CET CONTINUING EDUCATION & TRAINING

THIS ISSUE CET: FREE ✔

Approved for Optometrists ✔ Approved for DOs ✔

To gain more standard CET points for this year's PAYL series, purchase your credits at www.otbookshop.co.uk and then enter online at: www.otcet.co.uk to take your exams

Sponsored by: ACADEMY FOR EYECARE EXCELLENCE. CIBA VISION

Module 12 Part 9: Vision in the aged

COURSE CODE: C-11924

Profiling Glaucoma

Aachal Kotecha PhD, Alexander Spratt MRCOphth

Glaucoma is the leading cause of irreversible blindness worldwide[1], yet the majority of patients with glaucoma are unaware that they have the disease. It has been predicted that with our ageing population, the number of people in England and Wales suffering from glaucoma will increase by at least a third over the next two decades. [2,3]

The purpose of this article is to provide a brief overview of the pathophysiology behind primary open angle glaucoma (POAG) and the clinical signs that are indicative of the disease. Finally, the impact of glaucoma on patient quality of life will be considered.

Glaucoma – definitions

The glaucomas encompass a group of diseases that result in a progressive optic neuropathy that cause characteristic changes in the optic nerve head (ONH) and retinal nerve fibre layer. The disease can be classified as primary or secondary and as 'open' or 'closed' angle. Primary glaucoma exists in the absence of any underlying ocular or medical condition and secondary glaucoma is that which develops as a consequence of a discernable ocular or medical co-morbidity.[4] In open angle glaucoma, there is no obvious physical occlusion of the drainage angle, unlike closed angle glaucoma, where the peripheral iris causes a significant obstruction to aqueous outflow (Figure 1). The majority of glaucoma cases that present to the optometrist tend to be of the POAG variety.

The prevalence of POAG has been estimated to be approximately 2% in the Caucasian population aged over 40 years and 8% in the Afro-Caribbean population aged over 40 years and the disease increases in prevalence with advancing age.[5,6,7,8]

The prevalence of primary angle closure glaucoma (PACG) is less common in Caucasians, but it is a major cause of bilateral blindness worldwide.[3] PACG is defined as a glaucomatous optic neuropathy with a characteristic visual field (VF) defect in the presence of a narrow or closed anterior chamber angle (ACA). In instances where the ACA is obstructed causing a concomitant rise in IOP but in the absence of optic neuropathy, the term primary angle closure (PAC) is used.

Pathogenesis of primary open angle glacuoma

Normal aqueous production and changes with age

The role of the aqueous humour in the anterior chamber is threefold: it provides nutrients to the crystalline lens and corneal endothelium, removes metabolic waste by-products and maintains an intraocular pressure (IOP) to keep the eye inflated.

Aqueous humour is derived from the blood plasma of the capillaries within the ciliary processes of the ciliary body. The majority of aqueous is produced via active secretion by the pigmented-non pigmented epithelial syncitiuum that line the ciliary processes, although a small proportion is also produced via ultrafiltration and diffusion. In the normal human eye, the rate of aqueous production is estimated to be 2µl per minute. The rate of production varies diurnally, with much less being produced at night, and also reduces with age, with a reported 2.4% reduction per decade.[10]

Following production, the primary aqueous humour enters the posterior chamber, behind the iris and in front of the crystalline lens, and passes through the pupil into the anterior chamber where it circulates prior to exiting the internal eye via the ACA.

Aqueous exits the internal eye via the trabecular meshwork and Schlemm's canal, often referred to as the 'conventional' outflow pathway (Figure 2). The trabecular meshwork comprises a series of interlacing collagen beams, with inter-beam spaces that distinctively change as one goes deeper into the structure. The uveal meshwork, the area facing the anterior chamber, has the largest inter-beam spaces (or 'pores') and offers the least resistance to aqueous outflow. Next is the corneoscleral meshwork which has progressively smaller trabecular pores. Adjacent to this lies the juxtacanalicular connective tissue (JCT). This area is not formed of trabecular beams but comprises a loose connective tissue

containing trabecular meshwork cells lying within a fibrillar extracellular matrix (ECM) and a ground substance filled 'space'. The JCT abuts the inner wall endothelium of Schlemm's canal. It is believed that the greatest resistance to aqueous outflow lies in this JCT/Schlemm's canal inner wall endothelial area. The inner wall endothelium lies on an incomplete basal lamina and therefore in places has direct contact with the JCT. Aqueous humour pushes on the basal side of these cells forming 'giant vacuoles' that allow aqueous to percolate out of the JCT into Schlemm's canal. These cellular outpouchings form as a direct result of the pressure gradient of aqueous flow and have been associated with micro-sized intracellular pores present in the endothelium.[11]

