

RELATION BETWEEN OPTICAL COHERENCE TOMOGRAPHY AND VISUAL FIELD CHANGES IN GLAUCOMA

Essay

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List of Abbreviation

RNFL	Retinal nerve fiber layer
SAP	Standard automated perimetry
GON	Glaucomatous optic neuropathy
OCT	Optical coherence tomography
VF	Visual Field
RGCs	Retinal ganglion cells
ONH	Optic nerve head
SWAP	Short – wave length automated perimetry
FDT	Frequency doubling technology

Introduction

Glaucoma is a leading cause of irreversible blindness throughout the world. World Health Organization statistics, published in 1995, indicate that glaucoma accounts for blindness in 5.1 million persons. (*Thylefors et al., 1995*).

In glaucoma, the essential pathologic process is the loss of retinal ganglion cells and their axons. In the clinical setting, glaucoma damage is commonly evaluated by funduscopy, photographs of optic disc and retinal nerve fiber layer (RNFL), and visual field testing. It has been shown that structural injury precedes visual field loss detectable by Standard Automated Perimetry (SAP) in many eyes with early glaucomatous optic neuropathy (GON) (*Tuulonen et al., 1993*).

Although SAP is the gold standard psychophysical test for glaucoma diagnosis, histopathologic studies showed that approximately 25% to 35% of retinal ganglion cell axons may be lost before visual field abnormalities are detected using conventional SAP (*Kerrigan-Baumrind et al., 2000*).

Abnormalities of the RNFL were present in 60% of eyes as much as 6 years before visual field damage was detectable (*Sommer et al., 1991*).

Optical coherence tomography (OCT) is high resolution, cross-sectional imaging technique that allows in vivo measurement of tissue thickness (*Hee et al., 1995*).

The stratus OCT is a third-generation machine that has a resolution of 8 to 10 μm and is capable of differentiating healthy eyes and eyes with glaucoma (*Wollstein et al., 2005*).

The stratus OCT with its internal normative data base showed high sensitivity and specificity for diagnosing glaucoma with manifest visual field (VF) defects .However , there are few reports regarding the use of OCT for detecting glaucomatous damage before detectable changes in SAP (*Donald Budenz et al. , 2005*).

SAP measuerements of visual sensitivity and OCT measurements of RNFL thickness are collerated measures of the underlying population of retinal ganglion cells (RGCs). Employing procedures to drive RGCs from corresponding visual field location and RNFL sectors produced agreement between these two methods of assessing retinal neurology, both of retinae with normal population of RGCs and for retinae with progressively decreased population of RGCs from the neuropathy of glaucoma. Thus, when measurements are translated to their common parameters of RGCs there is concordance between the structure and function of normal and defective vision from glaucoma(*Ronald Harwerth, et al., 2007*).

The aim of the work

This review aims to compare the changes which occur in visual field with those in optical coherence tomography in cases of glaucoma.

Anatomy of Optic nerve and Retinal nerve fiber layer

Terminology

The optic nerve head is defined as the distal portion of optic nerve which is directly susceptible to elevated IOP. The optic nerve head extends from the retinal surface to the myelinated portion of optic nerve that begins behind the sclera posterior to lamina cribrosa. The term optic nerve head is preferred over optic disc because the latter suggest a flat surface without depth. However, term disc and papilla are frequently used for portion of optic nerve that is visible by ophthalmoscopy. (*Jonas et al., 1999*).

General description

The optic nerve head is composed of the nerve fibers that originate in the ganglion cell layer of the retina and converge upon the nerve head from all points of the retina . At the surface of nerve head these axons bend acutely to exit the globe throw a fenestrated scleral canal called lamina cribrosa. Withen the nerve head the axons are grouped into about 1000 fascicles or bundles and are supported by astrocytes. There is considrable variation in the size of optic nerve head. In one study, the diamter varied from 1.18 to 1.75 mm (*Kronfeld 1976*).

Other studies have revealed ranges from 0.85 to 2.43 mm in the shortest diameter and 1.21 to 2.86 mm in the longest (*Jonas et al., 1988*)

or a mean of 1.88mm vertically and 1.77mm horizontally (*Quigley et al., 1990*).

Divisions of optic nerve head:

The nerve head can be divided into four portions from anterior to posterior (*Hayreh 1974*) (Fig. 1).

