Università degli Studi di Catania
Clinica Oculistica
Direttore: T.Avitabile

QUANDO INIZIARE IL TRATTAMENTO

M.G. Uva, M. Toro

OHTS

1636 soggetti ipertesi oculari

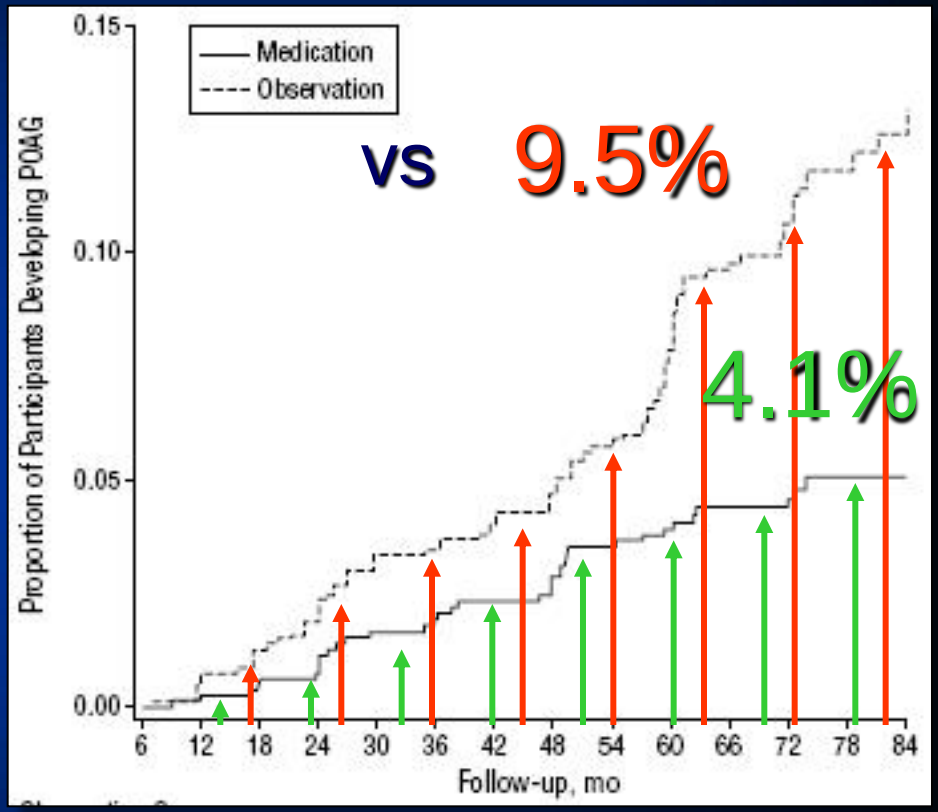
Eta' 40-80 anni

22 centri

IOP 24-32 mmHg

50% trattati / ↓ IOP 20%

50% osservati



CLINICAL SCIENCES

The Ocular Hypertension Treatment Study

ARCHIVES EXPRES

A Randomized Trial Determines That Topical Ocular Hypotensive Medication Delays or Prevents the Onset of Primary Open-Angle Glaucoma

Michael A. Kass, MD; Dale K. Heuer, MD; Eve J. Higginbotham, MD; Chris A. Johnson, PhD; John L. Keltner, MD; J. Philip Miller, AB; Richard K. Parrish II, MD; M. Roy Wilson, MD; Mae O. Gordon, PhD; for the Ocular Hypertension Treatment Study Group

Kass et al. *Arch Ophthalmol* 2002; 120: 701–713
Gordon et al. *Arch Ophthalmol* 2002; 120: 714–720

WHAT IS THE LIKELIHOOD A PATIENT WITH OH WILL BE BENEFITED IF TREATED?

The “number needed to treat” method suggests 20 patients need to be treated in order to benefit one patients

G.L. SPAETH

AMERICAN ACADEMY OF OPHTHALMOLOGY

SUB-SPECIALTY DAY, ANAHEIM, 2003

CLINICAL TRIALS

SECTION EDITOR: ANNE S. LINDBLAD, PhD

ARCHIVES EXPRESS

Delaying Treatment of Ocular Hypertension

The Ocular Hypertension Treatment Study

Michael A. Kass, MD; Mae O. Gordon, PhD; Feng Gao, PhD; Dale K. Heuer, MD; Eve J. Higginbotham, MD; Chris A. Johnson, PhD; John K. Keltner, MD; J. Philip Miller, BS; Richard K. Parrish, MD; M. Roy Wilson, MD; for the Ocular Hypertension Treatment Study Group

Objective: To compare the safety and efficacy of earlier vs later treatment in preventing primary open-angle glaucoma (POAG) in individuals with ocular hypertension.

Methods: One thousand six hundred thirty-six individuals with intraocular pressure (IOP) from 24 to 32 mm Hg in 1 eye and 21 to 32 mm Hg in the fellow eye were randomized to observation or to topical ocular hypotensive medication. Median time of treatment in the medication group was 13.0 years. After a median of 7.5 years without treatment, the observation group received medication for a median of 5.5 years. To determine if there is a penalty for delaying treatment, we compared the cumulative proportions of participants who developed POAG at a median follow-up of 13 years in the original observation group and in the original medication group.

Main Outcome Measures: Cumulative proportion of participants who developed POAG.

Results: The cumulative proportion of participants in the original observation group who developed POAG at 13 years was 0.22 (95% confidence interval [CI], 0.19-0.25), vs 0.16 (95% CI, 0.13-0.19) in the original medication group (P=.009). Among participants at the highest third of baseline risk of developing POAG, the cumulative proportion who developed POAG was 0.40 (95% CI, 0.33-0.46) in the original observation group and 0.28 (95% CI, 0.22-0.34) in the original medication group. There was little evidence of increased adverse events associated with medication.

Application to Clinical Practice: Absolute reduction was greatest among participants at the highest baseline risk of developing POAG. Individuals at high risk of developing POAG may benefit from more frequent examinations and early preventive treatment.

Trial Registration: clinicaltrials.gov Identifier: NCT00000125

Arch Ophthalmol. 2010;128(3):276-287

ARCHIVOS DE LA SOCIEDAD ESPAÑOLA DE OFTALMOLOGÍA

www.elsevier.es/oftalmologia

Editorial

OHTS 13 años después

OHTS 13 years later

F.J. Muñoz-Negrete[a], *G. Rebolleda*[a] y *D. Ruiz-Casas*[b]

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El OHTS *(Ocular Hypertension Treatment Study)* representó un hito en la comprensión y el manejo de los pacientes con hipertensión ocular (HTO). En la primera fase (1994-2002) se seleccionó a 1.636 hipertensos oculares a los que se aleatorizó a recibir tratamiento antiglaucomatoso o mantenerse en observación sin tratamiento. A los 5 años, un 4,4% de los pacientes en tratamiento y un 9,5% de los no tratados desarrollaron glaucoma, por lo que se concluyó que el tratamiento antiglaucomatoso tenía un efecto protector parcial en el desarrollo de glaucoma en HTO[1].

Una vez publicado que el tratamiento hipotensor ocular reducía y retrasaba el desarrollo del glaucoma, en 2002 se inició la segunda fase del OHTS, que prolongaba el estudio a 13 años y cuyos datos acaban de publicarse[2], lo que constituye una información valiosísima en el manejo y la información que podemos proporcionar a nuestros hipertensos oculares. En esta extensión longitudinal del estudio, a los pacientes del grupo control (mantenidos sin tratamiento antiglaucomatoso durante 7,5 años) se les ofreció la posibilidad de permanecer en el estudio comenzando con tratamiento antiglaucomatoso y continuar con éste durante 5,5 años. De esta forma, se podía evaluar si el hecho de retrasar el tratamiento antiglaucomatoso 7,5 años influía o no en la evolución de la enfermedad. Los criterios de diagnóstico de glaucoma fueron los mismos del estudio inicial, con una presión intraocular (PIO) objetivo tras tratamiento inferior a 24 mmHg y una reducción porcentual de al menos un 20%.

El porcentaje acumulado de pacientes que desarrollaron glaucoma de ángulo abierto fue del 22% en el grupo con tratamiento demorado y del 16% en el grupo con tratamiento temprano, lo que supone un 27% de reducción de riesgo de conversión a glaucoma en pacientes con tratamiento temprano. En este mismo sentido, el grupo de tratamiento demorado desarrolló glaucoma antes (media 6 años) respecto al de tratamiento temprano (media 8,7 años).

Aunque el tratamiento preventivo retrasa la aparición de glaucoma 2,7 años, globalmente el OHTS-2 no arroja una evidencia suficiente de que la demora en el tratamiento preventivo (7,5 años) en pacientes con HTO aumente la gravedad del glaucoma en los pacientes que lo desarrollan.

