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Advances in Ophthalmology

Glaucoma: Diagnosis and Treatment

Newer Diagnostic Techniques

Introduction

The nature and treatment of glaucoma has long been a problem for ophthalmologists throughout the world in that it has no 'fingerprint identity'. For years, ophthalmologists used the concept that an intraocular pressure (IOP) above 21 mm Hg was synonymous with glaucoma and this was used as the basis to identify the disease. However, a substantial number of people with IOPs above 21 mm Hg will never develop glaucoma, while nearly 25% of people with glaucoma have normal IOPs.

Therefore, while high IOP is a risk factor for glaucoma, other factors are also involved. These factors include the following:

- family history
- appearance of the optic disc – this is the single most important clue to the diagnosis of open angle glaucoma
- gonioscopic appearance – this is essential to detect angle closure glaucoma
- visual field status – this is only helpful in the later stages of the disease when optic disc changes have already occurred.

The conventional diagnostic techniques of optic nerve appearance, IOP levels, and visual fields are useful, but limited in their reliability due to their subjectivity. New diagnostic methods have recently been devised to objectively identify the progression and evolution of the disease at its earliest point.

Optical Coherence Tomography

Optical coherence tomography (OCT) is a non-invasive, non-contact transpupillary imaging technology that can image retinal structures in vivo with an axial resolution of 10 to 15 µm.[1] OCT provides quantitative objective, and reproducible assessment of retinal and retinal nerve fibre layer (RNFL) thickness (Figure 1). There is good correlation between peripapillary RNFL thickness measured by OCT and visual function, as well as histological measurements of the nerve fibre layer thickness. Although the main applications of OCT are retinal diseases, there are many reports of its use in glaucoma.[2] In addition, a significant correlation has been found between glaucomatous visual field defects and reductions in macular thickness.

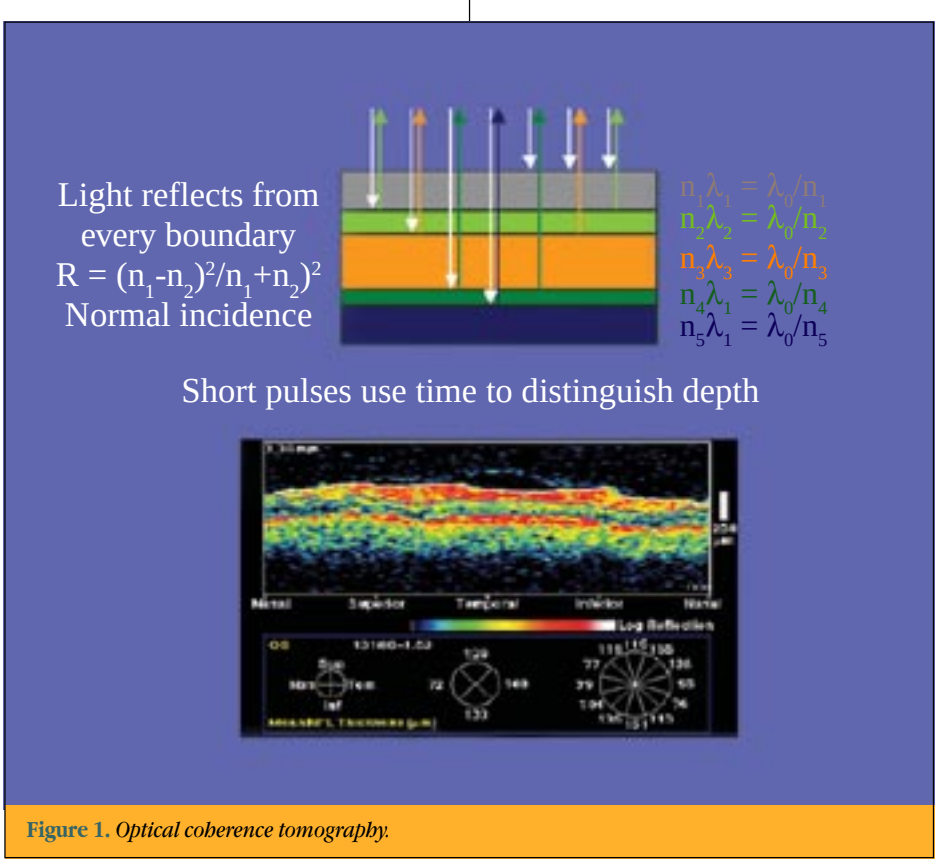

Figure 1. *Optical coherence tomography.*

A study was therefore performed to evaluate the correlation between macular thickness and RNFL thickness measured by OCT in normal eyes and those with moderate glaucomatous optic neuropathy. Greenfield et al performed complete examination, automated achromatic perimetry, and optical coherence tomography of the peripapillary RNFL and macula. Macular thickness measurements were generated using 6 radial optical coherence tomographic scans (5.9 mm) centred on the fovea, and mean and quadrantic macular thickness values were calculated.