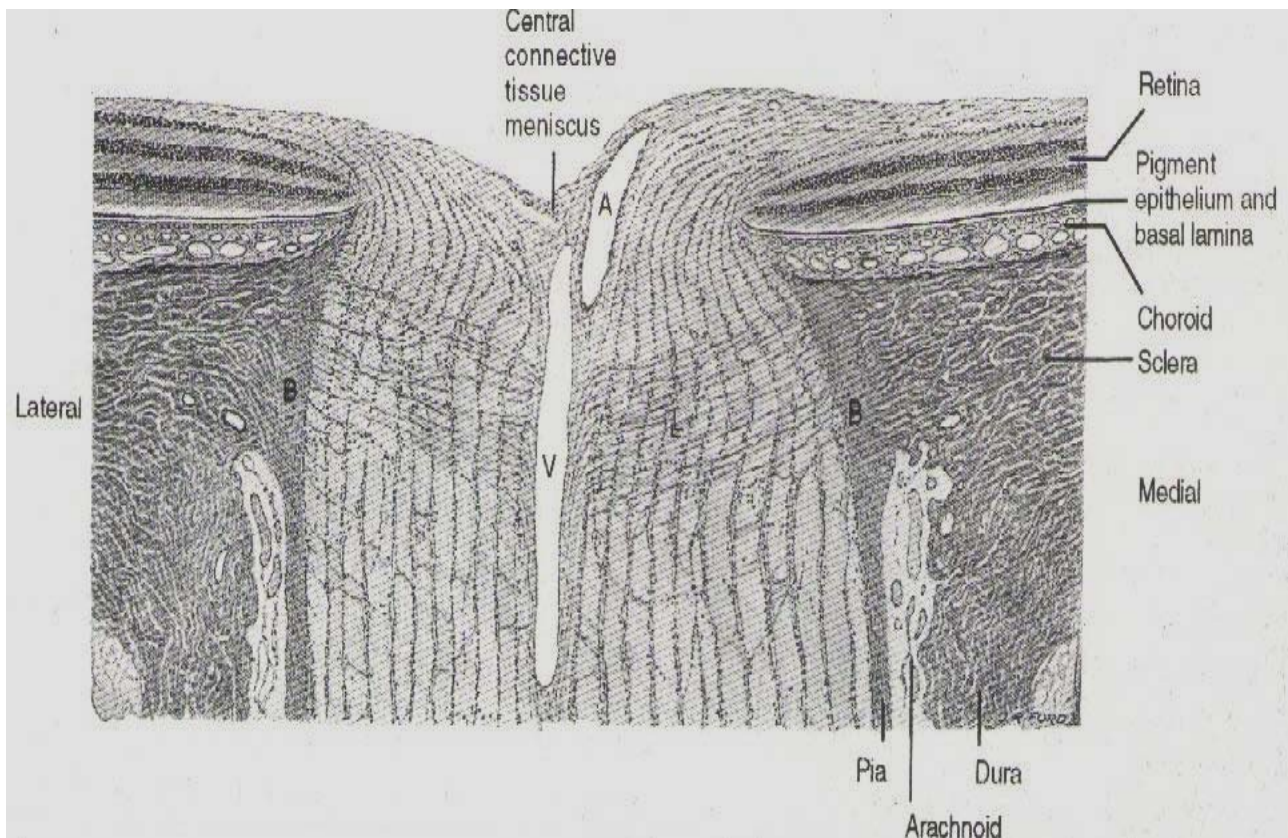


Fig. 1. Horizontal section of the optic nerve head. A= central retinal artery; V=central retinal vein; B=border tissue; L=lamina cribrose (*Bron, 1997*).

1-Surface nerve fiber layer

The innermost portion of the optic nerve head is composed mainly of nerve fibers. The axonal bundles acquire progressively more interaxonal glial tissue within the intraocular portion of the nerve head as this structure is followed posteriorly (*Minckler et al., 1976*).

2-Prelaminar region

The predominant structures at this level are nerve axons and astrocytes, with a significant increase in the quantity of astroglial tissue (*Anderson et al.,1969*).

3-Lamina cribrosa region

The portion contains fenestrated sheets of scleral connective tissue and occasional elastic fibers. Astrocytes separate the sheets and line the fenestrae, and the fascicles of neurons leave the eye through these openings (*Anderson et al.,1969*)

4-Retrolaminar region

This area is characterized by a decrease in astrocytes and the acquisition of myelin that is supplied by oligodendrocytes .The axonal bundles are surrounded by connective tissue septa. The posterior extent of the retrolaminar region is not clearly defined (*Hamasaki et al., 1967*).

Connective tissue support

Lamina Cribrosa

This structure is not simply a porous region of sclera but a specialized extracellular matrix that consists of fenestrated sheets of connective tissue and occasional elastic fibers lined by astrocytes (*Anderson 1969*)(*Fig.2*) . Astrocytes may respond to changes in IOP in glaucoma ,leading to axonal loss and retinal ganglion cell degeneration at the level of lamina cribrosa (*Hernandez, 2000*). The lamina cribrosa was