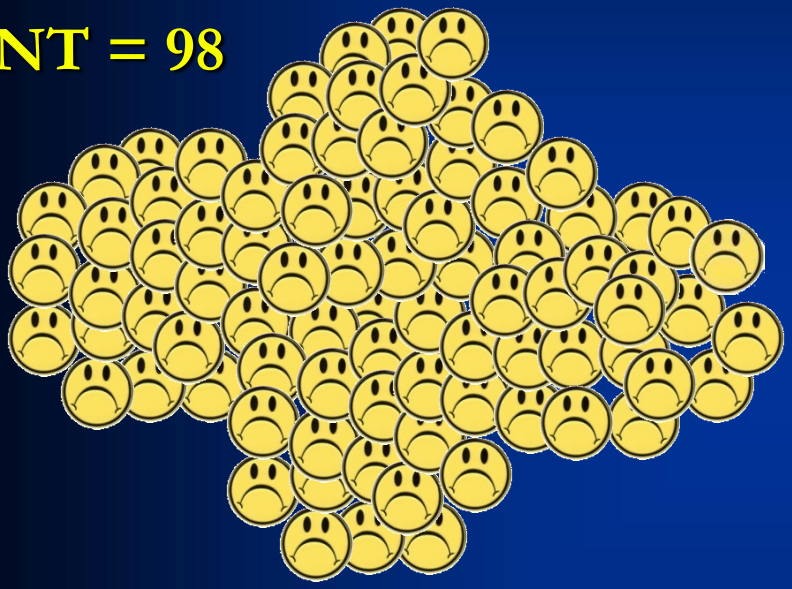
Quizás el dato más interesante de este estudio es la estratificación del riesgo de conversión a glaucoma entre pacientes hipertensos oculares. Se dividió a los pacientes en 3 grupos de riesgo, estimados con la calculadora de riesgo desarrollada del OHTS y el *European Glaucoma Prevention Study* (EGPS)[3], que estima la posibilidad de conversión a glaucoma en 5 años. Los 3 terciles de riesgo fueron: menor del 6% (riesgo bajo); 6-13% (riesgo medio), y mayor del 13% (riesgo alto).

En los pacientes de riesgo bajo inicial (menor del 6%), la proporción de pacientes que desarrolló glaucoma fue del 8% en el grupo de tratamiento demorado y del 7% en los de tratamiento temprano, por lo que el NNT *(number needed to treat)* era 98, lo que implicaba que sería necesario tratar a 98 individuos de forma inicial para evitar que uno desarrolle glaucoma tras 13 años de tratamiento. Fueron muy escasos los pacientes que desarrollaron glaucoma en ambos grupos en la primera fase, y no se observaron diferencias significativas entre ambos grupos. De estos datos se puede inferir que en este grupo de pacientes la simple observación parece ser el abordaje más coste/eficiente.

Sin embargo, en el grupo inicial de riesgo alto (mayor del 13%), al finalizar el estudio desarrollaron glaucoma el 28% de

WHAT IS THE LIKELIHOOD A PATIENT WITH OH WILL BE BENEFITED IF TREATED?

