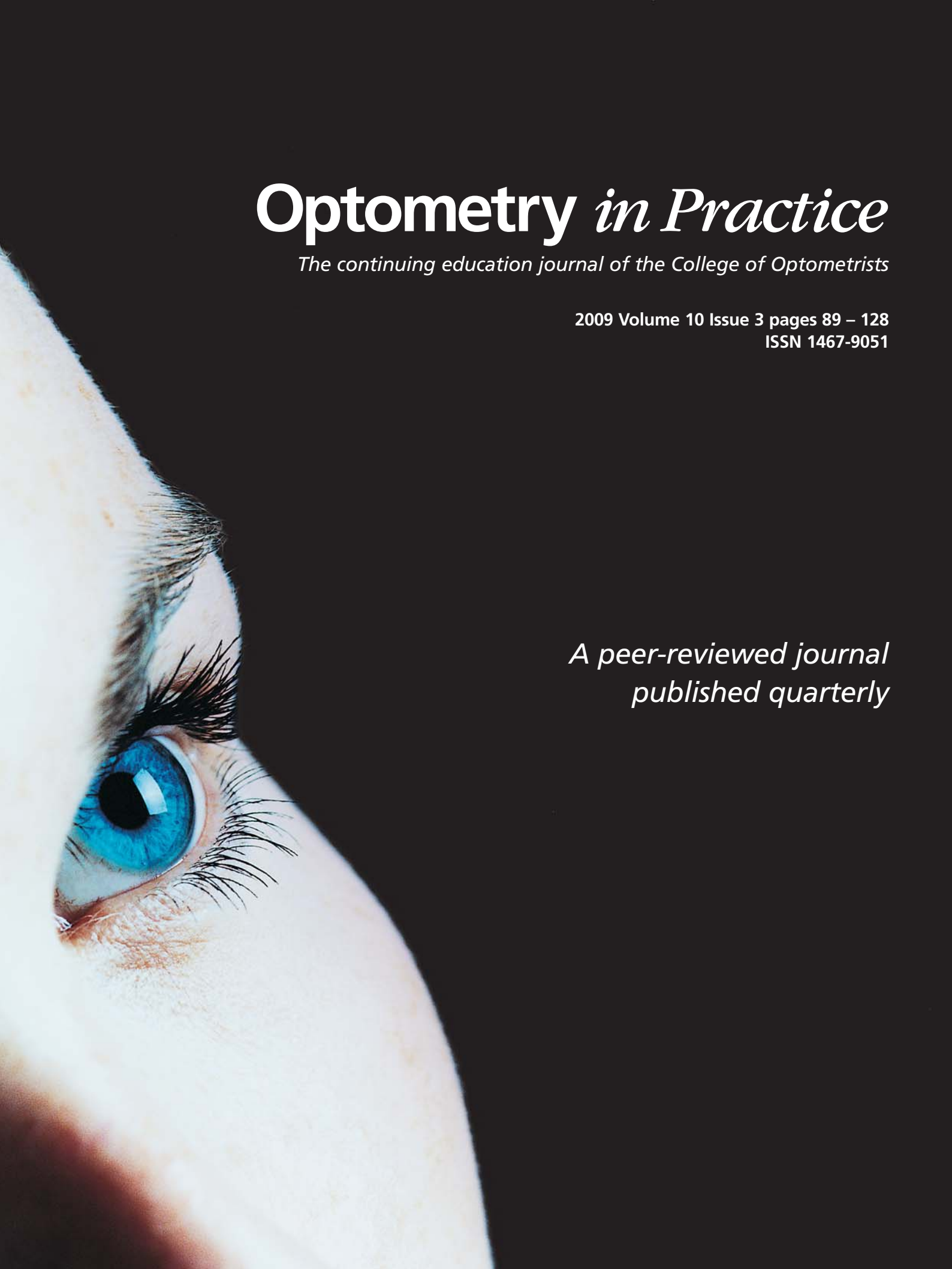

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Important CET information

2009 brings us to the end of the current continuing education and training (CET) cycle. To help those College members who may have fallen behind with their CET, or may be returning after a period of absence from the register, the *Optometry in Practice* editorial board has compiled a selection of 'favourite' papers from recent volumes of the journal, which retain their currency and relevance. These have been re-accredited for CET and are available online to members via the College website. We hope you will find this additional service of value. The multiple choice questions for these papers will remain open until the middle of December 2009.

Information for Authors

A Guide for Authors is available from the publishing office (see page i).

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Pass mark 60%. CET points available: papers with 21 MCQs 4 points, 15 MCQs 3 points, 12 MCQs 2 points, 6 MCQs 1 point.