Fifty nine eyes of 59 patients were enrolled. All 30 eyes with glaucoma had associated visual field loss (mean defect, -8.4 ± 5.8 dB). Mean macular thickness was significantly associated with visual field mean defect ($p < 0.001$), pattern standard deviation ($p < 0.001$), and mean RNFL thickness ($p < 0.001$; Table 1).

In glaucomatous eyes with visual field loss localised to 1 hemifield (n = 11), macular thickness in the quadrant associated with the field defect (277 ± 28 µm) was significantly less than in the unaffected quadrant (286 ± 27 µm; $p = 0.005$). Mean RNFL thickness in the affected quadrant (89 ± 53 µm) was significantly thinner than in the unaffected quadrant (121 ± 39 µm; $p = 0.009$). These authors concluded that macular thickness changes are well correlated with changes in visual function and RNFL structure in glaucoma and may be a surrogate indicator of retinal ganglion cell loss.

Meanwhile, Giovannini et al evaluated the macular volume in normal and glaucomatous eyes using OCT in 50 eyes of 30 patients (20 healthy, 15 with early glaucoma, and 15 with advanced glaucoma).[2] All eyes were examined at a scan length of 3.44 mm vertically across the fovea. OCT macular retinal thickness maps were used to calculate macular volume. Significant differences were observed between the groups — healthy eyes and those with early glaucoma had significantly greater volume than eyes with advanced glaucoma (Table 2). These researchers concluded that volumetric analysis of macular thickness with OCT may be a useful method of documenting and monitoring patients with early glaucoma and advanced glaucoma since OCT macular volumes correlate significantly with glaucoma status.

Table 1. *Macular and peripapillary retinal nerve fibre layer (RNFL) thickness in healthy and glaucomatous eyes (n = 59) by optical coherence tomography.*

Thickness (µm)	Healthy eyes (n = 29)	Glaucomatous eyes (n = 30)	p Value
Macular	*304 ± 15*	*278 ± 24*	*< 0.001*
RNFL	*140 ± 14*	*91 ± 31*	*< 0.001*

Similarly, Guedes et al found that both macular and RNFL thickness as measured by OCT showed statistically significant correlations with glaucoma using a prototype and commercial OCT device ($p < 0.001$; Table 3).[3] These authors concluded that macular and NFL thickness measurements made with OCT may be useful in the clinical assessment of glaucoma

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Arteriolar Pressure Attenuation Index

Vascular phenomena are considered important to optic nerve and visual field progression in POAG.[6] Patients with ocular hypertension and glaucoma demonstrate retinal arteriolar narrowing compared with healthy controls. Histopathological studies show optic disc capillary changes in glaucoma and the link between changes in ocular perfusion pressure and POAG progression also points to the importance of vascular phenomena in POAG. The arteriolar Pressure Attenuation Index (PAI) was therefore developed to relate retinal vessel calibre changes to pressure-attenuating effects.

Cohen et al examined whether the PAI could predict ocular hypertension progression to POAG. The PAI was calculated for 27 eyes of 14 patients with OHT using initial and final digitised optic disc photographs taken during a follow-up interval of 5 to 18 years. Serial stereo color disc photographs and visual fields were analysed to determine progression. At baseline, the arteriolar tree of 8 eyes with ocular hypertension that progressed to POAG demonstrated a 45.8% greater mean PAI value than that of 7 eyes that did not progress (5.31 ± 0.93 vs 3.64 ± 0.34; $r = 0.83$). Progression was independent of baseline cup-disc ratio.

