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A theoretical model of the increase in venous oxygen saturation levels in advanced glaucoma patients

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Glaucoma is the second leading cause of blindness in the world and is characterized by progressive retinal ganglion cell death and irreversible visual field loss. Although elevated intraocular pressure has been identified as the primary risk factor for glaucoma and is the main target of glaucoma treatments, several vascular risk factors that lead to impaired retinal blood flow have also been correlated with the progression and incidence of glaucoma. In this study, a theoretical model of the retinal vasculature is applied to a set of oximetry data obtained from healthy individuals and glaucoma patients and is used to propose possible explanations for the clinically observed increases in venous blood oxygen saturation in advanced glaucoma patients. The model predicts that a decrease in retinal tissue oxygen demand, an impairment in blood flow autoregulation, or a decrease in Krogh cylinder tissue width can independently lead to increased venous saturation. Overall, the combined theoretical and clinical predictions suggest that the mechanisms leading to increased venous saturation differ between primary open angle glaucoma patients and normal tension glaucoma patients.