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THE COLLEGE OF OPTOMETRISTS

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Editorial

Professor ST Parrish BSc PhD FCOptom MIET FHEA

Visiting Professor, Anglia Ruskin University, Cambridge and City University, London, College of Optometrists Examiner and Assessor

The term 'visual performance' can be widely used in ophthalmic literature to refer to any one of the plethora of different functions of the visual system. From an optometric perspective, we often think of visual performance in relation to measurement of visual acuity or visual fields; however it can relate to a wide and diverse range of parameters far in excess of those used routinely in practice.

All four papers in this edition of *Optometry in Practice* discuss different aspects of visual performance. Pointer looks at the relationship between visual acuity measurement and subjective assessment of visual satisfaction. Denniss and Henson look at the relationship in glaucoma between changes in visual function and structural changes in the eye, and Heath looks at modern medical treatments for macular hole and their effect on visual outcomes. He also mentions the limitations of pre- and posttreatment visual acuities as a measure of outcomes and references the use of patient visual function questionnaires as an additional assessment tool. Nourrit and Kelly discuss causes of light scatter in the eye and consider the clinical effect on vision. All these papers explore aspects of visual performance in different ways and have an obvious relevance to clinical practice. I hope you find them both enjoyable and useful and they provide a total of 10 continuing education and training (CET) points.

A further 12 CET points are available from the 'highlight papers' which have been selected by the Editorial Board from the last two volumes of *Optometry in Practice*. These can be accessed via the College website and details are provided inside the front cover. They provide the opportunity to obtain additional CET points before the end of the current CET cycle, which finishes at the end of this year.

Letter to the Editor

Sir

First we must compliment Aachal Kotecha on a timely paper, Detection of Glaucoma by the Primary Care Optometrist (*Optometry in Practice* 2009; 10: 51–64). However, we do feel that the paper contains a perpetuation of the inference that optometrist referrals into the hospital eye service (HES) for suspect glaucoma have a high false-positive rate because of some innate problem arising in optometric practice or amongst optometrists. Kotecha cites one paper on this subject in her second paragraph (Bowling *et al.* 2005) and further concludes the same later on but cites no other references.

It is clear that the vast majority of referrals to the HES for suspect chronic open-angle glaucoma (COAG) are initiated by optometrists. Therefore, if there is a high incidence of false-positive referral, logic infers that this must be because of something optometrists are doing incorrectly or perhaps not at all. Chief amongst suspects remains the preponderance of non-contact tonometry methods in optometric practice. Perhaps in this case the received wisdom is based on flawed logic.

Ten years ago the present authors, Rumney and Henson (1999) pointed out that the high false-positive referral rate by optometrists is at the very least an artefact of one principal issue, namely the low prevalence of glaucoma. Kotecha herself cites the prevalence of COAG as being 2% of the over-40s.

Suppose an optometrist saw 10 000 patients over the age of 40 years, and suppose we assume the incidence of COAG amongst this group to be 2%. If this optometrist had at his disposal a screening test that conferred hitherto unheard-of levels of 99% sensitivity and 99% specificity, the results would be as follows. A test that is 99% sensitive would detect 198 of the 200 glaucoma cases (true positive) and regrettably miss 2 (false negative). Similarly, a test that was 99% specific would show normal findings for 9702 patients (true negative) but would fail 98 normals (1% of 9800) as abnormal (false positive). The resultant false-positive referral rate would be 98/198 (49.5%), which is not enormously different from the typical figures quoted in many papers. Given that it is commonly stated that 50% of cases of glaucoma are already detected, the numbers requiring such opportunistic case detection may be significantly fewer than these assumed figures, which would lead to an even higher false-positive ratio.

In our letter to the editor 10 years ago we concluded:

> Fundamentally, the difficulty lies with glaucoma being a disease of relatively low prevalence that is difficult to diagnose unequivocally at a stage of minimal optic nerve head damage. Put alongside this the structure of the legislation circumscribing optometric practice and it can be seen that this mitigates directly counter to the clear need for more extensive and repeat testing prior to referral. In addition, the lack of nationally agreed referral criteria and good interprofessional communications including feedback make it difficult to see how any improvements are going to be made within the current system. Our professions need to work closer together so that optometrists can achieve acceptable false-positive referral rates.

Having said all that and correctly identified that optometrists actually do rather well under the circumstances, things could be better. Optometrists can use Goldmann or other applanation tonometry and furthermore, let us repeat it, they can undertake threshold-related fields assessment and repeat when required and they can undertake dilated disc assessments using slit-lamp binocular indirect ophthalmoscopy. That in the main they do not has very little to do with competence or diagnostic skill and everything to do with the General Ophthalmic Services (GOS) sight test. The current

primary eye care contract is simply unfit for purpose, being a refraction-based test of sight with an eye health check conducted at the same time and once only. For an optometrist to undertake a proper assessment of glaucoma risk before referring requires at least two and possibly three visits. This simply cannot be done under the current GOS contract unless the optometrist charges the patient for the time, subsidises the time spent via other practice economic activity or simply refers at the first indication (perpetuating the high false-positive argument).