Table 2. *Macular volume in healthy and glaucomatous eyes.*

Eyes	Macular volume (mm^3)
Healthy	*7.35 ± 0.455*
Early glaucoma	*7.09 ± 0.475*
Advanced glaucoma	*6.678 ± 0.455*

The PAI values of patients with stable ocular hypertension remained stable after a median follow-up of 12 years. The PAI values of patients with OHT who progressed demonstrated a further increase of 19.97% ± 11.24% during a median follow-up of 6 years. These results support the hypothesis that low end-arteriolar pressure predicts progression from ocular hypertension to POAG and suggest that the PAI provides a new, early, reproducible method to study vascular phenomena in glaucoma.

GDx

The GDx is a scanning laser polarimeter that has been developed to allow the quantitative analysis of RNFL thickness.[4,5] GDx

Table 3. *Macular and retinal nerve fibre layer (RNFL) thickness in relation to diagnosis.*

Thickness	Healthy eyes	Glaucoma suspect	Early glaucoma	Advanced glaucoma
Prototype device				
Macular *RNFL*	*229.0 ± 13.4* *113.6 ± 15.8*	*224.9 ± 16.0* *106.0 ± 18.9*	*224.0 ± 14.5* *94.7 ± 22.2*	*214.5 ± 17.0* *64.3 ± 27.3*
Commercial device				
Macular *RNFL*	*236.2 ± 13.9* *114.8 ± 13.1*	*230.9 ± 17.9* *1.4.9 ± 14.4*	*220.5 ± 16.7* *83.2 ± 14.2*	*204.0 ± 37.7* *47.2 ± 14.8*

The Disc Damage Likelihood Scale

The Disc Damage Likelihood Scale (DDLS) is a new method of estimating the amount of optic disc damage in patients with glaucoma based on the radial width of the neuroretinal rim measured at its thinnest point. There are 8 stages, extending from no damage to far advanced damage (Figure 2).

The unit of measurement is the rim/disc ratio, that is, the radial width of the rim compared with the diameter of the disc in the same axis. When there is no rim remaining, the rim/disc ratio is 0. The circumferential extent of rim absence (0 rim/disc ratio) is measured in degrees. Caution must be taken to differentiate the actual absence of rim from sloping of the rim as can occur temporally in some patients with myopia, for example. A sloping rim is not an absent rim. Since rim width is a function of disc size, disc size must be evaluated prior to attributing a DDLS stage. This is done with a 60 D to 90 D lens with appropriate corrective factors.

The reproducibility of the DDLS has been measured by 2 masked observers staging 48 optic nerve stereoscopic photographs by 2 different methods, the cup/disc ratio and the DDLS. In addition, reproducibility was assessed by 3 observers examining 34 eyes of 24 patients.

With regard to the photographs, the intraobserver and interobserver reproducibility was better using the DDLS than the cup/disc ratio (98% versus 85% for intraobserver reproducibility, and 85% versus 74% for interobserver reproducibility). The DDLS correlated better with the Humphrey Visual Field than did any Heidelberg Retina Tomograph parameter.

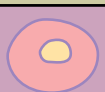
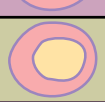
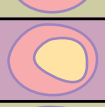
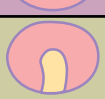
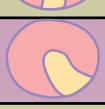
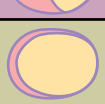
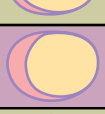
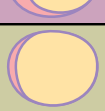
Narrowest width of rim (rim/disc ratio)				Examples			
DDLS stage	For small disc <1.50 mm	For average size disc 1.50-2.0	For large disc >2.00	DDLS stage	1.25 mm optic nerve	1.75 mm optic nerve	2.25 mm optic nerve
0a	>0.50	>0.40	>0.30	0a			
0b	0.40 - 0.49	0.30 - 0.39	0.20 - 0.29	0b			
1	0.30 - 0.39	0.20 - 0.29	0.10 - 0.19	1			
2	0.20 - 0.29	0.10 - 0.19	< 0.10	2			
3	0.10 - 0.19	<0.10	0 for <45º	3			
4	<0.10	0 for <45º	0 for 46º - 90º	4			
5	0 for <45º	0 for 46º - 90º	0 for 91º - 180º	5			
6	0 for 46º - 90º	0 for 91º - 180º	0 for 181º - 270º	6			
7a	0 for 91º - 180º	0 for 181º - 270º	0 for >270º	7a			
7b	0 for >180º	0 for >270º		7b			

Figure 2. *The disc damage likelihood scale (DDLS).*

Spaeth GL, Henderer J, Liu C, et al. The disc damage likelihood scale: reproducibility of a new method of estimating the amount of optic nerve damage caused by glaucoma. Trans Am Ophthalmol Soc 2002;100:181-185.