NNT = 98

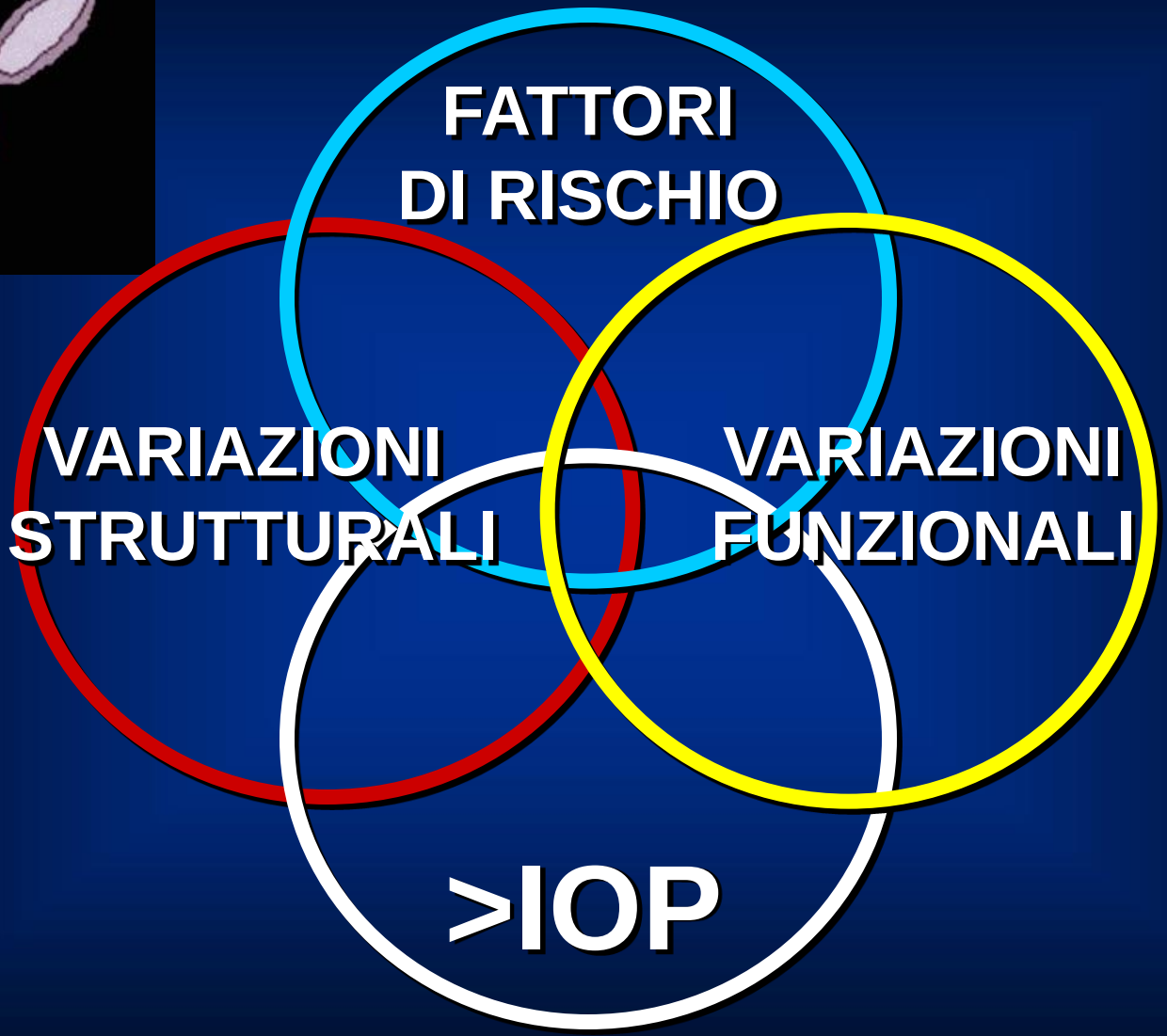


Low risk group

NNT = 7

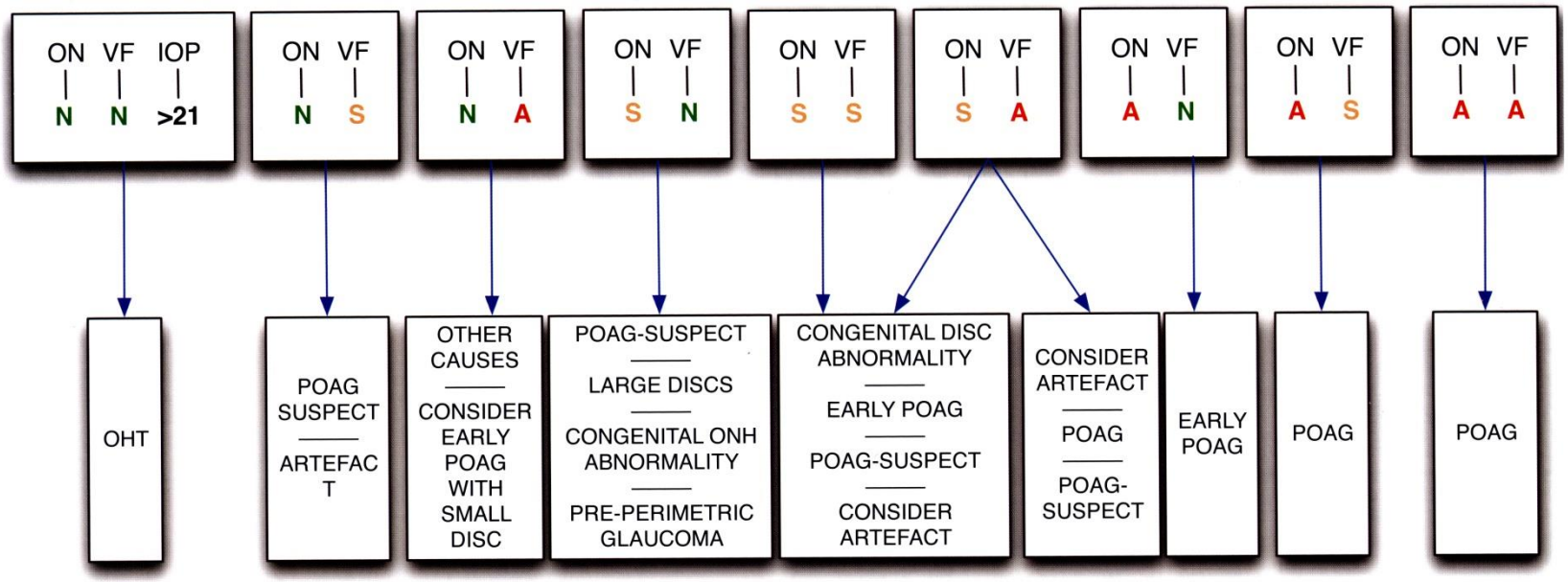
High risk group

F.J. Muñoz-Negrete, G. Rebolleda, D. Ruiz-Casas ARCH SOC ESP OFTALMOL. 2010


FATTORI
DI RISCHIO
VARIAZIONI
STRUTTURALI
VARIAZIONI
FUNZIONALI
>IOP

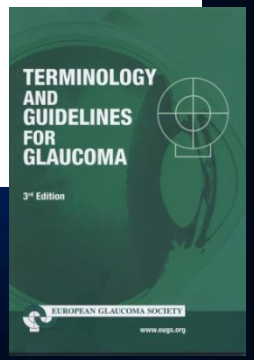
II. DIAGNOSTIC COMPONENTS

PRIMARY OPEN-ANGLE GLAUCOMA AND RELATED CONDITIONS



IOP = INTRAOCULAR PRESSURE
ON = OPTIC NERVE
VF = VISUAL FIELD
N = NORMAL
S = SUSPICIOUS
A = ABNORMAL

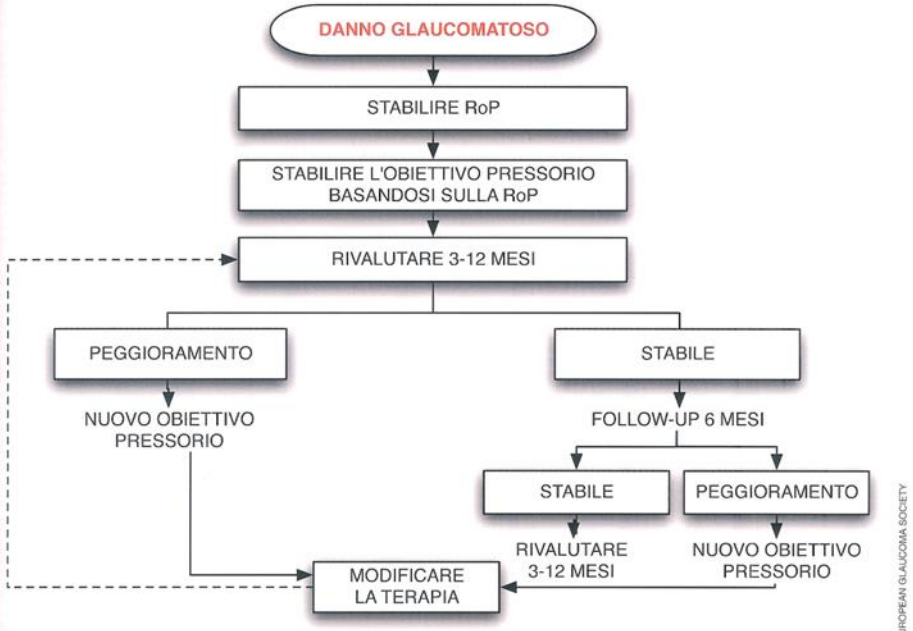
ABNORMAL FINDINGS SHOULD BE CONFIRMED BY REPEATED EXAMS



VALUTAZIONE E FOLLOW-UP

PAPILLA	IN CASO DI PIO ELEVATA CONSIDERARE LO SPESSORE CORNEALE CENTRALE	CAMPO VISIVO
	IN CASO DI VALORI TONOMETRICI MOLTO ALTI O COMPRESI TRA 25-29 mmHg E CONFERMATI, RIPETERE LE MISURAZIONI ENTRO 1-12 MESI IN RELAZIONE AI VALORI DI PIO	
NORMALE		NORMALE
NORMALE	ARTEFATTI O ALTRE CAUSE	PATOLOGICO
NORMALE	RIPETERE ESAME — INFORMARE	SOSPETTO
PATOLOGICA		NORMALE
SOSPETTA	RIPETERE ESAME — INFORMARE	NORMALE
SOSPETTA		SOSPETTO
SOSPETTA	GLAUCOMA INIZIALE/SOSPETTO RIPETERE ESAMI 3-12 MESI	PATOLOGICO
PATOLOGICA		SOSPETTO
PATOLOGICA	DANNO GLAUCOMATOSO	PATOLOGICO

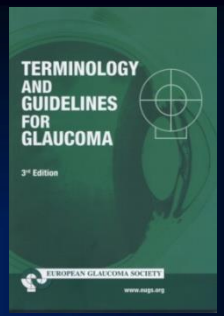
DIAGRAMMI



GLI INTERVALLI PER IL FOLLOW-UP SONO SOLO RACCOMANDAZIONI

Ipertensione Oculare

(Definizione – ***EGS)***



IOP	>21 mmHg costante
Gonioscopia	A.I.C. Aperto
Papilla e RNFL	Normali
Campo visivo	Normale
Familiarita’	Negativa
Fattori di rischio	Nessuno

Predictive Factors for Open-Angle Gl… Hy… Pr…

Ophthalmology *Volume 114, Number 1, January 2007*

Table 3. Univariate Hazard Ratios and 95% Confidence Intervals (CIs) for the Development of Open-Angle Glaucoma

Predictors	Hazard Ratio	95% CI	*P* Value
Age (per decade)	1.42	1.17–1.71	<0.001
Gender (male)	0.85	0.59–1.23	0.39
Diabetes	0.89	0.36–2.17	0.79
Cardiovascular diseases	1.84	1.16–2.9	0.01
High blood pressure	1.22	0.83–1.78	0.30
Systemic antihypertensive drugs (any)	1.46	0.99–2.15	0.053
Systemic β-blockers	1.35	0.73–2.51	0.34
Systemic calcium-channel blockers	1.27	0.66–2.42	0.47
Systemic ACE inhibitors	1.12	0.66–1.9	0.66
Systemic diuretics	1.36	0.79–2.41	0.26
IOP, baseline (per mmHg)	1.17	1.06–1.29	0.002
CCT (40 μm thinner)	1.50	1.22–1.83	<0.001
Myopia (≥−1-D spherical equivalent)	0.80	0.49–1.30	0.36
Vertical C/D ratio (per 0.1 larger)	1.32	1.17–1.50	<0.001
Vertical C/D ratio asymmetry (per 0.1 larger)	1.56	1.21–2.00	<0.001
Mean deviation	1.08	0.95–1.23	0.22
Pattern standard deviation (per 0.2 dB greater)	1.44	1.08–1.93	0.014
Corrected pattern standard deviation (per 0.2 dB greater)	1.06	0.79–1.43	0.70
Pseudoexfoliation syndrome	2.57	1.05–6.30	0.039
Pigmentary dispersion syndrome	1.06	0.26–4.29	0.93

ACE = angiotensin-converting enzyme; CCT = central corneal thickness; C/D = cup-to-disc; dB = decibels; IOP = intraocular pressure.

Europe…

Obj… …ocular hypert…

Des…

Par… …enters. The pa… …s (VFs) (on the… …isc, as determ…

Inte… …eyes.

Mai… …graphic and cli… …h were determ… …which particip…

Res… …hazard ratio [H… …4; 95% CI, 1.1… …5% CI, 1.15–2…

Con… …dictors of the … …of the develop… …dy and support the need for a thorough evaluation of patients with ocular hypertension. *Ophthalmology 2007;114:3–9*

Validated Prediction Model for the Development of Primary Open-Angle Glaucoma in Individuals with Ocular Hypertension

Table 6. A Point System for Estimating an Ocular Hypertensive Patient's 5-Year Risk of Developing Primary Open-Angle Glaucoma (POAG)

Baseline Predictor	Points for Baseline Predictor				
	0	*1*	*2*	*3*	*4*
Age (yrs)	<45	45 to <55	55 to <65	65 to <75	≥75
Mean IOP (mmHg)*	<22	22 to <24	24 to <26	26 to <28	≥28
Mean CCT (μm)*	≥600	576–600	551–575	526–550	≤525
Mean vertical cup-to-disc ratio by contour*	<0.3	0.3 to <0.4	0.4 to <0.5	0.5 to <0.6	≥0.6
Mean PSD (dB)*	<1.8	1.8 to <2.0	2.0 to <2.4	2.4 to <2.8	≥2.8
Sum of points	0–6	7–8	9–10	11–12	>12
Estimated 5-yr risk of POAG	≤4.0%	10%	15%	20%	≥33%

CCT = central corneal thickness; dB = decibels; IOP = intraocular pressure; PSD = pattern standard deviation.
*Eye-specific variables are the mean of right and left eyes.

observation group and the EGPS placebo group—baseline age, intraocular pressure, central corneal thickness, vertical cup-to-disc ratio, and Humphrey VF pattern standard deviation. The pooled multivariate model for the development of POAG had good discrimination (*c* statistic, 0.74) and accurate estimation of POAG risk (calibration χ^2, 7.05).