In order for the new National Institute for Health and Clinical Excellence (NICE) guidance on ocular hypertension and COAG to work efficiently, some check on demand into the HES has to occur to reduce the false-positive rates. It is therefore essential that primary care trusts (or preferably the Department of Health nationally) commission glaucoma case detection refinement schemes, of which there are several good examples, as soon as possible.

Yours sincerely

Nicholas Rumney (BBR Optometry Ltd, Hereford) and David Henson (Department of Ophthalmology, University of Manchester)

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Reply to Letter to the Editor

Dear Sir

I would like to thank Mr Nicholas Rumney and Professor David Henson for their letter regarding the role of optometrists in case-finding glaucoma in the community. The thrust of my article was directed towards the importance of the careful interpretation of results from the triad of tests traditionally used to detect glaucoma and the avoidance of referring based on the results of one test alone. In light of the new National Institute for Health and Clinical Excellence (NICE) guideline and advice from the Association of Optometrists, this 'ideal' practice is probably no longer always tenable; however, Rumney and Henson raise some interesting points.

There is no escaping the fact that there is a high false-positive referral rate of glaucoma suspects to the hospital eye service (HES). Prior to the implementation of the new NICE guidelines, it had been shown that approximately 40% of new suspect-glaucoma referrals were discharged following their visit to the HES (Bell and O'Brien 1997; Patel *et al.* 2006; Theodossiades and Murdoch 1999; Vernon and Ghosh 2001). In a study undertaken by Spry and Diamond in 2000 which examined the accuracy of 1085 community optometrist suspect-glaucoma referrals to Bristol Eye Hospital, 44% of the patients referred on the basis of abnormal visual fields alone were subsequently found to be normal (personal communication PG Spry, June 2009).

I agree that the relatively low prevalence of glaucoma in the Caucasian population and the absence of the perfect screening test to detect its presence make case-finding difficult. I also agree that the problem of the high false-positive referral rate does not lie in the ability of the optometrist and they are trained in the clinical skills of applanation tonometry, visual field examination and binocular indirect ophthalmoscopy. However, it is possible that some optometrists may not employ all these skills when examining a potential glaucoma suspect in the community, or repeat tests. It is true that this may be due to a combination of factors, including remuneration issues within the current General Ophthalmic Services contract in England and time constraints within general optometric practice. It is also likely that, in view of the low prevalence of the disease, average optometrists may not come across enough glaucoma cases in their lifetime for them to interpret their clinical findings confidently. As such they prefer to err on the side of caution and refer – a practice which should not be derided as it is better to be safe than to be sorry.

As Rumney and Henson point out, something needs to be done to improve the accuracy of referrals and this needs to be done soon in light of the new NICE guideline. It should not be forgotten that false-positive referrals can have a negative impact on the patient's well-being and peace of mind, in addition to the cost implications for the National Health Service (NHS).

Before primary care trusts (PCTs) start to commission optometrist-led glaucoma case detection schemes and possibly tender their contracts to the most financially attractive bidder, we should take a step back and consider what is in the patient's best interest. First and foremost, we need to open a dialogue with our ophthalmology colleagues and discuss how best we can jointly provide optimum patient care. An interdisciplinary approach to further training for the diagnosis and management of glaucoma in the community will ensure that harmony prevails between the professions and, most importantly, that there is no detriment to the patient. Whether this further training is funded by the NHS as is currently the case in Scotland is an avenue that should be explored. But that aside, a nationally accredited qualification approved by the College of Optometrists and endorsed by the Royal College of Ophthalmologists may well be the way forward.

Yours sincerely

Aachal Kotecha PhD, Department of Optometry and Visual Science, City University, London

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Optometry in Practice Vol 10 (2009) 89–94

Visual Acuity and Visual Satisfaction

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Date of acceptance 15 May 2009

Abstract

The aim of this concise study was to assess whether the attainment of a good level of clinical distance visual acuity (VA) was commensurate with subjective satisfaction with the visual experience. A group of healthy young adult myopic habitual spectacle wearers were each invited to rate their recent subjective distance visual experience using a single-item visual analogue scale (VAS). Each participant's binocular spectacle VA was then determined at 6m using a high-contrast logMAR letter chart. Finally, individuals were questioned (tick-box choice: 'No', 'Yes', 'Not Sure') as to the possibility of their needing a distance spectacle prescription change at their next routine sight test. The results recorded by these well-sighted spectacle wearers indicated that no clinical or statistically significant ($P = 0.1$) association was evident between subjective rating of recent visual experience and the level of VA. Similarly, the subjective opinion as to whether or not a distance spectacle prescription revision would be necessary in the near future was not statistically significantly associated with the VAS score ($P = 0.4$), and was also of doubtful clinical or statistical ($P = 0.04$) association with the habitually good acuity level recorded by these subjects. We conclude that when patients record a habitual distance VA on or better than the '0.0' logMAR chart line the subjective criterion of visual satisfaction – whether recorded in terms of the recent visual experience or the perceived necessity for a spectacle prescription change in the near future – is uniformly good.