A proportion of aqueous also leaves the internal eye via the interstitial spaces of the ciliary muscle and the uvea, referred to as the supraciliary space. In the past it was believed that this 'unconventional' uveoscleral pathway accounted for only 10% of aqueous outflow from the eye, with 90% leaving via the trabecular meshwork. However, following the arrival of topical prostaglandin analogues as medical therapies for lowering IOP and the dramatic effects they have on IOP reduction, research now suggests that in a healthy human adult, uveoscleral outflow accounts for between 25% and 50% of total aqueous outflow.[12]

In the ageing eye, there is increased resistance to aqueous outflow via both the trabecular pathway and uveoscleral pathway. This may be as a result of an increase in trabecular beam thickness (and possibly stiffness) and an increase in extracellular material within the supraciliary space in the aged eye. The increased prevalence of POAG with advancing age might be due to an exacerbation of these 'normal' ageing changes compounded by the microstructural trabecular meshwork changes caused by the disease.[11,12]

Site of damage: mechanical vs vascular theories

The increased resistance to aqueous outflow that occurs in glaucoma results in an increase in IOP. The primary site of axonal injury from raised IOP is at the level of the ONH, the posterior scleral opening through which retinal ganglion cell (RGC) axons exit the eye.[13]

In the human eye, RGC axons pass through a structure called the lamina cribrosa, a complex three-dimensional lattice of connective tissue beams and supportive glial tissue. The structure has an abundant intra-laminar vasculature that provides nutrition to the RGC axons as they pass through. However, the axons do not receive nutrients directly from the vessels; rather, nutrients diffuse out from the capillary walls, pass through the lamina beam basement membrane and through the astrocytes that cover the axons prior to reaching their destination.

Historically speaking, two mechanisms of glaucomatous optic neuropathy have been described; the 'mechanical' and the 'vascular' theory. In the former, it is believed that raised IOP causes a posterior deformation of the lamina cribrosa inducing shearing forces that result in a strangulation of RGC axons as they pass through. In the vascular theory, it is thought that poor ONH capillary perfusion leads to axonal death through lack of nutrition. That this occurs in the presence of normal IOP, and is as a direct result of vascular deregulation rather than a mechanical IOP effect, offers a rationale for the existence of 'normal tension (pressure)' glaucoma. However, there is much debate regarding the distinction between these two mechanisms of glaucomatous damage and emerging evidence suggests that there is a complex interaction between these two 'effects' that results in glaucomatous optic neuropathy.

A new paradigm for the ONH being a biomechanical structure is currently being explored. In the normal human eye, the predominantly collagenous corneoscleral shell is able to 'absorb' and compensate for the normal physiological changes in IOP. It is thought that in eyes that are predisposed to glaucoma, the IOP-related stress and strain forces on the globe gradually change the lamina cribrosa beam collagen biomechanics. These changes ultimately result in a 'stiffening' of the connective tissue around the beams and surrounding blood vasculature. As a result, the stiffer lamina cribrosa beams become less compliant to alterations in IOP, making the area (and subsequently the RGC axons) more susceptible to damage. Furthermore, the stiffer connective tissue around the intra-laminar blood vasculature and laminar beams will result in greater resistance to the diffusion of nutrients to axons which also leads to their damage.[14]

