

The Production of Orderly Glaucomatous Field Defects

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The etiology of visual field defects occurring in glaucoma is controversial. However, one thing certain is that nerve fibers (NFs) are being destroyed in an orderly and a specific sequence. Before I discuss the field loss in glaucoma it is imperative to discuss the arrangement NFs in the retina and optic nerve head (ONH).

First, the one million or so nerve fibers in the retina are arranged in layers superficial to deep. Second, the most central vision fibers originate closest to the ONH, lie most superficial (closest to vitreous) and exit from the most central part of the ONH. In contrast, the most peripheral NFs originate from the most distant retina or farthest from the optic disc, lie deepest (closest to sclera) and exit closest to the edge of the scleral opening. Third, the NFs originating from the nasal retina proceed directly to the nasal part of the ONH. However, the situation is different in the temporal retina because of the presence of the macular fibers. The NFs originating from the nasal aspect of the macular area proceed directly to the temporal part of the disc. Lastly, the fibers originating from the temporal macular and temporal retina arch above and below the macular fibers to reach the superior and inferior poles of the ONH respectively. They are hence known as the arcuate fibers which thus become separated from the macular fibers. The arcuate fibers are also lying in layers superficial to deep as do the macular and remaining NFs.

In glaucoma, the peripheral fields are lost first, resulting in generalized peripheral field constriction. However, the early peripheral field loss has poor diagnostic value because of normal variation in the limits of the peripheral field. Moreover, other diseases like cataract can also cause peripheral visual field depression.

The most diagnostic glaucomatous field defect is the production of isolated scotomas in the 10 to 20 degrees of the paracentral region belonging to superior and inferior arcuate fibers. These isolated scotomas coalesce to form full superior and inferior arcuate scotomas which, combined together, is called a ring scotoma. While both arcuate scotomas are being produced, the peripheral field

loss is also progressing and approaching inwards and joins the arcuate/ring scotoma. The 5 to 10 degrees of the central vision is retained until the end-stage of glaucoma.

Therefore, it is well recognized that glaucomatous field defects are produced in an orderly and predictable sequence - never randomly. I believe this is the most important lead we have in discovering the pathogenesis of glaucoma. Were this not true and the glaucomatous field defects were being produced randomly, the role of perimetry in glaucoma would have been of no value. Glaucomatous field defects are well corroborated with the arrangement of NFs in the ONH.

The most puzzling question in glaucoma - why are the NFs being destroyed in an orderly sequence? The NFs in glaucoma cannot be destroyed in an orderly sequence either due the direct role of IOP or due to ischemia of the NFs. Therefore both can be ruled out as the direct cause of glaucoma. The prevalent glaucoma theories such as cupping, posterior bowing of lamina cribrosa (LC), neurodegeneration, apoptosis and others could never result in an orderly and predictable loss of NFs. In fact, there is no direct biological mechanism which could lead to orderly loss of NFs in glaucoma.

Nevertheless, it is an established fact that raised IOP is the definitive cause of glaucoma. So there must be some unique indirect way in which raised IOP is causing the orderly loss of NFs in glaucoma.

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I hypothesize that glaucoma is a two-stage disease. During the first, biological stage, there is degeneration of the border tissue of Elschnig (BT) which is solely supplied by the ciliary circulation. The systemic pressure supplying the BT and IOP are opposing forces. Normally, the perfusion pressure supplying the BT should be higher than IOP for the healthy maintenance of the BT. However, this delicate relationship will be reversed either by IOP becoming higher due to an ocular problem (HTG) or due to perfusion pressure of the BT becoming lower than IOP due to systemic problems (e.g. chronic hypotension). In the latter scenario, even normal range IOP (10 to 21 mmHg), if becoming higher than systemic pressure of BT, will act as high IOP for that subject and compress circulation of the BT and normal-tension glaucoma (NTG) will ensue. Chronic compression of the BT circulation will result in chronic ischemia and its degeneration. Therefore, it is the IOP becoming higher than the perfusion pressure of BT, resulting in both HTG and NTG.

Due to degeneration of the BT, the LC becomes loose and starts sinking in the scleral canal. This is the initiation of the second, mechanical stage resulting in stretching and severance of the most peripheral NFs first, being closest to the scleral edge. As a result, the next in line fiber will move towards the scleral edge and become severed. This severance of NFs leads to further sinking of LC due to loss of anchorage provided by NFs as roots anchor a tree. The cascade of severance of NFs and sinking of LC becomes self-propagated until all the NFs have moved in an orderly sequence to the scleral edge and become severed [1]. The vasculature is also being severed along with NFs resulting in splinter hemorrhages at the margin of ONH.

Posterior displacement or sinking of the LC is revealed by OCT in several published articles [2, 3]. Severance of NFs is revealed by notching at the poles in early stages of glaucoma. The arcuate-shaped retinal defects in intermediate stages and disc excavation devoid of nerve fibers in end-stage histology, support severance, not atrophy of the NFs. In summary, the NFs are not being atrophied but severed in glaucoma. Therefore based on orderly destruction of NFs, glaucoma may not be an optic neuropathy but an optic axotomy [4-6].

References

1. Hasnain SS (2006) Scleral edge, not Optic Disc or Retina, is the Primary Site of Injury in Chronic Glaucoma. Med Hypotheses 67:1320-1325.

2. Yang H, et al (2010) Optic Nerve Head Lamina Cribrosa Insertion Migration and Pialization in Early Non-Human Primate Experimental Glaucoma. Investigative Ophthalmology and Visual Science 51: 1631.

3. Yang H, et al. (2011) Posterior (outward) Migration of the Lamina Cribrosa and Early Cupping in Monkey Experimental Glaucoma. Investigative Ophthalmology and Visual Science 52: 7109-7121.

4. Zeried FM, Osuagwu UL (2013) Changes in Retinal Nerve Fiber Layer and Optic Disc Algorithms by Optical Coherence Tomography in glaucomatous Arab subjects. Clin Ophthalmol 7: 1941-1949.

5. Hasnain SS (2016) The Missing Piece in Glaucoma? Open Journal of Ophthalmology 6: 56-62.

6. Hasnain SS (2016) Pathogenesis of Orderly Loss of Nerve Fibers in Glaucoma. Optom open access.

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