Introduction

Attaining 'good vision' implies more than the clinical ability to read down to the lowest lines of smallest letters on the ophthalmic test chart. The advent of techniques such as contrast sensitivity testing demonstrated the restricted information gained by simply recording a subject's spatial visual resolution in response to a high-contrast stimulus array (Campbell & Robson 1968). Under certain circumstances a more discriminating and informative approach to the determination of an individual's visual capacity is desirable. Occasions where this might be necessary include the screening of applicants for prospective employment on specific vision-related tasks, or when investigating certain ophthalmic pathologies and treatments.

In recent years the patient-centred questionnaire approach has been shown to provide a supplementary dimension to the succinct visual acuity (VA) statement (Carta *et al.* 1998). Individuals might be questioned on their impressions of their recent visual experience, and whether current visual capabilities are considered adequate to meet future visual demands in the short to mid term. Clinical impressions as well as statistical significance of responses can be established, guiding the ophthalmic professional in aspects of specific case handling or clinical decision-making (for diverse examples see Abdi *et al.* 2006; du Toit *et al.* 2002; Rushood *et al.* 1997).

A previous study by the present author has investigated the application of the visual analogue scale (VAS) to the evaluation of recent visual experience (Pointer 2003). The VA range of that earlier study's participants was moderately broad: there appeared to be a (statistically significant) discontinuity in VAS scores depending upon whether individuals recorded a clinical acuity better or worse than 0.10 logMAR (6/7.5 Snellen).

The short study to be reported here built on this previous work. We addressed the following question: 'Just because a good clinical level of VA is recorded does it follow that the patient is satisfied with their vision?' To this end a group of healthy pre-presbyopic spectacle wearers who each recorded a habitual corrected acuity on or below the '0.0' line of the logMAR letter test chart were recruited. The subjects were invited to indicate: (1) their recent visual experience using a paper-based VAS, and (2) their impression (tick-box choice) of whether their current spectacle prescription would remain adequate over the near future.

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Methods

Subjects

The study population comprised individuals who had indicated verbally to the author their willingness to participate in a short non-intrusive and anonymous clinical study: all volunteers had received a description of the non-invasive procedures to be undertaken. Results were obtained from 180 individuals (63% female), with VAS test repetition upon a subgroup of 30 of these persons (17% of main group) to establish response reliability. The five items of information collected in the study (patient gender; age; binocular logMAR acuity at 6m; VAS score; tick-box response) were non-attributable in analysis, thereby preserving patient confidentiality.

All subjects were in good general and ocular health, and habitually wore spectacles (not contact lenses) to correct mild to moderate myopia. Each subject was selected from patients who had undergone a sight test at the practice within the previous 12 months: on that occasion the corrected binocular VA had been recorded as being at least 6/7.5 using the conventional Snellen test chart. All subjects were currently aged less than 40 years, and had no need for a presbyopic (reading) prescription.

The visual analogue scale (VAS)

The history, development and application of the VAS in a wide range of clinical studies have been summarised previously (McCormack *et al.* 1988; Pointer 2003). For the present study a 100mm horizontal paper-based VAS was devised, with the minimum/maximum anchor labels of 'Vision Very Poor'/'Vision Very Good' respectively (Figure 1).

Figure 1. A depiction (not to scale) of the printed visual analogue scale (VAS) used in this study to record recent distance visual experience. On the test sheets marked by the subjects the length of the horizontal black line was 100mm.

Procedures

Over the course of a 2-week period each of the 180 subjects attended the test facility for a brief individual appointment. Patients were seated in a well-lit room, given a copy of the VAS sheet (Figure 1) and asked to indicate how they rated their spectacle-corrected distance vision by making a pen mark directly on the printed sheet before handing it back to the author. Subjects were then engaged in general conversation, including being asked if their spectacles needed any adjustment, for a period of approximately 5 minutes. On a randomly selected subgroup of 30 individuals the VAS test was then repeated, using a fresh printed sheet.

All subjects were then asked to turn their chair around and view the previously obscured high-contrast logMAR test chart (Bailey & Lovie 1976); this comprised lines of letters spanning the range '0.8' (6/38) to '–0.5' (6/2) logMAR, and was presented at a distance of 6m. Starting with the largest letters on the top line, subjects were instructed to read out each successive letter, moving from left to right, on each progressively smaller line down the chart. Guessing was encouraged (but not mandatory) when the smallest letters were reached (Smith 2005) and the test was terminated when four or more letters on a line were read incorrectly (Carkeet 2001). Each individual letter correctly read contributed to the logMAR acuity score (Bailey & Lovie 1976; Vanden Bosch & Wall 1997): the interconversion between logMAR units and the familiar clinical Snellen VA notation has been described elsewhere (Pointer 2000).