Conclusions: The OHTS prediction model was validated in the EGPS placebo group. A calculator to estimate the 5-year risk of developing POAG, based on the pooled OHTS–EGPS predictive model, has high precision and will be useful for clinicians and patients in deciding the frequency of tests and examinations during follow-up and advisability of initiating preventive treatment. *Ophthalmology 2007;114:10–19 © 2007 by the American Academy of Ophthalmology.*

TABLE 5

Predictive Factors (Hazard Ratios with 95% Confidence Intervals) for Progression of Open-angle Glaucoma in Randomized Clinical Trials on Open-angle Glaucoma Populations

	CNTGS (5 yrs follow-up)	AGIS (6 yrs follow-up)	EMGT (11 yrs follow-up)	CIGTS (4 yrs follow-up)
Baseline predictive factors				
Age (per older year)	-	-	-	1.04 (1.02–1.05)
Age (per 5 years)	-	1.28 (1.10–1.49)	-	-
Age ≥68 yrs	-	-	1.51 (1.11–2.07)	-
Ancestry	n.s. HR not reported	n.s. – multivariate 1.07 (0.63–1.81)	-	1.50 (1.08–2.07)
Sex male	n.s. – multivariate 0.54 (0.28–1.04)	2.23 (1.54–3.23) ATT	n.s. – HR not reported	n.s. – HR not reported
OAG family history	n.s. HR not reported	-	n.s.–HR not reported	n.s. – HR not reported
N° of eligible eyes (2)	-	-	1.88 (1.35–2.63)	-
Myopia	-	n.s. – multivariate 0.75 (0.43–1.31)	-	-
IOP (per mm Hg)	n.s. HR not reported	n.s. – multivariate 0.96 (0.92–1.003)	-	-
IOP ≥ 21	-	-	1.77 (1.29–2.43)	-
CCT (per 40 μm thinner)	-	-	1.25 (1.01–1.55)	-
SBP (≤125 mm Hg)	-	-	1.42 (1.04–1.94)	-
High BP	n.s. HR not reported	n.s. – multivariate 1.49 (0.91–2.43)	-	-
Diabetes	n.s. HR not reported	1.87 (1.18–2.97) TAT	n.s. – HR not reported	1.59 (1.07–2.38)
Migraine	2.58 (1.32–5.07)	-	-	-
Disc hemorrhages	2.72 (1.39–5.32)	-	n.s. – HR not reported	-
c/d ratio ≥ 0.7	n.s. HR not reported	-	n.s. – HR not reported	-
MD ≤ −0.4 dB	-	-	1.38 (1.00–1.91)	-
Higher AGIS VF defect score	-	0.86 (0.82–0.90) ATT 0.93 (0.89–0.97) TAT	-	-
Higher VA defect score	-	0.96 (0.94–0.98) ATT	-	-
PEX	-	-	2.12 (1.30–3.46)	-
Post-baseline predictive factors				
Mean IOP during follow-up (per mm Hg)	-	n.s. – multivariate 1.07 (0.97–1.18)	1.11 (1.06–1.17)	-
IOP fluctuation (SD of mean IOP)	-	1.31 (1.11–1.53)	n.s. – multivariate 1.00 (0.81–1.24)	-
Initial change in IOP (Baseline −3-mo IOP)	-	-	0.92 (0.89–0.96)	-
IOP at first follow-up visit (3-mo IOP)	-	-	1.13 (1.08–1.18)	-
Number of medications during follow-up	-	n.s. – multivariate 1.19 (0.85–1.66)	-	-
Treatment	p = 0.21° p = 0.018°°	n.s. – multivariate 1.46 (0.90–2.38)§	0.53 (0.39–0.72)	n.s. – multivariate 1.36 (0.99–1.85)**
Length of follow-up	-	1.18 (1.02–1.37)	-	1.01 (1.01–1.02)
No. of glaucoma interventions	-	1.73 (1.14–2.63)	-	-
Disk hemorrhages	-	-	1.02 (1.01–1.02)*	-
Cataract surgery	-	n.s. – multivariate 1.18 (1.02–1.37)	-	-

CNTGS = Collaborative Normal Tension Glaucoma Study; AGIS = Advanced Glaucoma Intervention Study; EMGT = Early Manifest Glaucoma Trial; CIGTS = Collaborative Initial Glaucoma Treatment Study; OAG = open-angle glaucoma; IOP = intraocular pressure; CCT = central cornea thickness; MPP = mean ocular perfusion pressure; SBP = systolic blood pressure; MD = mean deviation; VF = visual field; VA = visual acuity; PEX = pseudoesfoliation syndrome.

°No significant protective effect of treatment vs non-treatment.

°°Significant protective effect of treatment vs non-treatment when data of eyes developing cataracs were censored at the time of the diagnosis of cataract.

§Comparison of Trabeculectomy-ALT-Trabeculectomy vs ALT- Trabeculectomy- Trabeculectomy sequence.

*Percent of visits with disc hemorrhages (time dependent) – (per % higher).

**Treatment odds ratio is for surgery vs medicine.

Eta' avanzata	PSD alta
Familiarita'	c/d elevato
Cornea sottile	Atrofia β (?)
>IOP	PEX

. . . .

Giovane	PSD bassa
No familiarita'	c/d basso
Cornea spessa	No atrofia β
No PEX	

Il continuum del glaucoma:

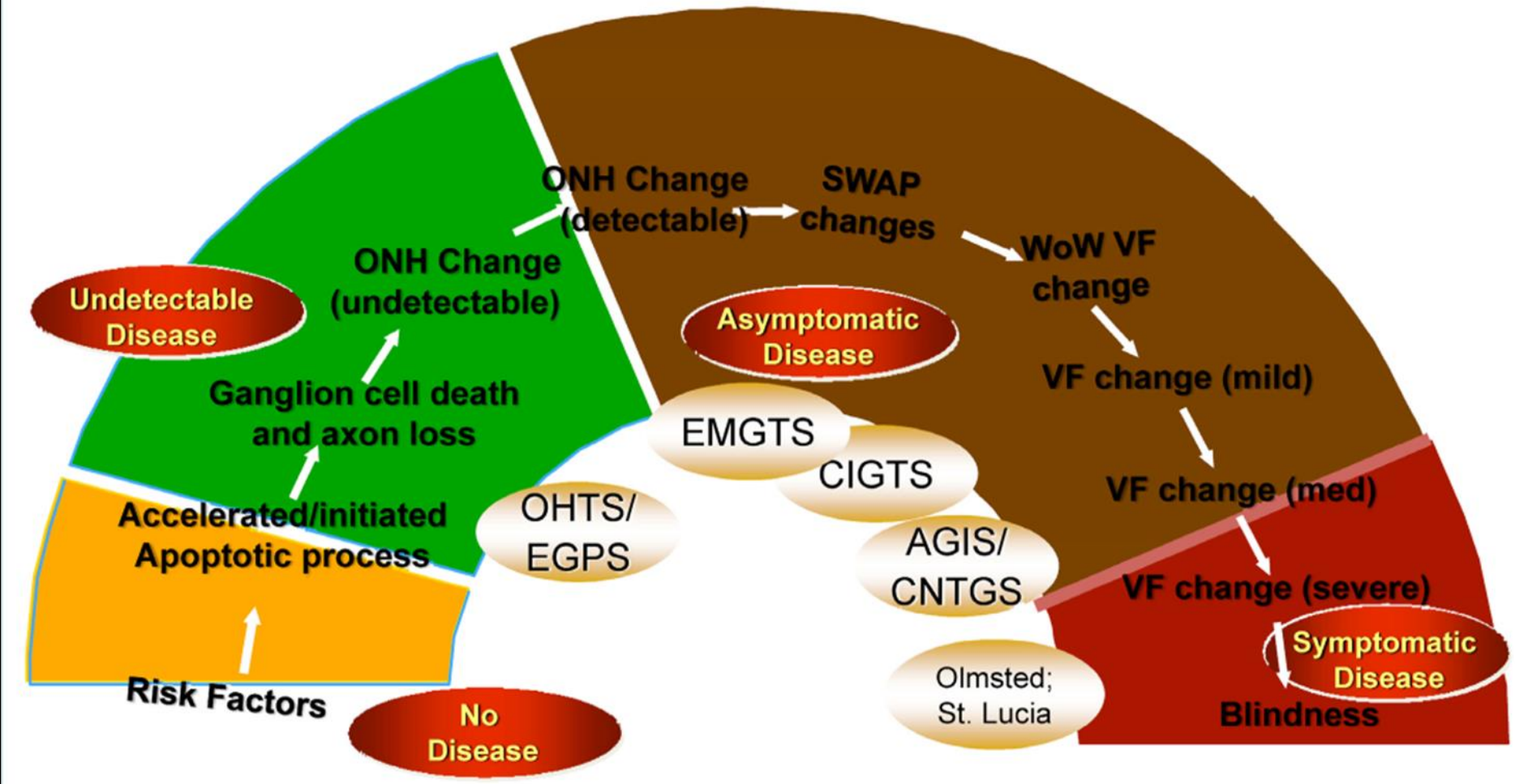
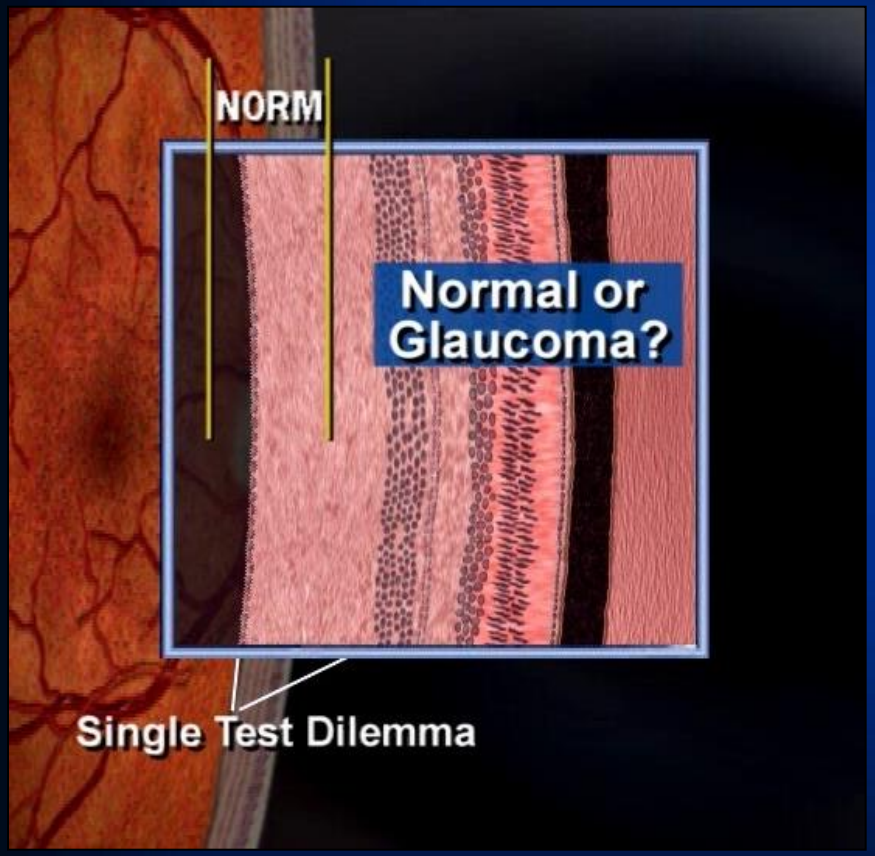


