Changes in IOP may alter ocular blood flow

Current research may indicate which glaucoma medications will best enhance axonal blood flow beyond the acute perfusion pressure effect.

by Elliot M. Kirstein, OD
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When intraocular pressure (IOP) is altered as the desired result of treatment or some change in ocular physiology, there may be a corresponding effect on ocular blood flow. The measurement and understanding of that dynamic may be a key element in glaucoma management.

The acute perfusion pressure effect (APPE) is the first and most dramatic change in the quality of the ocular pulse, which reflects change in IOP. With new technologies, such as Doppler flowmetry and ocular pneumoplethysmography (pulsatile ocular blood flow analysis), we may be able to observe and quantify this effect. Doppler technology enables us to study specific retinal vessels, while plethysmography yields a broader quotient, measuring the expansion and contraction of the globe caused by the entire choroidal pulse.

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The majority of the anterior portion of the optic nerve is nourished by the portion of the choroid that surrounds the disc (the circle of Zinn-Haller). The choroid is fed by the short and long posterior ciliary arteries, which, in turn, are fed by the ophthalmic and carotid arteries. Hayreh suggested that the peripapillary vascular supply is not evenly distributed around the optic disc (Hayreh SS. Optic nerve head blood supply in health and disease. In: Inlambrou GN, Greve EL, eds. Ocular Blood Flow in Glaucoma: Means, Methods, and Measurements. Amsterdam: Kugler and Ghedini; 1989. Hayreh SS. Interindividual variation in blood supply of the optic nerve head: its importance in various ischemic disorders of the nerve head and glaucoma, low-tension glaucoma, and allied disorders. Doc Ophthalmol. 1985;59:217.)

Watershed zones

The medial and temporal quadrants are, more often, well perfused, while the superior and inferior quadrants have less arterial influx and behave as watered zones. In these hypoperfused areas, optic nerve axons are more susceptible to ischemic insult.

Additionally, when IOP increases, there is an increased force against the systemic blood pressure and the perfu-
Sustaining the optic nerve axons: The majority of the nourishment of the anterior portion of optic nerve axons is derived from the short posterior ciliary arteries, which supply the circle of Zinn-Haller.

Ocular blood flow: Blood exits the internal carotid and enters the ophthalmic artery at 80 to 100 mm Hg and exits the globe through the venous outflow at 6 to 7 mm Hg. The choroidal blood flow represents 80% to 85% of the total ocular blood flow.

Autoregulation is the mechanism that modulates intraluminal pressure of the capillary bed amidst variations in systemic blood pressure. The goal of autoregulation is to accommodate the needs of ocular tissues in spite of the wide swings in the systemic pressure.

During sleep, when systemic pressure is typically low, the autoregulatory mechanism causes decreased microvascular resistance, therein facilitating necessary choroidal perfusion. Recent studies point to substances derived from the microvascular endothelium, which cause the relaxation and contraction of these vessels. The current understanding of autoregulation is that it is pre-eminent over autonomic influences and metabolic tissue demand. Researchers believe that autoregulatory defects are, in some way, responsible for some types of glaucomatous field loss.

As we consider the vascular component in glaucoma to be an indicator of visual field progression, we can develop a better understanding of glaucomatous individuals given information about their blood flow and how the APPE affects that status. Most often, when IOP increases, blood flow decreases. When IOP is somehow decreased, blood flow may improve.

Microcirculation

Although there is much debate about which drugs or circumstances are genuinely neuroprotective, it is difficult to argue the logic that supports the theory that adequate microcirculation is a requirement for the sustenance of optic nerve axons. A common denominator for all living animal tissue is that it requires a reasonable mechanism for transport of oxygen, nutrient and waste products. Further, it is likely that the cascade of axonal demise known as apoptosis is somehow linked to defects in microcirculation.

One of the obvious questions on our intellectual tables is: which medicines are best – or worst – for ocular blood flow? Which medicines elicit the greatest acute perfusion pressure effect? Ultimately, it would seem best to learn which medicine would fortuitously enhance axonal blood flow beyond the APPE. It seems likely that the wave of current research in that area will give glaucoma practitioners better direction.

My anecdotal findings are that the technique that most safely, effectively and appropriately reduces IOP is, for now, the most suitable choice for enhancing blood flow. We are still looking for acceptable scientific evidence that will yield more concise guidance for our therapeutic decisions.
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References