Before leaving, patients were invited to complete a printed single-item questionnaire: 'Do you feel that your distance glasses prescription will need changing at your next sight test?' Three answer options ('No', 'Yes', 'Not Sure') were available and patients indicated their choice by ticking one of three adjacent boxes.

The entire procedure lasted approximately 10 minutes. On departure each subject was thanked for their participation in the study; it should be noted that no incentive or reward was given for attendance.

VAS scoring

The paper sheet marked by each subject had to be scored before data analysis could proceed. This was a simple matter of measuring the linear distance (mm) along the horizontal printed line from the left-hand end (minimum anchor point: 'Vision Very Poor') to the subject's pen mark. To promote consistency, measurement of all 210 VAS sheets (ie 180 main experiment plus 30 subgroup repeats)

was made by the author within a single session after termination of the study; a transparent linear gauge graduated in millimetres was used, which enabled a single measurement physical resolution of ±0.5mm. To assess the author's VAS measurement accuracy the entire procedure was repeated (with the previous measurement values masked to the author) on a second occasion a few days later. The results of an analysis of the author's intermeasurement repeatability (procedure as described by Bland & Altman 1986) revealed a mean (± standard deviation (SD)) difference between the two measurement sequences of –0.05 (± 0.19) mm, producing a coefficient of repeatability (SD × 1.96) of 0.37mm (ie within the actual measurement resolution). On the basis of this outcome, and that of a Wilcoxon's matched-pairs test (P = 0.22), accurate quantification of the VAS material was felt to be assured: consequently the mean of the two measurements was the value utilised in all subsequent analyses using these VAS data.

Statistical analysis

All data were entered into a spreadsheet for descriptive and statistical analysis using STATISTICA/Mac software (v4.1: StatSoft, Inc., Tulsa, Oklahoma, USA). Parametric (Student t) tests were applied to the age and acuity data. Elsewhere, non-parametric testing was employed, primarily because VAS data cannot be assumed to be normally distributed (Philip 1990): these tests included Kolmogorov–Smirnov, Mann–Whitney U, Wilcoxon's matched-pairs and Kruskal–Wallis ranks analysis, also the Median (relevant for testing skewed distributions) and Sign tests. The level of statistical significance was set at P <0.05.

Results and Discussion

Summary details relating to the age and gender distribution of the subject group, along with statistics of the three main experimental variables (VA; VAS score; whether or not a prescription change was anticipated), are given in Table 1. No statistically significant gender-based difference in subject age was indicated (P = 0.5) in this study population.

VA was statistically significantly different (P = 0.002) between the sexes: males recorded a higher mean acuity than females, a difference equal to 0.038 logMAR units or better by 1.9 chart letters. Note that a similar gender-based acuity difference in clinical material has been documented previously (Pointer 2008) but has been considered to be an outcome of doubtful clinical relevance.

Table 1. Summary statistics (mean ± SD), with the outcome of an intergender comparison given in the far right-hand column

Variable	Group (n = 180)	Male (n = 66: 37%)	Female (n = 114: 63%)	P
Age (years)	30.76 ± 5.38	31.29 ± 3.95	30.45 ± 6.05	0.49
Visual acuity:				
logMAR	–0.067 ± 0.065	–0.091 ± 0.067	–0.053 ± 0.060	0.002
Snellen	6/5.1	6/4.9	6/5.3	
VAS score (mm)	87.66 ± 10.13	85.23 ± 11.70	89.07 ± 8.85	0.07
Prescription change anticipated?				
'No Change'	n = 114	n = 60	n = 54	0.0001
'Not Sure'	n = 66	n = 6	n = 60	

P, level of statistical significance; VAS, visual analogue scale.

The VAS results were not statistically significantly different between genders (P = 0.07). Furthermore, a comparative analysis of the VAS scores obtained from repeat testing of 30 subjects produced a not statistically significant outcome (P = 0.6): the mean (±SD) difference between the first and second series of VAS determinations was 0.48mm (± 2.98), generating a coefficient of repeatability of 5.84mm (after Bland & Altman 1986).

Subjective responses to the questionnaire enquiry regarding the possibility of a spectacle prescription change at the next sight test fell into two categories only: 'No Change Anticipated' and 'Not Sure'. Interestingly, none of the subject group felt unequivocally that a revision would be necessary. The responses of female subjects were almost equally distributed between the two response categories, whereas the majority (90%) of males asserted that no change would be necessary. Although this different pattern of response between genders was statistically significant (P = 0.0001) the questionnaire-based intercategory distributions, when considered in terms of subject age, VA or VAS score, were each closely aligned in statistical analysis (Table 2).