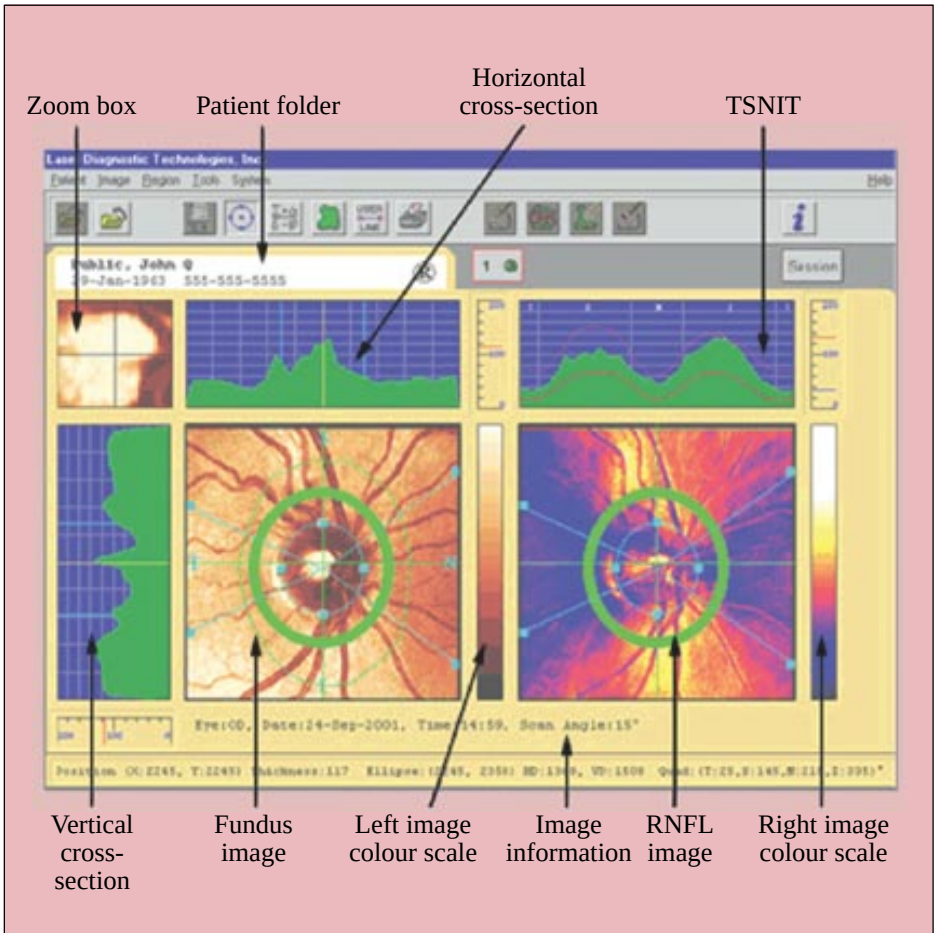


Figure 3. *Computer image of GDx scanning laser polarimetry.*

measurements are highly reproducible, with a mean SD consistently below 10 µm, and a large normative database has been collected in several centres in the USA, Europe and Asia.[4]

Valuable characteristics of GDx include the following:

- the examination is made without pupil dilation
- there is a short time required for image acquisition (0.7 seconds)
- there is a built in image quality assessment
- the examination may be performed for anyone wearing contact lenses, with silicon oil in the vitreous, or with intraocular lenses.

After acquiring the patient's image, the screen shows the image seen in Figure 3. From there it is a quick step to printing out the report.

A statistically significant difference between healthy and glaucomatous eyes, as well as between eyes with raised IOPs and those with glaucoma has been reported. Conflicting results have been reported for differences between healthy and ocular hypertensive eyes, although small differences have been noted between eyes with POAG and normal tension glaucoma.