Fig. (3). The Glaucoma Continuum showing disease states from "no disease" to "symptomatic disease".

Weinreb RN, et al. AJO. 2004.

Confronto con un „database“ normativo



Un soggetto, compreso nel range di normalità statistica di un determinato parametro, può subire importanti variazioni del disco ottico e dello strato delle fibre nel corso del tempo pur rimanendo all‘interno dell‘intervallo di normalità di qualsiasi esame eseguito una volta sola.

Per gentile concessione di Enrico Martini

Database Normativi

- Le valutazioni fatte su un singolo esame non sono affidabili.
- La ampia sovrapposizione del range di normalità e di valori patologici diminuisce la capacità diagnostica.
- I Database Normativi sono solo indicatori di „probabilità“ e non sono sufficientemente specifici per la diagnosi definitiva.

Per gentile concessione di Enrico Martini

Special Issue

Detection and Diagnosis of Glaucoma: Ocular Imaging

Joel S. Schuman

Imaging is a valuable tool in the assessment of glaucoma and glaucoma progression. Although glaucoma is a clinical diagnosis—there is no blood test or definitive genetic test, for

Automated objective, quantitative characterization of the optic nerve has been more successful. As mentioned above, the first step in this direction was optic nerve photography,

Imaging Basics

It is important to have realistic expectations for what ocular imaging in glaucoma can provide, as disappointment occurs when expectations exceed reality. CSLO, SLP, and OCT cannot diagnose glaucoma, nor can they diagnose glaucomatous progression. They can be used, however, to create the foundation on which to build these diagnoses. These technologies produce objective, quantitative, accurate, and precise measurements of ONH features, the retinal nerve fiber layer (RNFL), and macular substructure.

been validated to the level of perimetry, which remains the clinical gold standard, despite its shortcomings.

there was no normative database with which to compare the patient at hand. The devices can each statistically determine whether the structural features of the ONH and RNFL of a given individual fall within or outside the normal range and whether statistically significant change has occurred over time.

Technology Primer

CSLO takes a series of images of coronal planes that vary in tissue depth. The images are then combined and the surface topography of the tissue mapped. Depth information is present, but axial resolution is limited to approximately 300 μm. The ability to discriminate between glaucomatous and healthy eyes is good,[20] and the algorithm for detection of change over time has been validated in clinical studies. More change events are seen using CSLO than perimetry.[15]

SLP uses polarized light shone into the eye and reflected by structures in the eye back to a detector to determine the

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Disclosure: **J.S. Schuman**, Slack, Inc. (C), Vindico, Inc. (C), P

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DOI:10.1167/iovs.12-9483k
Investigative Ophthalmology & Visual Science, Special Issue 2012, Vol. 53, No. 5

The Venn Diagram Problem

A great problem in the evaluation of glaucoma progression is the lack of a gold standard. It is not clear which technology best defines glaucoma progression. Given a cohort of subjects, different technologies will define different eyes as progressing. Not only will structure and function be identified as occurring in different eyes, but even structure measured with different devices will show progression in different eyes in the same cohort.[29] This lack of overlap among eyes showing change is commonly referred to as the Venn diagram problem.

The major advantage of ocular imaging in glaucoma is that it is reproducible and provides an accurate, objective, quantitative assessment of the status of the ocular structure. This technology is useful for the diagnosis of glaucoma and the detection of progression and for identifying eyes at high risk of conversion to or progression of glaucoma. Ocular imaging reduces uncertainty and repeated perimetric testing, especially in cases of structure–function correspondence; however, progression identified by one technology is often not mirrored by others. This area requires additional investigation to determine the root of the Venn diagram problem.

Clinical & Experimental Ophthalmology

Clinical and Experimental Ophthalmology 2012; 40: 369–380 doi: 10.1111/j.1442-9071.2012.02770.x

Review

'Structure–function relationship' in glaucoma: past thinking and current concepts

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Abstract

An understanding of the relationship between functional and structural measures in primary open-angle glaucoma is necessary for both grading the severity of disease and for understanding the natural history of the condition. This article outlines the current evidence for the nature of this relationship and highlights the current mathematical models linking structure and function. Large clinical trials demonstrate that both structural and functional change are apparent in advanced stages of disease, and at an individual level, detectable structural abnormality may precede functional abnormality in some patients, whereas the converse is true in other patients. Although the exact nature of the 'structure–function' relationship in primary open-angle glaucoma is still the topic of scientific debate and the subject of continuing research, this article aims to provide the clinician with an understanding of the past concepts and contemporary thinking in relation to the structure–function relationship in primary open-angle glaucoma.

Key words: function, glaucoma, structure.

What is the 'structure–function relationship' in glaucoma?

Glaucoma is a family of chronic, progressive and potentially blinding optic neuropathies characterized by distinctive morphological (or 'structural') changes of the optic nerve head (ONH) and retinal nerve fibre layer (RNFL) associated with visual field changes.[1] Primary open-angle glaucoma (POAG) is the commonest subtype. Both structural and functional changes result from loss of retinal ganglion cells (RGCs) and their axons. The degree of structural (neuroretinal rim, RNFL) and visual field loss is used to grade the likelihood and severity of POAG both in the clinical setting[2] and in large-scale clinical trials.[3–5] Broadly speaking, 'functional change' in POAG can indicate a disturbance of any test of visual function, although often used synonymously with visual field change. Clinical measurements of structure and function, at any stage of glaucoma, exhibit a wide variability between individuals and on repeated measurements, so the true extent of damage is often difficult to ascertain. Consequently, clinicians use measurements in one domain (structure or function) to support the interpretation of measurements in the other domain. The relationship between

Which goes first – structure or function?

It is often asserted that structural abnormalities precede visual field changes in the development of glaucoma. In fact, there is substantial evidence to suggest that abnormalities of the visual field can precede detectable abnormalities of the ONH or RNFL. The contemporary opinion is that detectable structural and function changes can occur concurrently in some patients, whereas either structural or functional change is apparent first in others.[40]

Many clinicians will be familiar with the statement *'at least 25% to 35% RGC loss is associated with abnormalities in visual field testing'*, a conclusion arising largely from interpretation of the work of Quigley *et al*..[7,41] In fact, Quigley's articles on this topic are among the most frequently cited articles in the literature in ophthalmology.[42] However, their data do not fully support the aforementioned conclusion. In their 1989 study, Quigley *et al*. established normal ranges for RGC density at each visual field test location from only five eyes.[7] When normal confidence limits for healthy eyes are considered, all but four datapoints had RGC density below normal confidence limits for healthy eyes and also had perimetric

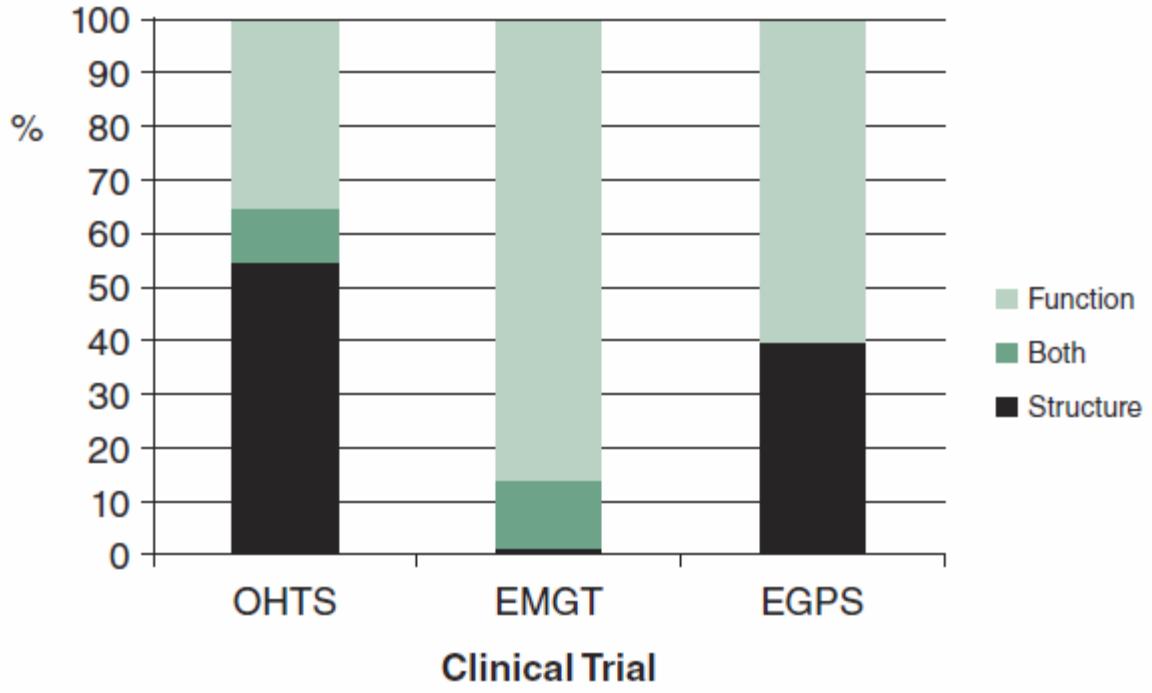

Figure 4. Percentage of patients who had the first detectable glaucomatous change on *structural or functional testing or both tests* in three major clinical trials; EGPS, European Glaucoma Prevention Study; EMGT, Early Manifest Glaucoma Trial; OHTS, Ocular Hypertension Treatment Study.