Table 2. Questionnaire interresponse category analysis, 'No' versus 'Not Sure' if a spectacle prescription change will be necessary

Variable	'No Change' (n = 114: 63.3%))	'Not Sure' (n = 66: 36.7%)	P
Age (years)	31.64 ± 4.53	29.25 ± 6.34	0.11
Visual acuity:			
logMAR	–0.074 ± 0.064	–0.054 ± 0.066	0.04
Snellen	6/5.1	6/5.3	
VAS score (mm)	87.68 ± 10.89	87.61 ± 8.75	0.36

Mean ± SD; P, level of statistical significance; VAS, visual analogue scale

Drawing these outcomes together, VA is evidently the variable that might repay further investigative consideration here. An intergender acuity difference has been found (Table 1) in this material, although this outcome was not unexpected (Pointer 2008). Also, on the

basis of the gender difference found with regard to the possibility of any prescription change anticipated in the near future, those patients who felt that no change would be necessary recorded a slightly higher group acuity (Table 2) than those who were not sure if their spectacle prescription would need a revision. Pertinently, are either of these objective or subjective acuity-related outcomes reflected in the central subjective measure under investigation here, namely the VAS scores?

Adopting the strategy of Pointer (2003), the VAS data were partitioned across three lower lines of the logMAR test chart (ie to the chart lines designated '–0.2', '–0.1', and '0.0': Bailey & Lovie 1976) spanning the acuity levels recorded by the habitually well-sighted subjects in this present study, and subjected to statistical testing. The outcome was one of no statistically significant difference (P = 0.1) between the group VAS scores of subjects recording acuities between –0.20 and 0.08 logMAR inclusive. The breakdown and analysis of the results on a per-chart line comparative basis were: grouped VAS scores of subjects attaining acuities on '–0.2' versus '–0.1' logMAR chart lines (P = 0.9); on '–0.2' versus '0.0' logMAR lines (P = 0.5); and on '–0.1' versus '0.0' logMAR lines (P = 0.2). Thus the visual experience of subjects recording a spectacle-corrected VA of at least 0.08 logMAR (6/7.2 Snellen or better) is apparently one of uniformity: Figure 2 illustrates the distribution (central tendency and dispersion) of the VAS scores stratified across the three bands of logMAR acuity corresponding to three lower lines on the test chart.

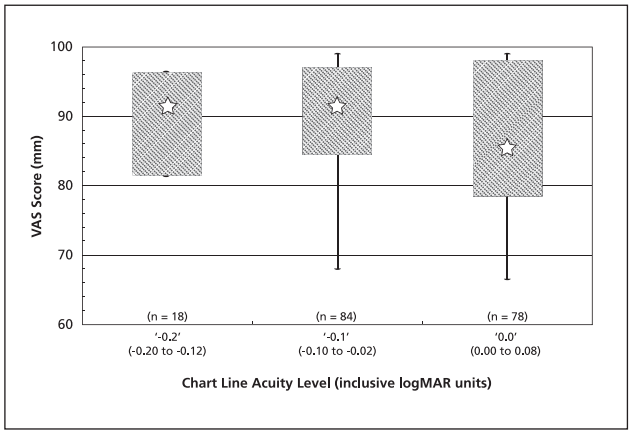

Figure 2. Box-whisker plots summarising the central tendency and dispersion of the visual analogue scale (VAS) scores (mm) stratified by logMAR acuity level (ie as corresponding to three lower lines on the test chart). The vertical rectangular hatched box indicates the interquartile range, the vertical whisker line the minimum/maximum range and the star symbol the median value. No statistically significant difference (P >0.1) was recorded between these three data distributions.

Conclusions

On the basis of these new results it is evident that when individuals record a habitually good level of distance VA the subjective indication of satisfaction with the visual result is also uniformly good. This outcome corroborates an earlier result published on this topic by the present author (Pointer 2003). However, an additional finding arising from this new study is that subjective opinion as to whether or not a prescription change will be necessary in the near future is also not associated with the VAS score and is not likely to be substantially influenced by the acuity level, provided that the habitual clinical VA is good.

In conclusion, it appears that the attainment of a good level of clinical VA is commensurate with subjective satisfaction with the visual experience.

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Multiple Choice Questions

This paper is reference C-11798. One point is available for optometrists and dispensing opticians. Please use the inserted answer sheet. Copies can be obtained from Optometry in Practice Administration, PO Box 6, Skelmersdale, Lancashire WN8 9FW. There is only one correct answer for each question.