Previous research has suggested that there is a geographical variation in lamina cribrosa pore size, with pores at the superior and inferior ONH being larger than those in the nasal and temporal areas.[15] This would lend itself to the theory that RGC axonal bundles passing through the superior and inferior areas of the ONH would be more susceptible to damage from the shearing forces in instances of raised IOP. And this is supported by the finding that RGC axonal loss at the poles of the ONH is often the first sign of glaucomatous damage. However, other studies have shown a great inter-individual variability in lamina cribrosa pore size distribution that does not always follow the 'rule' described above. Another possible reason for the apparent geographical selectivity of axonal damage may be due to the oval shape of the ONH. Computerised finite element modelling work suggests that the distribution of IOP-related stress and strain is more uniform around a circular scleral canal opening, but appears to concentrate around the poles in an oval canal opening. Perhaps this might explain the preferential damage

Figure 1
Acute angle closure glaucoma

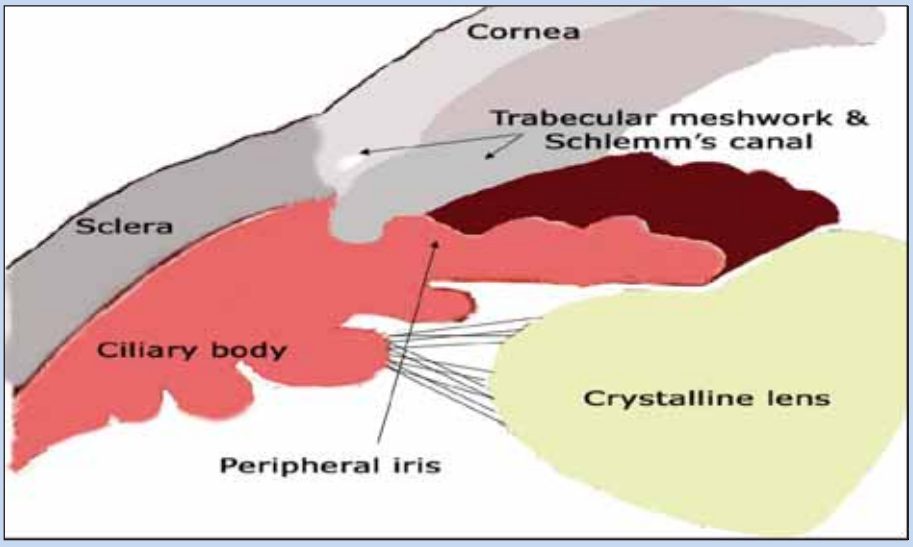


Figure 2
Schematic diagram showing anatomy of anterior chamber angle

of axons in the superior and inferior ONH areas. However, this is purely speculative and is the subject of further research.

Detection of glaucoma

Traditionally, a triad of tests are employed to detect the presence of glaucoma: optic disc assessment, VF testing and IOP measurement. The following provides a succinct overview of the clinical signs that should alert the practitioner to the presence of primary open angle glaucoma. However, the reader is directed to a more comprehensive review presented by the author in the College of Optometrists' quarterly publication *Optometry in Practice* published in June this year.

Optic disc assessment

It has been estimated that the loss of approximately 30% of ganglion cell axons is required prior to the detection of a defect in the VF using standardised automated white-on-white perimetry.[16] Therefore, a careful examination of the optic disc is required to detect the presence of glaucoma. Ideally, this should be done with binocular indirect ophthalmoscopy through a dilated pupil.

The optic disc is usually vertically oval and in Caucasians the average vertical by horizontal diameter is approximately 1.8mm x 1.7mm. However, optic disc size varies with ethnicity and Afro-Caribbeans have considerably larger discs. Disc size is only affected by refractive error greater than +5.00D ametropia, being significantly larger and smaller in highly myopic and highly hypermetropic eyes, respectively.[17]

Most optometrists will be familiar with the Jonas inferior-superior-nasal-temporal (ISNT) rule, which refers to the width of the neuroretinal rim tissue (NRR) in four quadrants of the optic disc. In the average eye, the NRR is widest inferiorly, followed by superiorly, then nasally, with the narrowest NRR usually found at the temporal disc area.[18] Glaucoma results in damage to RGC axons which manifests as a loss of NRR in the optic disc. Usually in glaucoma, damage initially occurs at the inferior and superior poles of the disc and thus in glaucomatous eyes the ISNT rule is no longer followed (Figures 3a and b).