The 'structure–function dissociation'

Data from large clinical trials show that in some patients structural changes may precede functional changes, whereas in others, the converse is true. In fact, both change together only in a small percentage of patients.[5,49,50] At first glance, this would seem surprising given that structure and function are related by the models described. However, this is in fact what would be predicted for equal loss with expected sources of variability: normal between-subject variability, measurement imprecision, different measurement (dynamic) ranges and different statistical boundaries for defining 'abnormality' all contribute to this dissociation.[60]

Trends in Use of Ancillary Glaucoma Tests for Patients with Open-Angle Glaucoma from 2001 to 2009

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Purpose: To assess trends in the use of ancillary diagnostic tests in the evaluation of patients with open-angle glaucoma (OAG) and glaucoma suspects over the past decade.

Design: Retrospective, longitudinal cohort analysis.

Participants: A total of 169 917 individuals with OAG and 395 721 individuals with suspected glaucoma aged ≥40 years enrolled in a national United States managed care network between 2001 and 2009.

Methods: Claims data were analyzed to assess trends in visual field (VF) testing, fundus photography (FP), and other ocular imaging (OOI) testing for patients with OAG or suspected glaucoma between 2001 and 2009. Repeated-measures logistic regression was performed to identify differences in the odds of undergoing these procedures in 2001, 2005, and 2009 and whether differences exist for patients under the exclusive care of optometrists versus ophthalmologists.

Main Outcome Measures: Odds and annual probabilities of undergoing VF testing, FP, and OOI for OAG from 2001 to 2009.

Results: For patients with OAG, the odds of undergoing VF testing decreased by 36% from 2001 to 2005, by 12% from 2005 to 2009, and by 44% from 2001 to 2009. By comparison, the odds of having OOI increased by 100% from 2001 to 2005, by 24% from 2005 to 2009, and by 147% from 2001 to 2009. Probabilities of undergoing FP were relatively low (13%–25%) for both provider types and remained fairly steady over the decade. For patients cared for exclusively by optometrists, the probability of VF testing decreased from 66% in 2001 to 44% in 2009. Among those seen exclusively by ophthalmologists, the probability of VF testing decreased from 65% in 2001 to 51% in 2009. The probability of undergoing OOI increased from 26% in 2001 to 47% in 2009 for patients of optometrists and from 30% in 2001 to 46% in 2009 for patients of ophthalmologists. By 2008, patients with OAG receiving care exclusively by optometrists had a higher probability of undergoing OOI than VF testing.

Conclusions: From 2001 to 2009, OOI increased dramatically whereas VF testing declined considerably. Because OOI has not been shown to be as effective at detecting OAG or disease progression compared with VF testing, increased reliance on OOI technology, in lieu of VF testing, may be detrimental to patient care.

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GLAUCOMA

Pattern ERG and RNFL thickness in hypertensive eyes with normal blue-yellow visual field

Maurizio G. Uva · Massimo Di Pietro · Antonio Longo · Katia Lauretta · Michele Reibaldi · Alfredo Reibaldi

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Abstract
Purpose To evaluate the transient pattern electroretinogram (t-PERG) and the retinal nerve fiber layer (RNFL) thickness in eyes with ocular hypertension (OH) and normal short-wavelength automated perimetry (SWAP).
Methods In 26 patients with bilateral OH with normal SWAP, and in 26 age and sex matched healthy controls, t-PERG recording and RNFL thickness measurement were performed. Mean deviation (MD) and pattern standard deviation (PSD) of a reliable full threshold 24-2 SWAP were considered. RNFL thickness was determined by OCT3. Monocular PERG were recorded by using a black and white checkerboard pattern (check size 0.9°, contrast 100 %, mean luminance 80 cd/m2) generated on a monitor and reversed in contrast (four reversals per second, 2 Hz) at a distance of 70 cm. Patients had optimal correction at viewing distance; no mydriatic or miotic eye drops were used. Silver/silver chloride skin electrodes were placed over the lower eyelids in the stimulated eye (active electrode) and in the patched eye (reference electrode); ground electrode was in the Fpz scalp. Peak-to peak amplitude of P50 (N35-P50) and N95 (P50-N95) waves, and implicit time of P50, were considered.
Results Compared to controls, in OH eyes, a reduction of N35-P50 amplitude (2.86±1.49 vs. 3.77±1.08 microvolts, −24.1 %, *t*-test $p=0.015$), of average RNFL thickness (88±11 vs. 96±10 μm, −9.5 %, *t*-test $p=0.002$), and of RNFL thickness in superior ($p=0.015$) and inferior quadrant ($p<0.001$), were found. Multivariate analysis showed that in OH eyes, N35-P50 amplitude was inversely related to intraocular pressure (IOP) ($p=0.001$); no correlation was found between N35-P50 amplitude and MD, PSD, CCT or RNFL thickness.
Conclusions In OH eyes, both PERG and RNFL thickness changes occur in hypertensive eyes with undamaged SWAP; the correlation of PERG amplitude with IOP, but not with RNFL thickness, suggests that such PERG changes are an effect of the IOP on retinal ganglion cells, rather than a sign of their loss.

Keywords Pattern electroretinogram · PERG · RNFL thickness · Ocular hypertension · Blue-yellow perimetry · Short-wavelength automated perimetry · SWAP

Introduction

Visual field alterations are a key feature of glaucomatous damage; whereas well established alterations can be evaluated by the white-on-white standard automatic perimetry (SAP), new instruments have been developed for the detection of early changes.

Short wavelength perimetry (SWAP) uses blue stimuli on a yellow background, and it has been reported to detect glaucomatous changes some years before their appearance at SAP [1–3]. The long duration of the test, and the need of a well trained and collaborative patient, reduce its clinical application [4].

Pattern electroretinogram (PERG) is generated by the retinal ganglion cells (RGC): it consists of a prominent positive component at approximately 50 ms and a larger negativity at approximately 95 ms. P50 component may

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The authors have full control of all primary data and they agree to allow Graefe's Archive for Clinical and Experimental Ophthalmology to review their data upon request.

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Neural Networks to Identify Glaucomatous Visual Field Progression

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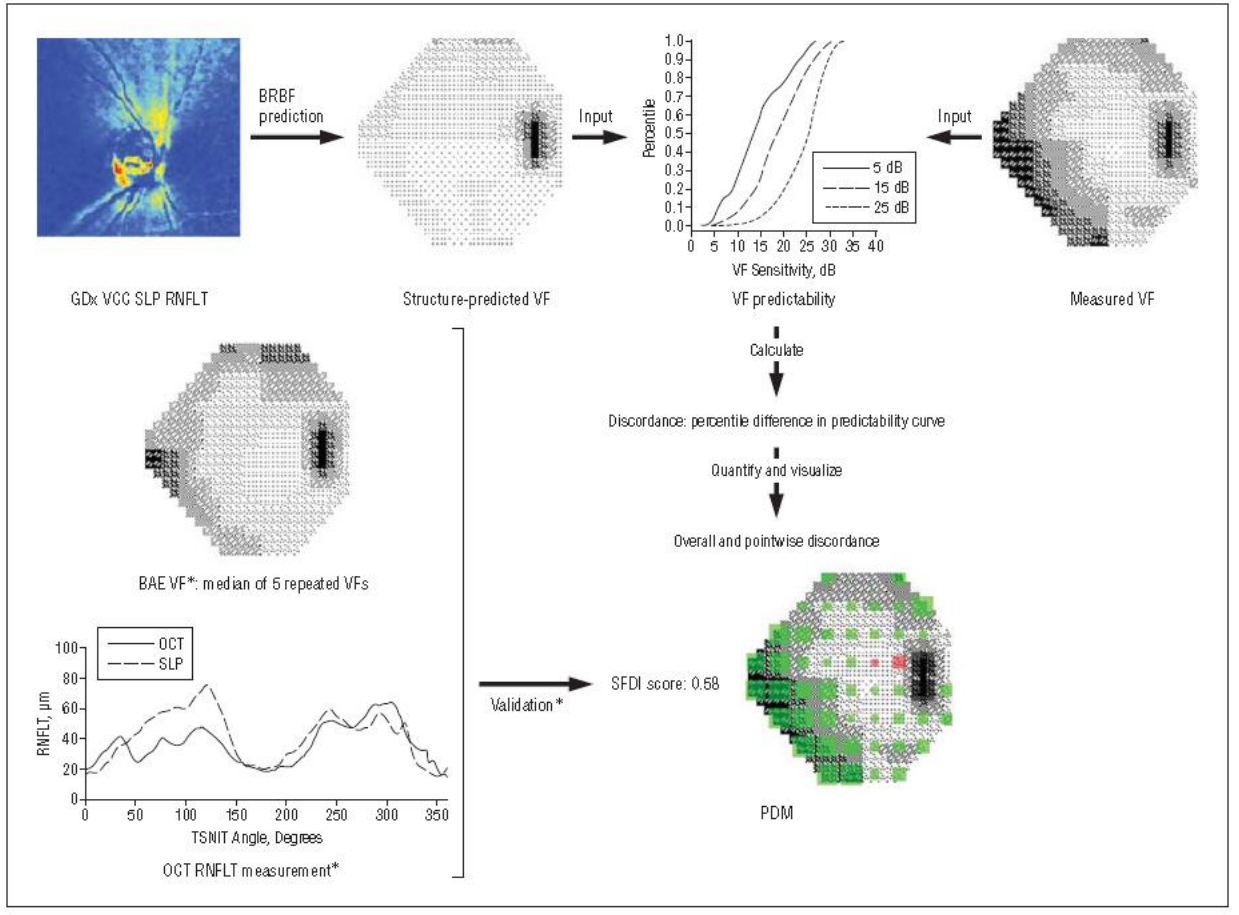
Optimizing and Validating an Approach for Identifying Glaucomatous Change in Optic Nerve Topography

