ABSTRACT PURPOSE: To test the hypothesis that blood flow autoregulation in the optic nerve head has less reserve to maintain normal blood flow in the face of blood pressure-induced ocular perfusion pressure decrease than a similar magnitude intraocular pressure-induced ocular perfusion pressure decrease.

MATERIALS AND METHODS: Twelve normal non-human primates were anesthetized by continuous intravenous infusion of pentobarbital. Optic nerve blood flow was monitored by laser speckle flowgraphy. In the first group of animals (n = 6), the experimental eye intraocular pressure was maintained at 10 mmHg using a saline reservoir connected to the anterior chamber. The blood pressure was gradually reduced by a slow injection of pentobarbital. In the second group (n = 6), the intraocular pressure was slowly increased from 10 mmHg to 50 mmHg by raising the reservoir. In both experimental groups, optic nerve head blood flow was measured continuously. The blood pressure and intraocular pressure were simultaneously recorded in all experiments.

RESULTS: The optic nerve head blood flow showed significant difference between the two groups (p = 0.021, repeat measures analysis of variance). It declined significantly more in the blood pressure group compared to the intraocular pressure group when the ocular perfusion pressure was reduced to 35 mmHg (p < 0.045) and below. There was also a significant interaction between blood flow changes and the ocular perfusion pressure treatment (p = 0.004, adjusted Greenhouse & Geisser univariate test), indicating the gradually enlarged blood flow difference between the two groups was due to the ocular perfusion pressure decrease.

CONCLUSIONS: The results show that optic nerve head blood flow is more susceptible to an ocular perfusion pressure decrease induced by lowering the blood pressure compared with that induced by increasing the intraocular pressure. This blood flow autoregulation capacity vulnerability to low blood pressure may provide experimental evidence related to the hemodynamic pathophysiology in glaucoma.


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