Glaucoma Considered as an Imbalance Between Production and Clearance of Neurotoxins
Wostyn, Peter; De Groot, Veva; Van Dam, Debby; Audenaert, Kurt; Killer, Hanspeter Esriel; De Deyn, Peter Paul

Published in:
Investigative ophthalmology & visual science

DOI:
10.1167/iovs.14-15041

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2014

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):

Copyright
Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

Take-down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): http://www.rug.nl/research/portal. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Download date: 02-02-2020
Glaucoma Considered as an Imbalance Between Production and Clearance of Neurotoxins

We read with great interest the paper by Yang et al.1 titled “Optic Neuropathy Induced by Experimentally Reduced Cerebrospinal Fluid Pressure in Monkeys” published recently in Investigative Ophthalmology & Visual Science. We are grateful to the authors for sharing their findings with the scientific community, and we appreciate the opportunity to comment on an issue raised by the authors.

To examine the influence of experimentally reduced cerebrospinal fluid (CSF) pressure on retinal nerve fiber layer (RNFL) thickness and neuroretinal rim area of the optic nerve head, Yang et al.1 conducted a study on monkeys subjected to an implantation of a lumbar-peritoneal CSF shunt. In the study group (n = 4 monkeys), the shunt was opened to achieve a CSF pressure of approximately 40 mm H₂O, while the shunt remained closed in the control group (n = 5 monkeys). During a follow-up of 1 year, two out of the four monkeys of the active intervention group showed a progressive reduction in RNFL thickness in both of their eyes, accompanied by a significant reduction in the area and volume of the neuroretinal rim and a significant increase in the cup-to-disc area ratio. Two monkeys with artificially low CSF pressure did not develop the optic neuropathy observed in the other two monkeys, nor did any monkey of the control group. The authors emphasized that they did not examine any morphological changes in the lamina cribrosa of the monkeys, and therefore concluded that their study did not present any evidence that low CSF pressure caused glaucoma at normal levels of intraocular pressure (IOP). Their study, however, supported the concept that low CSF pressure alone may cause retinal ganglion cell injury and loss and thus that a low CSF pressure may be a risk factor in all forms of optic neuropathy including glaucoma.

The hypothesis of low CSF pressure, that is, intracranial pressure (ICP), as pathogenically important for glaucoma has attracted much attention in recent years. A growing body of evidence indicates that ICP is lower in patients with open-angle glaucoma and normal-tension glaucoma when compared with nonglaucomatous control subjects.2–4 These findings support the notion that the relationship between IOP and ICP is decreased with artificially low CSF pressure could be altered CSF dynamics related to shunt placement, that is, increased CSF flow from the ventricles, leading to enhanced removal of potentially neurotoxic waste products that accumulate in the optic nerve. Although the reduction in CSF pressure by the lumbar-peritoneal CSF shunt may increase the risk of developing optic neuropathy, shunt placement may also have a protective effect due to increased CSF turnover and clearance. We believe that this may explain why two monkeys with artificially low CSF pressure did not develop an optic neuropathy.

Peter Wostyn*1
Vera De Groot2
Debby Van Dam5
Kurt Audenaert1
Hanspeter Esriel Killer5
Peter Paul De Deyn5,6,7

*Department of Psychiatry, PC Sint-Amandus, Beernem, Belgium; 1Department of Ophthalmology, Antwerp University Hospital, Antwerp, Belgium; 2Laboratory of Neurochemistry and Behavior, Institute Born-Bunge, University of Antwerp, Department of Biomedical Sciences, Antwerp, Belgium; 3Department of Psychiatry, Ghent University Hospital, Ghent, Belgium; 4Department of Ophthalmology, Kantonsspitael Aarau, Aarau, Switzerland; 5Department of Neurology and Memory Clinic, Middelheim General Hospital (ZNA), Antwerp, Belgium; and the 6Department of Neurology and Alzheimer Research Center, University of Groningen and University Medical Center Groningen, Groningen, The Netherlands.

E-mail: wostyn.peter@skynet.be

Copyright 2014 The Association for Research in Vision and Ophthalmology, Inc.

www.iovs.org | ISSN: 1552-5785

5351

Downloaded From: http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/932996/ on 04/30/2018
References


Citation: *Invest Ophthalmol Vis Sci.* 2014;55:5351–5352. doi:10.1167/iovs.14-15041