James C. H. Tan and Roger A. Hitchings

Integrating Event- and Trend-Based Analyses to Improve Detection of Glaucomatous Visual Field Progression

Felipe A. Medeiros, MD, PhD,[1] Robert N. Weinreb, MD,[1] Grant Moore, MD,[1] Jeffrey M. Liebmann, MD,[2] Christopher A. Girkin, MD,[3] Linda M. Zangwill, PhD[1]

Strumenti futuri per valutare meglio la discordanza tra struttura e funzione



L'utilizzo di mappe e/o di modelli in cui l'analisi struttura /funzione concorda, aiuteranno i clinici a porre una diagnosi precoce e a valutare l'andamento della malattia nel tempo.

Zhu et al. Arch Ophthalmol 2011;129:167–74.

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Combining measurements from three anatomical areas for glaucoma diagnosis using Fourier-domain optical coherence tomography

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ABSTRACT

Aims To improve the diagnostic power for glaucoma by combining measurements of peripapillary nerve fibre layer (NFL), macular ganglion cell complex (GCC) and disc variables obtained with Fourier-domain optical coherence tomography (FD-OCT) into the glaucoma structural diagnostic index (GSDI).

Methods In this observational, cross-sectional study of subjects from the Advanced Imaging of Glaucoma Study, GCC and NFL of healthy and perimetrical glaucoma subjects from four major academic referral centres of the Advanced Imaging of Glaucoma Study were mapped with the RTVue FD-OCT. Global loss volume and focal loss volume parameters were defined using NFL and GCC normative reference maps. Optimal weights for NFL, GCC and disc variables were combined using multivariate logistic regression to build the GSDI. Glaucoma severity was classified using the Enhanced Glaucoma Staging System (GSS2). Diagnostic accuracy was assessed by sensitivity, specificity and the area under the receiver operator characteristic curve (AUC).

Results We analysed 118 normal eyes of 60 subjects, 236 matched eyes of 166 subjects with perimetrical glaucoma, and 105 eyes from a healthy reference group of 61 subjects. The GSDI included composite overall thickness and focal loss volume with weighted NFL and GCC components, as well as the vertical cup-to-disc ratio. The AUC of 0.922 from leave-one-out cross validation was better than the best component variable alone (p=0.047). The partial AUC in the high specificity region was also better (p=0.01), with a sensitivity of 69% at 99% specificity, and a sensitivity of 80.3% at 95% specificity. For GSS2 stages 3–5 the sensitivity was 98% at 99% specificity, and 100% at 95% specificity.

Conclusions Combining structural measurements of GCC, NFL and disc variables from FD-OCT created a GSDI that improved the accuracy for glaucoma diagnos

Trial registration number NCT01314326.

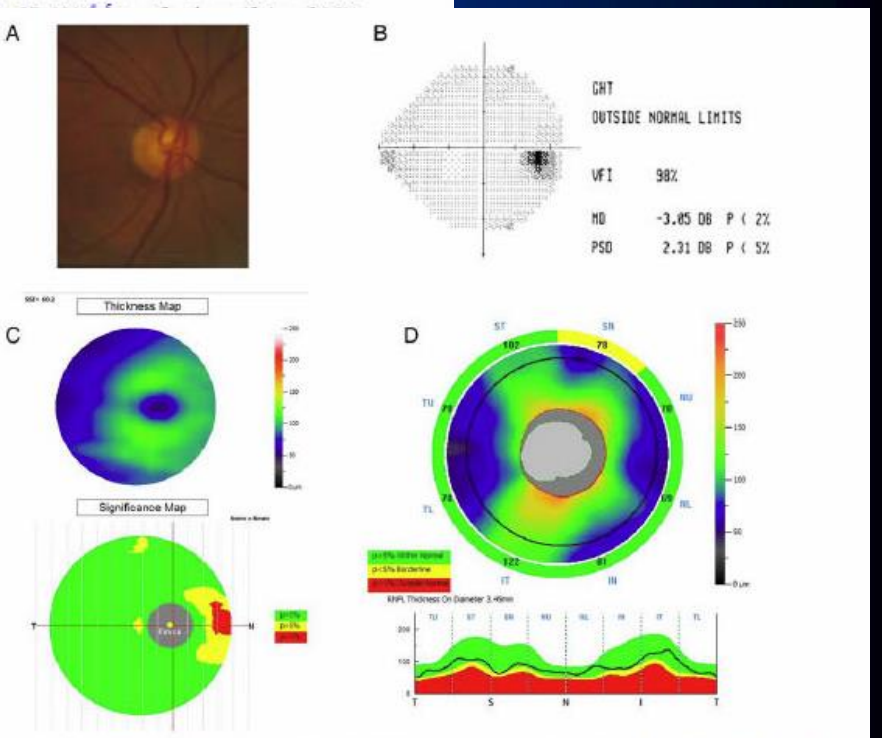

Figure 3 Example of the utility of the glaucoma structural diagnostic index (GSDI). Glaucoma was detected at 99% specificity cut-off using the combined GSDI (0.93) but not with any single optical coherence tomography (OCT) variable. (A) Disc photograph. (B) Visual field test (mean deviation (MD), pattern SD (PSD)). (C) Ganglion cell complex (GCC) thickness and significance map. (D) Retinal nerve fibre layer (RNFL) thickness profile.

DOCTORS VS MACHINES

Can new diagnostic technologies replace experienced glaucoma specialists?

Roibeard O'hEineachain reports

Will advances in technology render ophthalmologists obsolete in the diagnosis and monitoring of glaucoma? Two noted glaucoma specialists debated the question during a session of the 11th Congress of the European Glaucoma Society in Nice.

Taking up the argument that machines will replace doctors, was Francisco Goni MD, PhD, Barcelona, Spain. "With the increasing number of patients in our healthcare systems we really need time-saving, effective, reliable, reproducible, and standardised methodology for disease diagnosis and follow-up," he said.

He noted that although diagnosis by physicians remains the current gold standard, even experienced glaucoma specialists disagree on whether or not an eye has developed glaucomatous damage or if damage that is already present has worsened.

Dr Goni also noted that automated diagnosis is already in use by most glaucoma specialists in the form of computerised perimetry, with both diagnosis and progression analysis software. In fact, the European Glaucoma Society guidelines currently recommend the use of automated progression algorithms.

"Many doctors are making direct clinical decisions based on perimetric diagnostic indicators, like hemifield test or pattern standard deviation, statistical tools that help us separate patients as likely normal or abnormal. Similarly, progression algorithms allow us to detect and measure change in an objective manner. Clinical criteria developed from studies have shown a good sensitivity/specificity balance," he said.

The automation of the detection of the structural abnormalities that characterise glaucoma has kept pace with functional testing. The past decade has seen rapid development in technologies such as scanning laser polarimetry, confocal scanning laser ophthalmoscopy and optical coherence tomography.

"These imaging devices have similar or even better diagnostic performance than clinical assessment by doctors. Their measurements are quantitative and show a high reproducibility, allowing the detection of change with strong confidence contrary to the qualitative, subjective evaluation of progression performed in a classic doctor's clinical examination. Thus nowadays an increasing number of doctors rely only on imaging devices to detect and measure structural progression," he said.

If the visual field in the same patient stays normal it is probably justified to start medical therapy...

Anton Hommer MD

DOCTORS STILL NEEDED

Taking the opposing view, Anton Hommer MD, Vienna, Austria, agreed that computerised perimetry is here to stay, but without the supervision of a trained physician the automated tests are still prone to error because of fixation loss, and other diseases that can affect visual fields.

