Editorial: Disc damage and raised IOP

Although the relationship between raised intraocular pressure and optic disc damage in primary open-angle glaucoma remains enigmatic, we naturally suspect that some sort of relationship does exist. Most attempts to establish the relationship have involved studies of the pressure in its relation to visual field decay or to visible deterioration of the disc by photography, but there has not been much success so far in finding histopathological evidence to link pressure with disc change. Indeed Miller's even went so far as to suggest there was not necessarily a causal link but that disc damage and raised pressure might simply be associated symptoms caused by a third factor. Miller's suggestion was a vascular abnormality which by affecting the outflow system caused aberrations in pressure or by affecting the disc caused typical glaucomatous damage. It would not be surprising if such a (hypothetical) abnormality were to affect both anterior and posterior segments of the eye simultaneously, giving rise to the familiar combination of raised pressure and disc damage in ordinary primary open-angle glaucoma.

There are great difficulties in dismissing raised pressure as a cause of disc damage, since in many cases of secondary glaucoma and indeed in primary angle closure it obviously is the cause of the damage. Nevertheless the notion is attractive.

But does the common factor have to be a vascular anomaly? How would it be if an abnormality were to be present in the collagen both of the trabecular meshwork and of the lamina cribrosa? Might not this give rise to weakness and consequent malfunction of the meshwork and at the same time weakness of the lamina cribrosa with obvious consequences.

The paper in this issue by Rehnberg, Ammitzboll, and Tengroth takes a small step forward in the process of establishing this possibility. In essence what the authors claim to have shown is that the characteristics of the collagen in the lamina cribrosa and trabecular meshwork are similar to one another but different from the sclera. Admittedly this study is on normal eyes only, but one awaits with great interest some evidence of possible variation from the normal in cases of glaucoma.

The search for a true step forward in chronic glaucoma has been a long and a weary one. The literature is full of painstaking efforts to relate the minutiae of visual field changes, whether computerised or not, to cleverly managed statistical manipulations of the intraocular pressure from the point of view of its mean, its peaks, its maximum, and its swings, not to mention a host of 'risk factors'—many of dubious relevance: but so far it has got us nowhere. It will certainly be a red letter day when someone can produce solid evidence to explain the peculiar discrepancies between pressure and field decay which we find so enigmatic at the moment.

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Reference