

The Role of the Cerebrospinal Fluid Pressure in the Pathogenesis of Glaucomatous Optic Neuropathy: Implications for Medical Therapy

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Glaucomatous optic neuropathy shows characteristic morphologic differences when compared with non-glaucomatous (vascular) optic nerve damage: loss of neuroretinal rim versus no rim loss; deepening of the optic cup versus shallowing of the cup; and enlargement of beta zone of parapapillary atrophy versus no change in beta zone. "High-pressure glaucoma" and "normal-pressure glaucoma", however, can show strikingly similar appearances of the optic nerve head, quite in contrast to any vascular optic neuropathy. It may imply, that "high-pressure glaucoma" and "normal-pressure glaucoma" may have some aspects in their pathogenesis in common. Since the transcorneal pressure difference of "intraocular" pressure minus atmospheric pressure (so-called intraocular pressure as we measure it) is not the same as the trans-lamina cribrosa pressure difference ("IOP" minus orbital cerebrospinal fluid pressure), and since the trans-lamina cribrosa pressure difference is the pressure of real importance for the optic nerve head, one may postulate that in "normal-(eye-)pressure glaucoma" the orbital cerebrospinal fluid pressure is abnormally low. This has been postulated and reported in three previous experimental and clinical studies. The potential pathogenic and clinical consequences of these hypothesis and findings will be discussed.