1. The acronym 'VAS' means:
(a) Visual acuity score
(b) Visual analogue system
(c) Visual acuity standard
(d) Visual analogue scale

2. The number of subjects participating in this study was:
(a) 180
(b) 63
(c) 30
(d) 210

3. The VAS was marked by each subject:
(a) Verbally
(b) Manually
(c) Electronically
(d) By all of the above methods

4. Which statement is correct?
(a) A subject's VAS score was indicated by the linear measurement in millimetres from their mark to the maximum anchor point
(b) Single measurement resolution available for VAS quantification was ±0.19mm
(c) The author's coefficient of measurement repeatability as regards this VAS was 0.37mm
(d) Measurement repeatability was worse than single measurement resolution

5. A high VAS score indicated that the subject:
(a) Was certain that a prescription update would be necessary
(b) Felt good vision was experienced
(c) Was equivocal about their current vision
(d) Felt poor vision was experienced

6. As regards this study, which statement is correct?
(a) Monocular logMAR acuity was recorded
(b) Only contact lens-wearing subjects participated
(c) Male subjects recorded statistically significantly higher VAS scores than females
(d) There was no statistically significant difference between the VAS scores of subjects with acuities between –0.20 and 0.08 logMAR inclusive

Optometry in Practice Vol 10 (2009) 95–104

The Structure–Function Relationship in Glaucoma: Implications for Disease Detection

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Date of acceptance 19 June 2009

Introduction

Primary open-angle glaucoma (POAG) is an age-related optic neuropathy characterised by the death of retinal ganglion cells (RGCs) and irreversible visual field loss. The disease is commonly progressive, and is often, but not always, associated with raised intraocular pressure.

The precise mechanism of damage in glaucoma is poorly understood with several competing published theories (Fechtner and Weinreb 1994); however generally all theories agree that there is some physical or biochemical event which takes place at the optic nerve head, damaging RGC axons. The RGC axons then degrade along their length within the retinal nerve fibre layer (RNFL), ultimately leading to death of the RGC by apoptosis (Quigley 1998).

The number of individuals with glaucoma (of any type) is estimated at over 66 million worldwide, with at least 6.7 million bilaterally blind (Quigley 1996). Glaucoma is the second-leading cause of blindness in the UK and indeed the entire developed world (Kroese and Burton 2003; Quigley 1996) and its prevalence (proportion of the population with the disease at a given time) is expected to increase with demographic changes, including increased life expectancy (Tuck and Crick 2003). POAG is the most common type of glaucoma in the UK (Kroese and Burton 2003) and has a prevalence of around 2% in the Caucasian population (Tielsch *et al.* 1991). As POAG is usually asymptomatic until late in the progression of the disease, 80–90% of diagnosed cases in the UK are initially detected by optometrists during routine eye examination (Laidlaw *et al.* 1994).

Structural and Functional Measures in Detection of Glaucoma

The loss of RGC axons in glaucoma results in visible narrowing of the neuroretinal rim of the optic nerve head and thinning of the RNFL. These structural changes often have an appearance characteristic of glaucoma and have been described in detail elsewhere (Broadway *et al.* 1999; Caprioli 1994; Fingeret *et al.* 2005; Harding and Harper 2007; Jonas *et al.* 1999, 2000; Susanna 2007). Structural changes in glaucoma have traditionally been detected by clinical examination of the optic nerve head and RNFL by ophthalmoscopy; however in recent times objective structural measures have become achievable through methods such as confocal scanning laser ophthalmoscopy (Heidelberg retina tomograph (HRT), Heidelberg Engineering, Germany), scanning laser polarimetry (GDx, Carl Zeiss Meditec, CA, USA), and optical coherence tomography (OCT).

Glaucomatous functional change in the form of decreased visual field sensitivity is predominantly detected by standard automated perimetry (SAP) with white-spot stimuli on a white background. Alternative technologies, such as short-wavelength automated perimetry (SWAP), frequency-doubling perimetry (FDP/FDT), high-pass resolution perimetry (HRP) and multifocal visual evoked potentials (mfVEP), have all shown initial promise in improved sensitivity to early glaucomatous damage but currently have not shown clear evidence of significant improvements over SAP in the majority of patients (Bengtsson and Heijl 2006, Burgansky-Eliash *et al.*, 2007; Fortune *et al.*, 2007; Sample *et al.* 2006). SAP therefore remains the most commonly used functional test in glaucoma diagnosis and monitoring.

The Structure–Function Relationship in Glaucoma

As the relationship between RGC damage and visual field loss appears to be one of cause and effect it would seem reasonable to assume a relationship between the amount of RGC loss and degree of losses in visual field sensitivity. Two key questions arise from this assumption:

1. Which occurs first, structural changes or functional loss?
2. Can change be detected earliest with structural or functional measures?

This review aims to answer these questions.

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Early work

A very common quote amongst optometrists/ophthalmologists is that up to 50% of optic nerve fibres have already been lost before a visual field defect can be detected. This figure initially came from early work aiming to relate glaucomatous structural and functional changes (Quigley *et al.* 1982). The study compared the visual fields, as manually assessed by Goldmann kinetic perimetry, to the histological count of RGC axons post mortem. The sample sizes used in this study were very small, with only five normal eyes being used to establish the normal number of RGC axons, and only three 'glaucoma suspect' eyes who were without Goldmann visual field loss. It was only one of these patients who had an RGC count of 50% of the somewhat loosely established norm. Whilst the limitations of this study are obvious – the small sample sizes, the insensitive method used to detect visual field damage, the elapsed time between visual field measurement and optic nerve histological assessment – this paper provided a basic method for future studies, and has become one of the most cited papers in ophthalmology, with over 700 citations (Ohba *et al.* 2007).