However, it is important to remember that there are a relatively finite number of RGC axons in each human eye and that their distribution around the disc will vary according to disc size. Thus, the ISNT rule cannot be applied in eyes with very small (< 1.5mm) or large (> 2.2mm) optic discs, and the rule is quite impossible to interpret in tilted optic discs. Very small discs may exhibit little cupping if at all, whilst very large discs will have large cupping and an even distribution of NRR that does not vary in width around the disc. Therefore, it is difficult to detect progressive NRR loss at extremes of disc size. Very small discs require a significant loss of retinal nerve fibres before cupping presents itself. Conversely, large optic discs may appear to show little change in the NRR distribution in the presence of progressive glaucoma.

In cases where the ISNT rule is difficult to ascertain, other clues to the presence of acquired NRR loss should be sought. In progressive disease, the configuration of blood vessels within the optic disc area may change. Branch vessels that run along the surface of the optic disc may start to lose their support with progressive NRR loss, appearing to be 'suspended in mid-air'. This is often termed the 'baring of blood vessels'. There is usually an inter-ocular symmetry in optic disc size; therefore a difference in vertical cup-to-disc ratio of 0.2 or greater should arouse suspicions. Finally, the presence of disc haemorrhages should also alert the optometrist to the presence of an abnormality. Although disc haemorrhages are not necessarily pathognomonic of the disease – the prevalence of disc haemorrhages in the normal population has been estimated at 0.2% – they are much more common in glaucoma.[19] Disc haemorrhages are precursors to retinal nerve fibre layer defects, although they tend to resolve within a matter of weeks. It is therefore wise to routinely refer a patient presenting with a disc haemorrhage to an ophthalmologist, even if IOP and VF testing show no abnormality.

Visual field assessment

The VF test usually measures the differential light sensitivity of the retina; that is, its ability to perceive a difference in brightness between a test target and the background upon which it is presented. In glaucoma, there is progressive damage to the RGC axons that results in characteristic changes in differential light sensitivity across the retina (Figure 4). There is a 'structure-function' concordance in glaucoma, such that axonal loss in the inferior optic disc will produce a superior VF

defect, and so on. Patterns of VF loss in glaucoma include an enlargement of the blind spot, small paracentral defects 10 to 20 degrees from fixation that eventually coalesce to produce an arcuate defect, a nasal step (which refers to a difference in retinal sensitivity either side of the temporal retinal horizontal midline), a temporal wedge of vision, and finally, in end-stage disease, a small central island of vision. However, there are no hard and fast rules as to how a VF defect caused by glaucoma will manifest; it is, however, important that if a VF defect is found, it is reproducible and appears to have structural concordance.

It is good practice to repeat a VF test if a defect is found in order to avoid an inappropriate referral. There is a significant 'learning effect' found in patients performing VF tests; patient test performance improves with practice. Furthermore, it has been shown that defects present in a single VF test often disappear on subsequent testing, with reports of up to 80% of patients losing their VF defect on repeat testing.[20]

IOP measurement

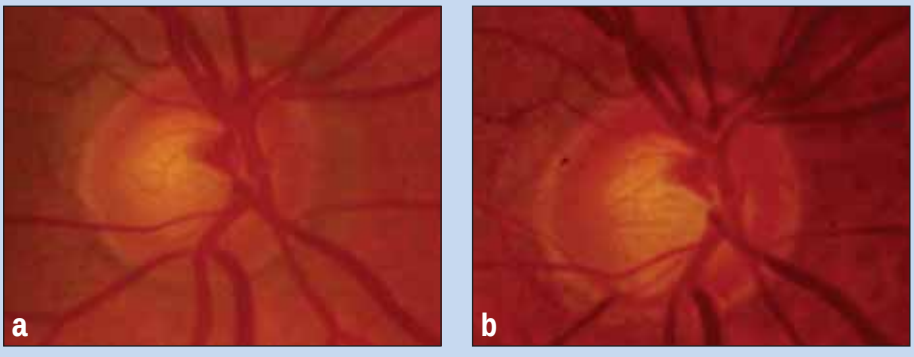
Raised IOP is the most important modifiable risk factor for the development and progression of glaucoma. In the normal (i.e. non-glaucomatous) Caucasian population, the average measured IOP has been reported to be 15.5mmHg, with a standard deviation of 2.5mmHg. However, IOP is not normally distributed and is skewed slightly towards the higher pressures.

