be good to innovate toward a mode of therapy that recognizes and respects such important and poorly understood attributes of TM [8-11]. Thanks to a research group at Bascom Palmer Eye Institute of the University of Miami, a newer technique: using near-infrared Laser was shown to selectively destroy dark pigmented cells that were lodged in the meshwork while sparing the meshwork itself [12].

## Dispersed Pigmented Cells Block Trabecular Outflow

Effects of dispersed pigmented on cells of TM have been characterized in a report from year 2019: Such as reduced macrophage activity, impaired cellular mobility, spasmodic contraction, and formation of fibrous material [13]. It appears likely, that during the acts of near focusing accommodation, pupillary dilation and constriction, and day-to-day movements, some dispersion of pigmented cells from the colored iris, and from ciliary processes, might happen naturally: by rubbing against the adjacent zonular fibers and crystalline lens, and from being "shaken off" by mechanical bodily jerks and vibrations [14,15].

Cases of pigment dispersion glaucoma could then merely be demonstrating "exaggerated dispersion" compared with "normal rates of dispersion," occurring in what is termed "primary" open-angle glaucoma. A novel infrared technique developed at the Pomeranian University in Poland, is reported as being able to quantify granular melanin pigment concentration in the living human eye [16]. The densely pigmented iris region adjacent to the pupillary aperture transmitted infrared nearly 15 times lesser than did peripheral (outer) regions of the iris. It remains to be seen whether this technique shall progress from experimental prototype to clinical instrumentation in the coming years [16].

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