Furthermore, structural features detected by some of the newer imaging technologies can also be misleading, not only because changes similar to those that occur in glaucoma can also occur in other optic neuropathies, but also because there is so much variation in the optic nerve head and retinal nerve fibre layer among individuals without any disease at all.

For example, in the normal population the retinal nerve fibre layer thickness at the optic nerve head can vary by between 800,000 and 1.6 million nerve fibres. Similar differences exist in the normal population regarding the size of the optic nerve head and cup/disc ratios and the degree of asymmetry between the two eyes.

In addition, all of the current technologies have certain shortcomings. For example, the accuracy of early diagnosis with both the GDx-VCC scanning laser polarimeter (Carl Zeiss Meditec) and the HRT II confocal scanning laser ophthalmoscope (Heidelberg Engineering) is dependent on the disc size of the eye being examined. The GDx-VCC is more sensitive with small discs and the HRT II is more sensitive with large discs.

Spectral domain OCT with the Spectralis* system (Heidelberg Engineering), for its part, is limited by the size of its normative database, which includes only 246 eyes of 123 patients, all of whom were Caucasian and between the ages of 20 and 87 years. Normative database is limited in all machines/OCTs, Dr Hommer pointed out.

However the new imaging technologies have much to offer in the detection of progression, Dr Hommer said.

He suggested using a two-step approach.

"We know that with the available techniques nowadays structural changes are more likely to be detected at the early stage of glaucoma and functional examination is superior in advanced stages for detecting progression. Structural deviation from normal does not automatically mean that there is a disease.

"But if we have confirmed change in OHT or early glaucoma with the structural measurements (not only one follow-up picture) it may be justified to start or change treatment. If the visual field in the same patient stays normal it is probably justified to start medical therapy, but not to perform surgery, because of more potential severe complications for a patient that had no loss in QoL before. If we have on the other side confirmed visual field progression due to glaucoma, but structure stays unchanged, because of advanced damage yet and the measurements are not providing useful information, then the functional tests are more valuable," he said.

"Therefore, all these high tech measurements are good for follow-up, but we always have to consider the patient individually as a whole, and this cannot be done by machines," he said.

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Treating patients presenting with advanced glaucoma—should we reconsider current practice?

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ABSTRACT
The management of patients presenting with advanced glaucoma presents a challenge to glaucoma clinicians. Presentation with advanced visual field loss is an important risk factor for progression to blindness in the affected eye(s) during the patients' lifetime. Maximising intraocular pressure (IOP) control in such situations is likely to minimise the risk of further visual field deterioration thus either preventing or slowing progression to blindness. Currently most patients presenting with advanced disease in the UK are managed on an escalating regime of medical treatment.

In the UK and other similar populations, glaucoma is estimated to be present in about 2% of the population over the age of 40 years, increasing with age[3–7] and affecting as many 10% of those in their 80s. It is the second commonest cause for registration as visually impaired in the UK, accounting for 11.6% of registrations over the age of 65 years,[8] although, again, this is likely to be underestimate.[9]

Between 10% and 39% of patients with glaucoma present with advanced disease in at least one eye in the UK.[10–15] In the most recent study, more than a third of patients presenting had severe

Between 10% and 39% of patients with glaucoma present with advanced disease in at least one eye in the UK.[10–15] In the most recent study, more than a third of patients presenting had severe disease in at least one eye at presentation.[10] Those

Institute for Health and Clinical Excellence (NICE) guidelines in which the suggested management approaches for patients presenting with advanced and early disease differ.[1] The principle difference is the recommendation that primary surgical intervention should be offered to patients with advanced disease. This review examines the evidence supporting this recommendation.

BACKGROUND AND EPIDEMIOLOGY

The WHO estimates that in 2010, 4.5 million people are blind due to glaucoma[1a], accounting for 12.5% of global blindness. A substantial increase is predicted over the next few years.[2] These figures are almost certainly an underestimate because of the way in which causes of blindness in patients with more than one pathology are assigned in prevalence surveys, and because most blindness surveys do not consider subjects functionally blind due to severely restricted visual field.

when large, rapid linear progression of visual field loss occurred. Oliver found that unilateral blindness more than doubled the risk of blindness.[22]

CURRENT TREATMENT OPTIONS

Reducing IOP is currently the only effective treatment for glaucoma.[23–26] The Advanced Glaucoma Intervention Study (AGIS) demonstrated that the extent of IOP-lowering was related to the progression of visual fields over an 8-year period, showing that progression was least when IOPs were maintained below 18 mm Hg at all follow-up visits.[27]

Primary treatment may involve a medical, laser or surgical intervention. Currently most ophthalmologists treat patients medically starting with topical drop monotherapy followed by escalating drop therapy until maximum tolerated therapy is achieved. In patients who continue to progress or in whom target IOP is not achieved, clinicians may opt for surgical intervention, most frequently

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Clinical Study

Comparison of Newly Diagnosed Ocular Hypertension and Open-Angle Glaucoma: Ocular Variables, Risk Factors, and Disease Severity

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Purpose. To describe the distribution of ocular variables, risk factors, and disease severity in newly diagnosed ocular hypertension (OH) or open-angle glaucoma (OAG). *Methods*. Eligible subjects underwent a complete history and examination. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) obtained from multiple logistic regression models were used to compare OAG to OH and advanced to early/moderate OAG. *Results*. 405 subjects were enrolled: 292 (72.1%) with OAG and 113 (27.9%) with OH. 51.7% had early, 27.1% moderate, and 20.9% advanced OAG. The OR for OAG versus OH was 8.19 ($P < 0.0001$) for disc notch, 5.36 ($P < 0.0001$) for abnormal visual field, 1.45 ($P = 0.001$) for worsening mean deviation, 1.91 ($P < 0.0001$) for increased cupping, 1.03 for increased age ($P = 0.030$), and 0.36 ($P = 0.010$) for smoking. *Conclusions*. Increased age was a risk for OAG, and smoking decreased the risk of OAG compared to OH. Almost half of the OAG subjects had moderate/advanced disease at diagnosis.

diagnosis.

The 12-year Incidence of Glaucoma and Glaucoma-related Visual Field Loss in Italy: The Ponza Eye Study

Claudio Cedrone, PhD, Raffaele Mancino, MD,* Federico Ricci, MD,* Angelica Cerulli, MD,* Franco Culasso, PhD,† and Carlo Nucci, MD, PhD**

Results: The 12-year incidence of definite POAG was 3.8% (95% confidence intervals (CI), 2.3-6.2), that is, an average annual rate of 0.32%. Corresponding rates for PACG and PEX glaucoma were 0.5% (95% CI, 0.1-1.8) and 0.8% (95% CI, 0.3-2.2), respectively. Half the incident glaucoma cases (45%) had not been diagnosed earlier. Fifty-five percent of the incident POAG eyes had Bascom-Palmer stage 1 or 2 disease and 40% of the incident PACG or PEX glaucoma eyes had stage 3 or 4 disease. Seven of 20 incident glaucoma cases presented with monocular or binocular visual loss because of advanced visual field loss. Significant risk factors for POAG included high myopia (> 6.0 D), intraocular pressure ≥ 22 mm Hg, and glaucoma family history.

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Disclosure: The authors declare no conflict of interest.
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chamber. Participants meeting these criteria and 50% of those who did not (randomly selected) underwent VF testing with the Humphrey 30-2 Program. VF changes were defined as glaucomatous when the following criteria were met: (1) corrected pattern standard deviation < 5% and/or glaucoma hemifield test results exceeding normal limits; (2) absence of other disease that would explain the field abnormality; (3) fixation loss rate of < 25%; and (4) a combined false-positive and false-negative rate of < 33%. The Bascom-Palmer (or Hoddapp-Anderson-Parish) system based on VF

Avoidable sight loss from glaucoma: is it unavoidable?

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ABSTRACT

Aims To review the characteristics of patients attending a tertiary ophthalmic referral centre certified as sight impaired (SI) or severely sight impaired (SSI) from glaucoma.

Methods One hundred consecutive patients certified SI/SSI from the Glaucoma Service at Moorfields Eye Hospital, London, from January 2007 were identified from the England and Wales certification of visual impairment database. Clinical and demographic data were collected from hospital case records.

Results The median (IQR) age of patients at presentation was 66.3 (55.6 to 75.3) years; median (IQR) interval to certification was 62.2 (22.5 to 129.3)

coma, patients are referred through the National Health Service (NHS) to their local hospital eye service (HES) to confirm diagnosis and commence treatment.

In England and Wales, patients with sight impairment (SI; formerly known as 'partially sighted') or severe visual loss ('severely sight impaired' (SSI), formerly known as 'blind') are offered the certificate of visual impairment (CVI; formerly known as the BD8 certificate). The definition of the terms SI and SSI have been outlined by the Department of Health (DoH) (England).[5] The decision to certify a patient as SI or SSI lies with the consultant ophthalmologist and is

Conclusions Over 80% patients on the certification of visual impairment register from Moorfields Eye Hospital with glaucoma as the primary cause had a significant visual disability at presentation, with almost two-thirds of patients presenting bilaterally 'blind'. There appear to be delays to certification. Despite being under the hospital eye service, a number of glaucoma patients still progress to certifiable visual impairment.

from primary open angle glaucoma (POAG).

It is thought that POAG remains relatively asymptomatic until advanced stages of the disease.

METHODS

In this retrospective study, the England and Wales electronic CVI database was examined and the first

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18:30-19:30	Meet the expert session

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