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EDITORIAL

Impaired regulation of blood flow as a risk for inadequate blood flow to the optic nerve under challenge

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In this issue of *Eye*, Okuno and his colleagues present experimental results that add to the growing body of information that suggests the regulation of blood flow in patients with normal tension glaucoma is somehow different from those without glaucoma. Moreover, they seemed to have observed, even over just a few months, a greater tendency for some visual field deterioration in those with less tightly regulated velocity of blood flow in the optic nerve head. As both blood velocity and visual field measurements are subject to inter-test variation, even the small differences observed in this study merit notice when they are statistically significant.

The small differences, the small number of subjects, and the fact that velocity rather than flow was measured make it difficult to reach crisp conclusions from these data alone. However, they do fit with other data that suggest differences in vascular physiology in those who suffer from glaucomatous optic nerve damage. In concept, a person who has difficulty with local regulation[1] may have trouble maintaining nourishment of the optic nerve tissues when intraocular pressure rises, blood pressure falls, or other events challenge the blood flow in the optic nerve head, but in another person these challenges would be inconsequential because of efficient ability to regulate blood flow to meet local tissue needs.

Evidence of circulatory abnormalities, and particularly an erratic vascular regulation manifested by vasospasm and some aspects of migraine, has been found in some studies of glaucoma, particularly cases in which elevated intraocular pressure is not a dominant feature.[2] The inconstancy of finding these associations among studies of glaucoma patients[3] suggests that other important pathophysiologic events determine the impact of imperfect vascular competency and regulation on the optic nerve. Laboratory studies give suggestive evidence that the optic nerve can be impacted by vascular condition in combination with intraocular pressure.[4] Possibly, the numerous pericytes that surround optic disc capillaries help regulate flow[5] and can be caused to show reduced ability to regulate.[6] All together, there is enough suggestion of vascular participation in initiating the cascade of events that produce glaucomatous cupping[7,8] that we should make further efforts to understand the process of controlling the optic nerve head blood flow.

Lacking a complete understanding as yet, we are not really in a position to treat the vascular component of the pathogenic process of glaucomatous optic atrophy. For example, while a systemic vasodilator might seem a good idea, generalized vasodilation, rather than helping, might steal from the optic nerve and other tissues that are pathophysiologically unable to achieve adequate autoregulatory dilation. Therapy focused at increasing blood flow at the target location or restoring the ability to regulate might be needed. The present article by Okuno and colleagues reminds us that the focus of this line of therapy might best be directed at improving the ability to regulate blood flow adequately, rather than simply increasing blood flow.

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