Saudi Journal of Ophthalmology (2013) 27, 97–106

Review Article

The role of cerebrospinal fluid pressure in glaucoma and other ophthalmic diseases: A review

David Fleischman, MD [a]; R. Rand Allingham, MD [b,*]

Abstract

Glaucoma is one of the most common causes of blindness in the world. Well-known risk factors include age, race, a positive family history and elevated intraocular pressures. A newly proposed risk factor is decreased cerebrospinal fluid pressure (CSFP). This concept is based on the notion that a pressure differential exists across the lamina cribrosa, which separates the intraocular space from the subarachnoid fluid space. In this construct, an increased translaminar pressure difference will occur with a relative increase in elevated intraocular pressure or a reduction in CSFP. This net change in pressure is proposed to act on the tissues within the optic nerve head, potentially contributing to glaucomatous optic neuropathy. Similarly, patients with ocular hypertension who have elevated CSFPs, would enjoy a relatively protective effect from glaucomatous damage. This review will focus on the current literature pertaining to the role of CSFP in glaucoma. Additionally, the authors examine the relationship between glaucoma and other known CSFP-related ophthalmic disorders.

Keywords: Cerebrospinal fluid pressure, Translaminar pressure, Glaucoma, Papilledema, Idiopathic intracranial hypertension, Microgravity

http://dx.doi.org/10.1016/j.sjopt.2013.03.002

Introduction

Glaucoma is a distinct optic neuropathy, which results in a characteristic nerve damage and a typical pattern of visual field loss. Intraocular pressure (IOP) is one of the most important risk factors. One approach to the classification of glaucoma is open versus closed angle forms. Closed-angle variants of glaucoma have elevated IOP that is caused by primary or secondary pathologies that share anatomic crowding or closure of the aqueous humor drainage system or angle. In open-angle forms of glaucoma the drainage angle is open, but for a variety of reasons the IOP may be elevated or remains in the normal range. Regardless of the type of glaucoma, intraocular pressure reduction by a medical or surgical approach has been shown to decrease incidence and progression of glaucoma.

Why glaucoma occurs with normal tension glaucoma (NTG) or does not occur in some patients with ocular hypertension (OHT) has been debated for decades. Explanations for NTG include hypotheses of a ''susceptible optic nerve,'' intrinsic retinal or ganglion cell pathology, inflammatory causes, undocumented elevations in IOP, and others. There is likely an interplay of susceptibility factors that decreases the threshold for glaucomatous injury in these patients despite seemingly normal intraocular pressures, or alternatively causes a different type of glaucoma. Vascular or perfusion abnormalities may be important, perhaps in concert with pressure fluctuations, neuronal excitotoxicity, genetic

Received 9 January 2013; accepted 11 March 2013; available online 17 March 2013.

[a] Department of Ophthalmology, University of North Carolina Hospitals, Chapel Hill, NC, USA
[b] Ophthalmology, Glaucoma Service, Duke University Eye Center, Durham, NC, USA

* Corresponding author. Address: Ophthalmology, Duke University Eye Center, 2351 Erwin Rd Box 3802, Durham, NC 27710, USA. Tel.: +1 919 684 0421.
e-mail address: rand.allingham@duke.edu (R.R. Allingham).

Peer review under responsibility of Saudi Ophthalmological Society, King Saud University

Production and hosting by Elsevier

Access this article online:
www.saudiophthaljournal.com
www.sciencedirect.com

mutations or predisposition, or other pressure-independent factors.[1–3] However, it has been determined that reducing IOP is beneficial in patients with normal tension glaucoma. Conversely, a variety of explanations have been offered for the observation that a relatively small percentage of patients with documented elevation of IOP out of the normal range ultimately develop glaucomatous damage. For these reasons it has become clear that factors in addition to IOP play an important role in glaucoma pathogenesis.