The diagnostic capacity for discriminating between healthy and glaucomatous eyes has been reported to range from <70% to >90% and the number of both false-negative and false-positive results may be relatively high.[4] Therefore, further assessment of the technique is required to determine whether it may be useful in screening for the presence of early glaucomatous damage.[5] However, GDx may be used for monitoring purposes given the high reproducibility of the measurement and the stability of corneal polarisation which allow accurate comparisons to be performed.

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Neuroprotective Agents in the Treatment of Glaucoma

Introduction

While there is a consensus that intraocular pressure (IOP) is a major risk factor for glaucoma, the most recent definition of primary open angle glaucoma (POAG) is a progressive multifactorial optic neuropathy characterised by specific morphological changes (optic disc cupping) resulting in acquired loss of retinal ganglion cells (RGCs) and RGC axons. This process is characterised by visual field loss and other functional changes.

It is clear that there is a relationship between IOP and glaucoma since patients with higher IOPs have a greater risk for glaucomatous nerve damage.[1] However, not all patients with elevated IOPs go on to develop glaucoma and not all people with glaucoma have elevated IOPs. Indeed, many people with glaucoma continue to experience visual field loss long after normalisation of their IOP.[2] The current thinking is that glaucoma is a neurodegenerative disease that may be amenable to neuroprotective therapy.

Mechanisms of Optic Nerve Injury

The most common optic neuropathy is glaucomatous optic neuropathy, distinguished by a distinctive and progressive excavation of the optic nerve head without significant pallor of the remaining neuroretinal rim.[3] The final common pathway of optic nerve injury is retinal ganglion cell death as a result of programmed cell death (apoptosis). While this process is necessary for the normal renewal of many tissues such as corneal epithelium and skin, this loss is permanent in the case of neural tissues. Ganglion cell injury and eventual death in glaucoma is caused by several factors, including mechanical stress, blockage of axoplasmic transport, deficient autoregulation and chronic ischaemia, increase in excitatory neurotoxins, genetic influences, immune phenomena, and secondary degeneration.

Neuroprotection

The finding that glaucoma is a neurodegenerative disease has led to suggestions that its treatment should include neuroprotective therapy in addition to lowering IOP.[4] Neuroprotection can be achieved by counteracting risk factors or increasing the resistance of cells to the stressful conditions. A more favourable approach, however, is to harness and augment the tissue's own defence mechanisms.

Among the toxic risk factors triggered by the degenerating nerve is an uncontrolled increase in such compounds as the excitatory amino acid glutamate, with harmful consequences for the tissues. Glutamate is a neurotransmitter, but is neurotoxic when its physiological levels are exceeded (secondary degeneration). Glutamate has been found to be increased in the vitreous of patients with glaucoma. Similarly, increased nitric oxide has been found in the retinas of eyes with damaged optic nerves.

Attempts to halt the spread of damage have included neutralising the mediators of toxicity, inhibiting signal transduction associated with death signals, and increasing the resistance of vulnerable neurones to the injurious conditions. There is evidence that neuroprotection can be achieved both pharmacologically and immunologically. Pharmacological intervention (using selective α_2-adrenergic receptor agonists) neutralises some of the effects of the nerve-derived toxic factors and possibly increases the ability of the remaining healthy neurones, at any given time, to cope with the stressful conditions. There are 4 criteria for neuroprotective drugs in the retina — brimonidine, an α_2-adrenoreceptor agonist, meets all these criteria, as shown in Table 1.[5]

Brimonidine

Brimonidine both reduces IOP in glaucoma and protects the optic nerve and retinal ganglion cells from secondary degeneration caused by the cascade of autodestructive events relating to the primary injury.[6] It is thought that activation of the α_2-receptor results in a neuroprotective signalling pathway that enhances neuronal survival.[5] Brimonidine decreases IOP by suppressing aqueous humour flow and increasing uveoscleral outflow.[6]

Lafuente et al performed a study to investigate the dose-response effects of topically administered brimonidine on retinal ganglion cell survival after transient retinal ischaemia in adult Sprague-Dawley rats.[7] The following results were found:

- 7 days after 90 minutes of transient ischaemia there is a loss of approximately 46% of retinal ganglion cells
- topical pre-treatment with brimonidine prevents ischaemia-induced retinal ganglion cell death in a dose-dependent manner (Table 2)
- between 7 and 21 days after the ischaemia occurred, there was an additional slow cell loss of approximately 25% of the retinal ganglion cell population. Pre-treatment with 0.1% brimonidine significantly reduced this slow cell death.