IOP varies in a circadian fashion, usually being highest in the morning and decreasing over the course of the day. In normal eyes, the diurnal fluctuation has been reported to be between 3 and 5mmHg, with untreated glaucomatous patients displaying significantly greater fluctuations.[21]

There are a number of devices available to measure IOP, but the slit-lamp mounted Goldmann applanation tonometer remains the reference standard by which all new tonometers are compared. The device was first introduced around 1957 and it is based on the Imbert-Fick principle, which states that for a fluid-filled encapsulated sphere, the pressure of the fluid within the sphere is proportional to the force required to applanate an external area of the sphere. The principle holds true for a perfectly spherical, dry, elastic and infinitely thin encapsulating surface. The designers of the Goldmann tonometer, Hans Goldmann and Theo Schmidt, were well aware that the cornea did not satisfy any of these requirements and made calculations to compensate for the finite thickness and rigidity of the cornea as well as for the capillary action of the tear meniscus drawing the tonometer to the corneal surface. In spite of this, the cornea is a major obstacle to IOP measurement accuracy. Variations in corneal thickness, curvature and rigidity all contribute to the measurement error of all tonometers. Until recently, most of the literature on IOP measurement accuracy concentrated on the effects of variations in central corneal thickness, with studies showing that thin corneas cause an under-estimation of IOP whilst thick corneas cause an over-estimation of IOP. Variations in central corneal curvature also affect the accuracy of IOP measurements, but to a much lesser degree than central corneal thickness. Steep corneas require more force to applanate, thus resulting in an over-estimation of IOP, and the converse is true for flat corneas. However, studies modelling the effects of the cornea on IOP measurement accuracy have shown that variations in corneal thickness and curvature do not account for all the variability in measured IOP.

Furthermore, the effects of these two parameters on IOP measurement accuracy is not linear and is very dependent on other corneal properties. It has been shown that two corneas of the same thickness and curvature will induce very different IOP measurement errors depending on their rigidity. A patient with a thick yet 'soft' cornea will have an under-estimated IOP measurement, compared with one who has a 'stiffer' cornea of the same thickness. Therefore, it is strongly advised that linear nomograms that attempt to 'correct' for the central corneal thickness effect on IOP measurement be avoided. If corneal pachymetry is performed along with IOP measurement, it is much better practice to consider whether the IOP measurement is relatively over- or under-estimated, rather than trying to calculate an absolute (and incorrect) IOP value using a nomogram. Research is currently underway to establish the validity of a new in vivo measurement of corneal biomechanics that may help characterise the corneal effect on IOP measurement in a more complete way, making for better IOP measurement correction algorithms.

It is generally accepted in optometric practice to repeat IOP measurements at different times of the day should an elevated IOP reading be found at first testing. It is also important to note that IOP may be artefactually elevated (eg from breath hold or tight neck ties) and so it may be worth repeating IOP measures at the same time to be sure to eliminate spurious findings.



Figures 3a and 3b
Progressive glaucoma. Optic disc images taken seven years apart. Notice the loss of neuroretinal rim from image 'a' to 'b'. Image 'b' clearly shows an inferior notch (with corresponding change in blood vessel configuration) and loss of the inferonasal and nasal neuroretinal rim tissue.

National treatment guidelines

The recent development of guidelines for the diagnosis and management of adult chronic open angle glaucoma and ocular hypertension by the National Institute for Health and Clinical Excellence (NICE) should standardise high quality care for these patients and prevent more people from going blind. Community based optometrists, as the main source of glaucoma referrals to hospital eye services, should be familiar with these guidelines which are available at www.nice.org.uk.

The guidelines provide definitions that may prompt specialist referral and stipulate what tests and expertise are needed to effectively diagnose and monitor these patients. The definitions rely largely on consideration of patients' optic discs, VF and IOP, as discussed in this article. Ocular hypertension, with healthy optic discs and VF, should only be suspected if IOP exceeds 21mmHg when measured by slitlamp mounted Goldmann applanation tonometry on more than one occasion.

