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Abstract Information

Abstract Title:

Time-course of change in optic nerve head circulation after acute reduction in intraocular pressure

Purpose:

To study the time course of changes in circulation in the optic nerve head (ONH) following acute reduction in intraocular pressure (IOP), and to evaluate the effects of a calcium antagonist, a nitric oxide synthetase (NOS) inhibitor, indomethacin, and sympathetic nerve amputation on the changes in ONH circulation following reduction of IOP.

Design:

Animal experiment

Participants:

Albino rabbits

Main Outcome Measures:

ONH blood flow (normalized blur value)

Methods:

In anesthetized rabbits, acute reduction of IOP (acute increase in ocular perfusion pressure (OPP)) was manometrically achieved and normalized blur value (NB), a quantitative index of tissue blood velocity obtained with the laser speckle method, was serially monitored for upto 60 min. The effects of systemic administration of 1 μ g/kg/hr nilvadipine (a calcium antagonist), 300 μ g/kg L-NAME, a nonselective NOS inhibitor), 5 mg/kg indomethacin, or sympathetic

nerve amputation on the changes in NB after reduction of IOP were studied.

Results:

During changes in IOP from 10 to 40, and then back to 10 mmHg, NB exhibited no significant change. In the nilvadipine-treated animals, during changes in IOP from 10 to 40 and back to 10 mmHg, NB decreased with increase in IOP to 40 mmHg and then increased to slightly above the baseline when IOP returned to 10 mmHg. L-NAME, indomethacin, and sympathetic nerve amputation each had little effect on the time course of change in NB.

Conclusion:

ONH circulation was stably maintained after acute reduction of IOP. The changes in NB following reduction of IOP occurred quickly and were partially impaired with a calcium antagonist, but not with a NOS inhibitor, indomethacin, or sympathetic nerve amputation. These findings suggest the importance of vascular smooth muscle in maintaining ONH circulation stable against reduction of IOP in a fashion nearly independent of nitric oxide, endogenous prostaglandins, and the sympathetic nervous system at least in rabbits.