These authors concluded that the neuroprotective effects of brimonidine, when administered topically, are dose-dependent and that the 0.1% concentration achieves optimal neuroprotective effects against the early loss of retinal ganglion cells. Furthermore, this concentration is also effective at diminishing the protracted loss of retinal ganglion cells that occurs with time after a transient ischaemic attack.

In a 3-month multi-center, open-label study of patients with glaucoma, topical β-blockers were substituted with brimonidine 0.2%.[8] The primary outcome measures were reduction in intraocular pressure (IOP) from baseline at

Table 1. *Criteria for neuroprotective drugs in the retina.*

Criteria	Action of brimonidine
Has a specific target in the retina/optic nerve	*The α_2-receptor is present in the retina and is targeted by brimonidine*
Reaches the retina at pharmacological levels	*Brimonidine is a highly potent and selective α_2-adrenoreceptor agonist with the ability to activate its target at concentrations above 2 nM*
Has a mechanism of action that enhances a neurone's survival of stress or blocks a toxic insult	*The activation of the receptor results in ganglion cell protection under conditions of acute and chronic stress and/or injury*
Demonstrates activity in human clinical trials	*Brimonidine has demonstrated a broad neuroprotective profile in different types of injury — further study should demonstrate a proof of principle for neuroprotective therapy in humans*

Table 2. *Ischaemia-induced retinal ganglion cell death after different doses of brimonidine.*

Brimonidine dose	Retinal ganglion cell death (% cell population)
0.0001%	*37%*
0.001%	*24%*
0.01%	*10%*
0.1%	*0*

2 hours post-dose, change in cardiac and respiratory function, quality of life, and patient satisfaction. The average duration of topical β-blocker therapy was 5 years.

Brimonidine twice daily produced an additional mean reduction in IOP of 8.2% (1.4 mm Hg; $p < 0.001$). Eighty eight percent of patients (60/68) reported being at least as satisfied with brimonidine as with their previous regimen, and 46% (31/68) reported greater satisfaction with brimonidine.

Additionally, 40% of patients (27/68) noted increased energy after the switch, and 43% (29/68) described brimonidine as more soothing than topical β-blockers. In this population, the replacement of topical β-blockers with brimonidine twice daily significantly decreased IOP, improved quality of life, and enhanced patient satisfaction.

Brimonidine has been shown to increase mean sensitivity in visual field tests and it is possible that the neuroprotective qualities of brimonidine may contribute to visual field preservation in glaucomatous eyes.[9] Certainly, brimonidine possesses a mix of interesting properties that should make it a useful addition to the tools available for the chronic treatment of glaucoma.[6]

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Optic Disc and Nerve Fibre Imaging

From the South East Asian Glaucoma Interest Group Meeting — Glaucoma: Global and South East Asian Perspectives — Manila, The Philippines, 26-28 September 2002

GDx Nerve Fibre Analyser

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Current methods for screening and monitoring glaucoma are wanting. On occasion, intraocular pressure measurements, perimetry results, or disc examination are not sufficient and may need to be repeated before management decisions can be made. New structural imaging tests such as the GDx offer quantitative information on the thickness of the peripapillary retinal nerve fibre layer at precise locations around the disc. Since glaucoma is more accurately defined as a disease caused by progressive retinal nerve fibre layer loss, to quantify the amount of nerve fibres at any given moment presents a theoretical advantage.

The GDx is a scanning laser polarimeter that measures direct changes in polarisation (retardation) due to biorefringent properties of the RNFL. The test is comfortable and quick to perform, without the need for dilation and is less dependent on clear media. Most importantly, the sensitivity, specificity, and reproducibility values are sufficiently high for clinical use.

Optical Coherence Tomography

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Clinical examination of the optic disc and nerve fibre layer, and fundus photography are the most common methods for detecting and monitoring structural changes in glaucoma. However, these methods are subjective and only semi-quantitative. Recent advances in computer-based ocular imaging technology provide an objective and quantitative means for detecting structural abnormalities of the optic disc and RNFL.

With optical coherence tomography, a scanning inferometer is used to obtain cross-sectional data of the retina based on the temporal delay and reflectance of back-scattered low-coherence near infra-red light from the different layers of the retina. A high reflectance layer located posterior to the inner surface of the retina, which corresponds with the RNFL, is measured with a computer algorithm to give the RNFL thickness. Optical coherence tomography therefore has a potential role in the detection and management of glaucoma.

