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EFFECT OF SCLERAL ANCHORAGE ON THE PERFUSION OF THE LAMINA CRIBROSA

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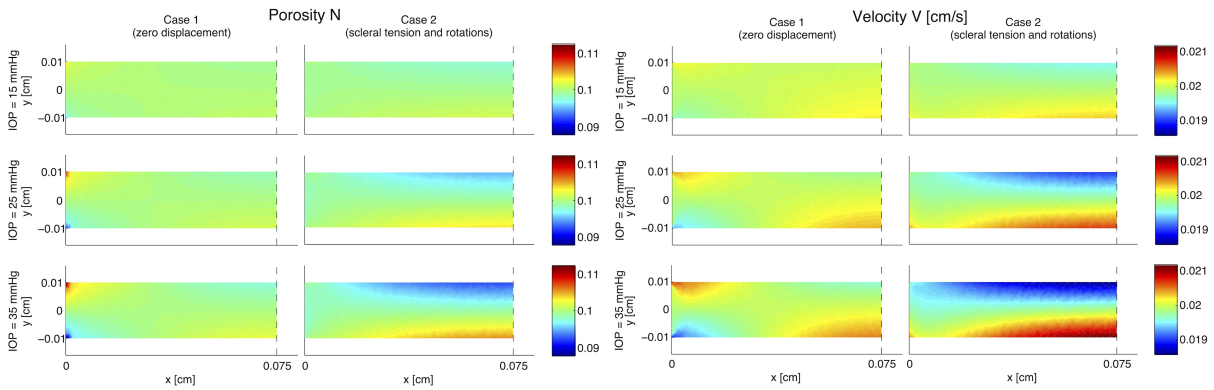
The biomechanics of the lamina cribrosa (LC) is believed to play an important role in retinal ganglion cell loss in glaucoma [1]. Studies have suggested that the development and progression of glaucoma are accompanied by significant structural changes in the LC [2], including changes in the scleral anchorage. In this study, we use a mathematical model to theoretically investigate the influence of scleral anchorage on the blood flow within the LC for various levels of intraocular pressure (IOP).

The LC is modeled as a two-dimensional poroelastic material, where blood vessels are viewed as pores in a solid matrix. LC porosity (N) (ratio between blood volume and LC total volume) changes with the local state of stress and strain; LC permeability is adapted proportionally to the square of N in view of Poiseuille's law; the solid matrix is assumed to behave as linear poroelastic material; the blood in the capillaries is incompressible and isothermal.

Blood flow is driven by the difference between the arterial pressure (Pa) in the short posterior ciliary arteries and the venous pressure (Pv) in the central retinal vein. The LC deforms under the difference between IOP and retrolaminar tissue pressure (RLTp). At the scleral junction ($x = 0$), scleral anchorage is modeled either by enforcing zero displacement (case 1) or by imposing a normal tension, calculated via Laplace's law, while allowing rotations (case 2). At the LC central axis ($x = 0.075$) displacement symmetry is imposed.



(a) (b)

Figure 1: Distributions of porosity N (left) and velocity V (right).

A numerical procedure is proposed to iteratively solve the nonlinear coupled system of partial differential equations describing the blood flow in a poroelastic material. N and blood velocity (V) are simulated and compared for IOP $= 15, 25$ and $35\,\text{mmHg}$. Control values are $\text{N} = 0.1, \text{V} = 0.02\,\text{cm}\,\text{s}^{-1}, \text{Pa} = 30\,\text{mmHg}, \text{Pv} = 20\,\text{mmHg}, \text{RLTp} = 7\,\text{mmHg}$. The model predicts that, for a given IOP level, values and distributions of N (see Figure 1a) and V (see Figure 1b) in cases 1 and 2 are noticeably different. As IOP increases from 15 to 35 mmHg, N undergoes a maximum percent reduction from baseline of 12.2% in case 1 and 7.7% in case 2. In case 1, the minimum values of N and V are attained at the scleral junction, whereas in case 2 they are attained at the LC central axis.

This exploratory two-dimensional analysis suggests that scleral anchorage has a strong influence on LC perfusion and might then contribute to LC remodeling and axonal damage in the retinal ganglion cells. It would be interesting to further extend the model to account for more realistic three-dimensional geometries and nonlinear constitutive equations for the solid matrix [3].

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