Definitive diagnosis needs gonioscopy and measurement of central corneal thickness extending the required competency of practitioners beyond the minimum level currently needed for entry to the optometric profession. However, the College of Optometrists' core competencies (including Goldmann applanation tonometry) are sufficient to allow optometrists to take responsibility for ongoing monitoring of patients with confirmed diagnoses and established management plans, without the need for additional training.

The impact of glaucoma on the individual

In the last decade, a large proportion of the research within ophthalmology has focussed on the technological development of sophisticated imaging devices that give high resolution images of retinal integrity and psychophysical tests that assess visual status. However, whilst these tests give vital information to aid diagnoses and evaluate the success of disease management, they are clinician-based measures and interpretations of patient disease status.

Compared with clinicians, patients place importance on different outcomes of their disease diagnosis and treatment, specifically how their vision interacts with other conditions and whether they will be able to lead a personally satisfying life. In recognition of this, as part of their evaluation of new healthcare technology, NICE requires evidence from both patients and carers of the impact on the patient's disability, function, quality of life and lifestyle. It states that "Patient evidence can identify the limitations in the published research literature - in particular, the failure to capture the true concerns of individual patients related to quality of life over and above measurements using standardised instruments (such as questionnaires) developed using psychometric techniques'.[22] NICE uses patient quality of life (QoL) measures to assess both the effects and cost-effectiveness of new treatments.

The QoL impact of disease and treatment assessed using questionnaires is sometimes referred to as 'patient reported outcomes'. These require a patient's self-evaluation of their general well-being and their ability to perform day-to-day tasks. Vision-specific questionnaires require patients to self-evaluate their visual disabilities. Studies have assessed the correlation between patient-perceived visual disability and clinical measures of glaucoma with mixed results: some authors have found modest associations between perceived visual disability and the severity of glaucoma whilst others have found only weak correlations.[23,24,25] It is also clear that individuals with similar levels of disease severity report very different QoL experiences.[26] Thus, whilst QoL questionnaires and other subjective instruments may provide interesting adjuncts to the assessment of our patients' functional vision, more objective methods of assessment are required to formally examine how well they perform visually demanding tasks that are relevant to their everyday life experiences.

Glaucoma and risk of falls

It has been shown that a reduced VF is a significant predictor of mobility performance and risk of falling.[27,28,29,30] Postural stability is maintained through a complex interaction of vestibular, somatosensory and visual inputs. It has been shown that visual impairment is a major risk factor for falls in the elderly and the association may in part be explained by failure to see ground-level objects, thereby causing the individual to trip over. However, studies examining postural sway in a group of glaucoma patients have found that a decrease in VF sensitivity is associated with a decrease in postural steadiness and an increase in body sway. These studies suggest that glaucoma is associated with an increased risk of falling.

IOP lowering therapies

As has been previously mentioned, raised IOP is the only modifiable risk factor for glaucoma development and progression and thus IOP reduction is the mainstay of glaucoma treatment. Whilst a discussion of the topical hypotensive treatments available to treat glaucoma falls outside the remit of this article, a special cautionary note should be made regarding some of the adverse effects of glaucoma therapy on the older individual.

Although less widely used today, miotic therapy has been associated with an increased risk of falling. This is likely to be due to the induced restriction in VF caused by pupillary constriction compounding the effects of VF loss from the disease itself.

The use of beta-blocker eye drops have been associated with many cardiovascular side-effects, including syncope, arrhythmia and, more seriously, myocardial infarction. The use of topical beta-blocker therapy has also been found to be the greatest risk factor for serious falls in glaucoma patients aged 65 years or older, even when taking into consideration the degree of glaucomatous VF defect.[31]

Topical beta-blockers are absorbed into the systemic blood system after passage through the nasolacrimal system through both the nasal mucosa and by pulmonary absorption (the

latter being from the inhalation of drug particles). This results in a high concentration of drug in the system, as this method of drug absorption avoids hepatic first-pass metabolism. If the patient is also on systemic beta-blockers for hypertension, the impact of this 'double dose' could be disastrous.