Recent studies have implicated the role of pressure of the primary fluid within the central nervous system, the cerebrospinal fluid (CSF), as a major contributor to glaucoma. The anatomical landmark of interest is the lamina cribrosa, a thin area of scleral tissue that separates two differentially pressurized compartments – the intraocular space and the orbital subarachnoid space. (Fig. 1) The difference in pressure between these two fluid spaces is termed the translaminar pressure. When described as a function of pressure across the

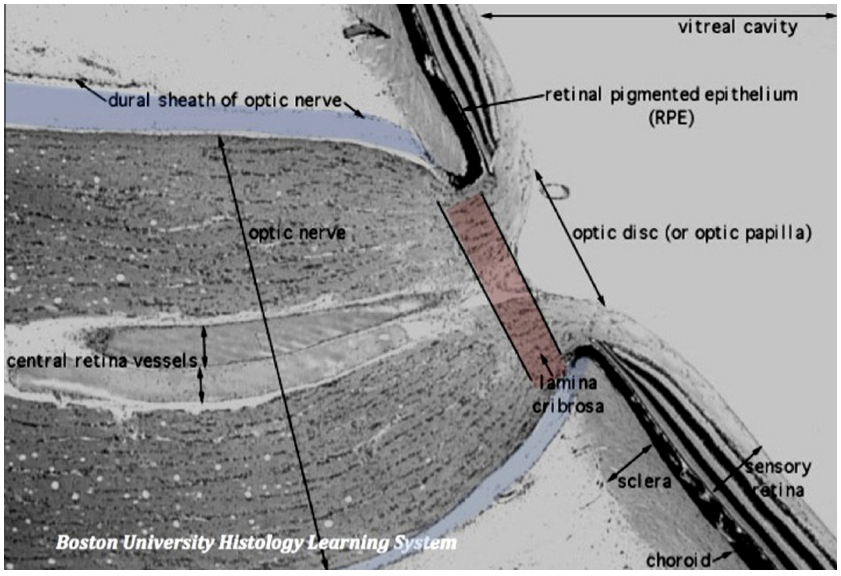

Figure 1. Histologic anatomy of the posterior globe and optic nerve with surrounding tissue. The lamina cribrosa (red) is a thin section of scleral tissue that acts as a sieve and allows the passage of exiting retinal ganglion cell axons and the retinal vessels. The cerebrospinal fluid (blue) flows within the subarachnoid space between the dura mater and the pia mater investing the optic nerve. Modified and reproduced with permission, Boston University Histology Learning System (Deborah W. Vaughan, PhD).

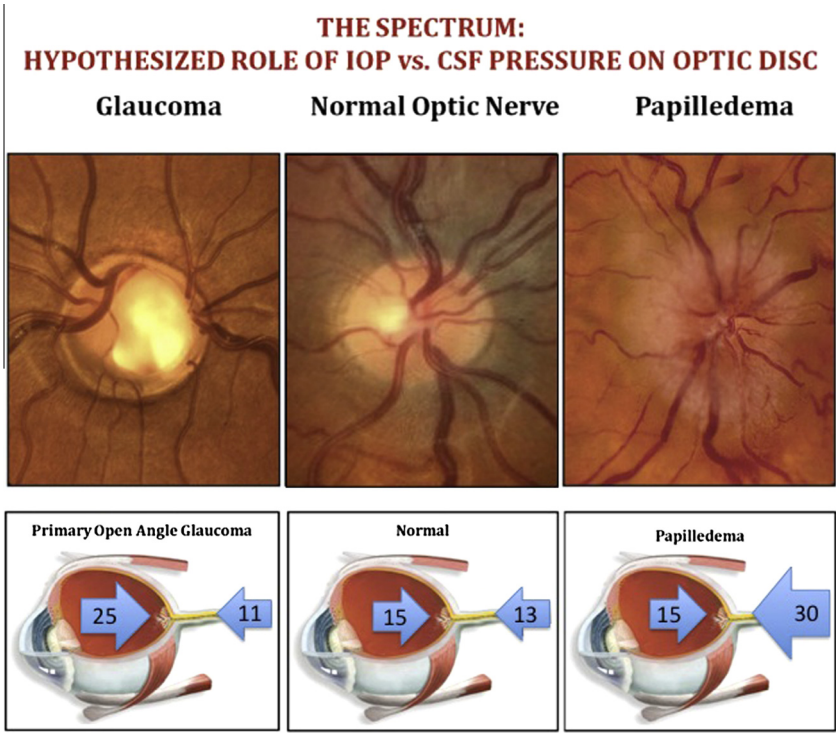

Figure 2. Spectrum of cerebrospinal fluid pressure-related ophthalmic disease.