Diagnostic Techniques for Visual Field Evaluation

Results with the Medmont and Humphrey Perimeters

The Humphrey field analyser (HFA), Humphrey-Zeiss frequency doubling perimeter, and the Medmont automated perimeter (MAP) are 3 commonly used automated perimeters with threshold achromatic methodologies. Visual field loss may be detected earlier with strategies that target cell lines with reduced redundancy or that suffer selective damage.

To compare these 3 perimeters, 63 patients with suspected glaucoma, ocular hypertension, or glaucoma, plus healthy controls were selectively recruited. All patients underwent testing using MAP central threshold, MAP flicker perimetry, HFA full threshold perimetry, HFA SITA perimetry, HFA short wavelength perimetry (SWAP), and frequency doubling perimetry (FDP). After visual field testing, equivalent tests were compared: MAP central threshold with HFA full threshold perimetry and HFA SITA perimetry; Medmont flicker perimetry with HFA SWAP and FDP.

On analysis of the MAP central threshold a kappa statistic and an area under the receiver operator curve (AUC) of 0.90 and 0.94, respectively, were found compared with HFA full threshold strategies, and 0.87 and 0.92 respectively, compared with HFA SITA. For MAP flicker perimetry, a kappa statistic and an AUC of 0.65 and 0.81, respectively, were found compared with HFA SWAP and 0.87 and 0.96, respectively, compared with FDP. A quadrant analysis and comparison of mean defect between tests was also highly significant. Medmont and Humphrey perimeters correlated well and both may be used for clinical and research purposes with similar confidence.

Landers J, Sharma A, Goldberg I, Graham S. A comparison of perimetric results with the Medmont and Humphrey perimeters. Br J Ophthalmol. 2003;87:690-694.

Automated Perimetry by the American Academy of Ophthalmology

The purpose of this paper was to summarise and evaluate the effectiveness of new automated perimetry tests and algorithms in diagnosing glaucoma and detecting disease progression. A literature search of automated perimetry retrieved more than 300 citations from 1994 to 2001, of which 71 were selected as relevant to this assessment. The quality of the evidence obtained from these studies was assessed by the methodologist.

The 4 automated perimetry techniques described in this assessment are short wavelength automated perimetry (SWAP), frequency doubling technology perimetry (FDT), high-pass resolution perimetry (HPRP), and motion automated perimetry (MAP). The algorithms described are Swedish interactive threshold algorithm (SITA) and SITA fast. With the exception of SWAP, these techniques and algorithms reduce testing time and inconsistent patient performance when compared with conventional full threshold testing.

SWAP detected visual field loss earlier than standard threshold automated perimetry, with a sensitivity and specificity of approximately 88% and 92%, respectively. However, this is a lengthy, demanding test, is sensitive to media opacities, and has a greater magnitude of long-term fluctuation compared with standard threshold automated perimetry, which make it difficult to accurately assess disease progression.

When compared with standard threshold automated perimetry, FDT perimetry showed sensitivity and specificity greater than 97% for detecting moderate and advanced glaucoma, and sensitivity of 85% and specificity of 90% for early glaucoma. Since FDT perimetry has a short testing time and is resistant to blur and pupil size, it may be a useful screening tool.

In a longitudinal study, high-pass resolution perimetry was more effective than standard threshold automated perimetry in monitoring progressive glaucomatous loss, detecting progression at a median of 12 months earlier in 54% of patients studied. Motion automated perimetry demonstrated usefulness in detecting early glaucomatous visual loss in a longitudinal study.

Studies on SITA demonstrated greater sensitivity and reproducibility and less intertest variability when compared with standard full threshold testing and a 50% reduction in testing times. A study comparing standard full threshold, SITA, and SITA fast found a sensitivity of 95% for the first 2 techniques and 93% for SITA fast.

Long-term follow-up studies are needed to assess the ability of these techniques to detect progression of glaucoma over time.

Delgado MF, Nguyen NT, Cox TA, et al. American Academy of Ophthalmology. Ophthalmic Technology Assessment Committee 2001-2002 Glaucoma Panel. Automated perimetry: a report by the American Academy of Ophthalmology. Ophthalmology 2002;109:2362-2374.

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