If a glaucoma patient reports a history of falls, it is probably worth making note of their current topical and systemic medication to examine any medication associations and, if necessary, report them to the GP. It is also important to note that many new combination topical therapies for the treatment of glaucoma contain a beta-blocker (0.5% timolol), but no clue to their content is given in their name. Examples of such drugs include Combigan, Xalacom, Cosopt, Duotrav and Ganfort.

Glaucoma and driving

Driving safety in patients with glaucoma has been studied extensively with mixed results. A retrospective cohort study evaluating the relationship between glaucoma and risk of motor vehicle crash (MVC) involvement found that older drivers with glaucoma were up to 50% less likely to be involved in a MVC. This is in stark contrast to another study which found that people with glaucoma were six times more likely to have been involved in a MVC compared with non-glaucomatous controls.[32] The differences may reflect the effects of self-imposed driving restrictions amongst drivers with known glaucoma. Individuals who perceive that they have a visual impairment are likely to reduce their exposure to 'difficult' driving situations.[33,34] However, studies using driving simulators looking at the driving performance of patients with peripheral VF loss caused by glaucoma have found a strong correlation between the horizontal extent of VF and the number of virtual MVCs experienced. It has been suggested that a VF reduction to less than 100 degrees horizontally would put an individual at a greater risk of having an accident.[35] Most studies suggest that the loss of VF is an important and valid predictor for determining driving safety and the likelihood of involvement in MVCs.

New research within the Measurement Techniques in Vision laboratory at the Department of Optometry, City University, London, is currently investigating eye movements in the visual search tasks of patients with glaucoma. Recent work found that eye movement patterns in patients with bilateral glaucoma differed significantly from healthy controls when viewing a moving road and traffic scene presented on a computer screen.[36] In some patients, eye movement patterns appeared to be directly related to the extent of VF loss with patients making more saccades, suggesting a possible compensatory response to their scotoma.

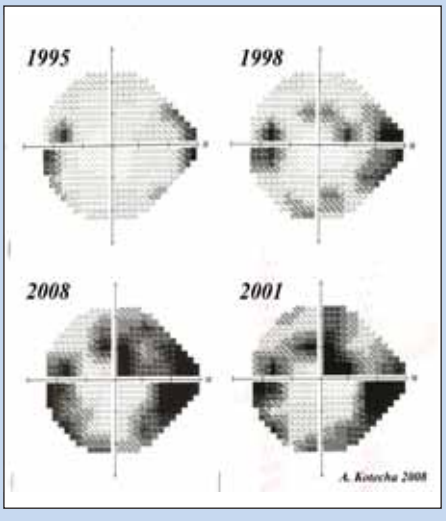


Figure 4
Progressive visual field loss in glaucoma. Images show the greyscale plot from a Humphrey Visual Field analyser. The patient presented to the hospital eye service with a nasal defect in 1995. Despite maximally tolerated treatment, the patient showed a deepening and progression of his visual defect over a ten year period.

Glaucoma and tasks of everyday living

In another recent study at the Department of Optometry at City University, the ability of glaucoma patients to reach out and grasp a table-top object was compared to a control group of subjects without the disease. The study found that glaucoma patients exhibited delays in movement planning and initiation and made slower, more tentative reaches to table-top objects, particularly those placed at far distances, compared with normal control subjects. This was found even in subjects with relatively minor binocular overlapping VF defects and suggests that glaucoma patients with relatively 'minor' disease may have difficulties with the tasks of everyday living. However, this is the subject of further investigation.

Conclusion

This article has provided a brief overview of the pathophysiology behind POAG and the clinical signs that may present. Having explored the impact of glaucoma on the tasks of daily living, it is clear that not only individuals with 'end-stage' disease have difficulties but also those with relatively 'early' disease.

About the author

Dr Aachal Kotecha is a senior lecturer at the Department of Optometry and Visual Science, City University, London. She is also a senior research associate for NIHR Biomedical Research for Ophthalmology, Moorfields Eye Hospital NHS Foundation Trust and UCL Institute of Ophthalmology, London.

Dr Alexander Spratt is an ophthalmologist at Moorfields Eye Hospital, London and visiting lecturer at the Department of Optometry and Visual Science, City University, London.

Acknowledgements

Dr Kotecha receives a proportion of her funding from the Department of Health's National Institute for Health Research Biomedical Research Centre for Ophthalmology at Moorfields Eye Hospital NHS Foundation Trust and the UCL Institute of Ophthalmology. The views expressed in this publication are those of the authors and not necessarily those of the Department of Health.

References

See www.optometry.co.uk/references

Module questions

Course code: C-11924

Please note, there is only one correct answer. Enter online or by the form provided

An answer return form is included in this issue. It should be completed and returned to CET initiatives (C-11924) *OT*, Ten Alps plc, 9 Savoy Street, London WC2E 7HR by October 14 2009

1. Which one of the following statements is FALSE?
a. glaucoma can occur even when there is no obvious occlusion of the trabecular meshwork by the peripheral iris
b. the glaucomas encompass a group of diseases that result in a progressive optic neuropathy
c. glaucoma may be an asymptomatic condition
d. secondary glaucoma exists in the absence of any underlying condition

2. What is the estimated prevalence of primary open angle glaucoma in the Caucasian population?
a. 10%
b. 8%
c. 15%
d. 2%

3. Which one of the following statements regarding aqueous humour dynamics is TRUE?
a. the majority of aqueous is produced in the evening
b. aqueous is derived predominantly through diffusion
c. aqueous production increases with advancing age
d. in some individuals, aqueous outflow may be evenly distributed between the trabecular meshwork and the uveoscleral pathway

4. Where is the site of most resistance to aqueous outflow in POAG?
a. the juxtacanalicular meshwork/Schlemm's canal inner wall endothelial area
b. the pigmented-non pigmented ciliary epithelium syncitiuum
c. the uveal meshwork
d. the corneoscleral meshwork

5. Which one of the following is NOT a likely mechanism of retinal ganglion cell axonal damage in POAG?
a. a stiffening of the lamina cribrosa making it less compliant to alterations in IOP
b. a reduction in diffusion of nutrients available to the retinal ganglion cells
c. a mechanical compression of retinal ganglion cell axons
d. a stretching of the lamina cribosa and an increase in diffusion of nutrients

6. Which one of following statements is FALSE?
a. most optic discs are slightly vertically oval
b. an optic disc of <1.5 mm vertical diameter is considered very small
c. the size of the cup is not related to the size of the disc
d. optic disc size is greater in some racial groups

7. Which one of the following statements is FALSE?
a. in early glaucoma, neuroretinal rim damage usually occurs at the superior and inferior poles of the disc
b. myopic eyes have smaller discs compared to normal
c. the ISNT rule is difficult to assess in tilted discs
d. in large discs, the ISNT rule cannot be applied

8. Patterns of visual field loss in glaucoma do NOT usually include:
a. a nasal step
b. baring of the blind spot
c. paracentral scotomas
d. a dense central scotoma

9. Which of the following statements is FALSE?
a. the prevalence of glaucoma is greater at higher IOPs
b. the distribution of IOP in the normal population is skewed towards the lower pressures
c. the diurnal variation in IOP in a normal individual is usually between 3 and 5mmHg
d. IOP is highest upon awakening

10. An individual's IOP reading will be under-estimated if:
a. they have a thick cornea
b. they hold their breath
c. they have a flat cornea
d. they have a stiff cornea

11. What has NOT been shown to be a significant risk factor for falls in the elderly glaucoma patient?
a. a decrease in the visual field
b. topical beta-blocker therapy
c. miotic therapy
d. a trabeculectomy

12. Which one of the following statements regarding the impact of glaucoma on the individual is TRUE?
a. a decrease in the retinal differential light sensitivity will lead to an decrease in body sway
b. only patients with advanced glaucoma have difficulties in undertaking the reaching and grasping of table top objects
c. a horizontal visual field reduction to less than 100 degrees will have no impact on the driving safety of the individual
d. glaucoma patients with 'minor' disease may show delays in movement planning and initiation

Please complete online by midnight on October 14 2009 - You will be unable to submit exams after this date – answers to the module will be published on www.optometry.co.uk

THIS ISSUE CET: FREE ✔

Approved for Optometrists ✔ Approved for DOs ✘

To gain more standard CET points for this year's PAYL series, purchase your credits at www.otbookshop.co.uk and then enter online at: www.otcet.co.uk to take your exams