In later work by the same group of researchers, a similar experiment was performed but using more modern SAP. This study was also limited by a small sample size (six glaucomatous eyes, one of which also had uveal melanoma, and four of which were a fellow eye to one with other ocular pathology; five healthy control eyes to establish normal range). The study concluded that a 10dB decrease in visual field sensitivity corresponded to 40% fewer RGCs than their established normal number (Quigley *et al.* 1989).

A later paper, also by the same group (Kerrigan-Baumrind *et al.* 2000), described another similar study but with slightly larger sample sizes (17 normal eyes, 17 glaucomatous eyes) and the use of the Humphrey field analyzer (HFA). The study reported that areas of visual field loss with Humphrey total deviation probability of $P < 0.5\%$ corresponded to 28.5% loss of RGCs. It is, however, important to note that within the $P < 0.5\%$ category are absolute scotomas, which one would logically associate with areas of no remaining RGCs. The value of 28.5% loss is not, therefore, surprising, being the average of a series of test locations ranging from losses that are just below $P = 0.5\%$ to absolute scotomas. What is surprising is that the paper concludes that 'at least 25% to 35% RGC loss is associated with statistical abnormalities in automated visual field testing'.

Reanalysis of the mean RGC counts in the control eyes in this study shows wide 95% confidence intervals of ±42% of the mean RGC count, likely due to the small sample size. This means that there was a large degree of overlap in RGC counts between glaucoma patients and controls such that even the upper limit of 35% RGC loss in their conclusion is not a statistically significant loss in their sample (it is within the range that can be considered feasibly normal according to their measurements) and as such the data presented do not support this conclusion (Hood and Kardon 2007). Indeed, looking at the figures presented, those with 100% of the 'normal' RGC axon count have, on average, a visual field mean deviation of worse than –6dB and pattern standard deviation of worse than 2.5dB, which probably would be statistically significant. The data presented by Kerrigan-Baumrind *et al.* therefore seem to support the opposite conclusion, that visual field loss occurs before RGC loss, equally as well as the conclusion made.

Behavioural experiments

In order to establish better the relationship between RGC count and visual function in glaucoma, Harwerth *et al.* carried out an elegant series of experiments using behavioural perimetry in rhesus monkeys (Harwerth *et al.* 1999, 2002, 2004, 2005). The monkeys were trained to perform visual field tests using an HFA, modified only in ways which did not affect the test outcomes. The monkeys were seated in a custom-made chair in a primate testing cubicle attached to the perimeter. This allowed their eyes to be correctly aligned for testing, their mouths to be positioned on a juice spout used to deliver behavioural rewards, and for them to hold a response switch. The monkeys were trained to press and hold down the response switch to initiate a trial, and subsequently to release the switch in the presence of a visual stimulus. The stimulus would be presented at a random time within 5.5s interval and if the monkey's response was within 900ms of stimulus presentation, then it was recorded as a true-positive response and the monkey rewarded. Alternatively, if the response was not within this time it was recorded as a miss and the monkey was neither rewarded nor punished.

Once the monkeys were trained to perform perimetric tests, glaucoma was surgically induced in one eye. This was achieved through argon laser treatment to the trabecular meshwork which resulted in intraocular pressure raised consistently above 40mmHg, leading to glaucomatous damage similar in many ways to that in human patients (Harwerth *et al.* 1997). The fellow eye served as a control eye, allowing measures of glaucomatous loss with greater confidence than previous studies. Once the visual fields had been accurately measured at the desired stage of damage, the eyes were enucleated for histological analysis, giving a direct and almost immediate comparison between structural and functional measures.

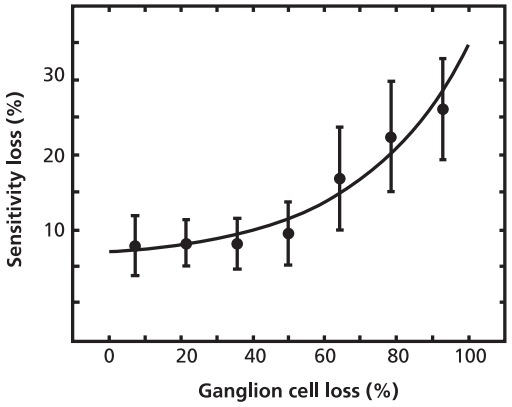

Figure 1. Primate retinal ganglion cell loss versus visual sensitivity loss plotted on log-linear axis (after Harwerth et al. 1999).

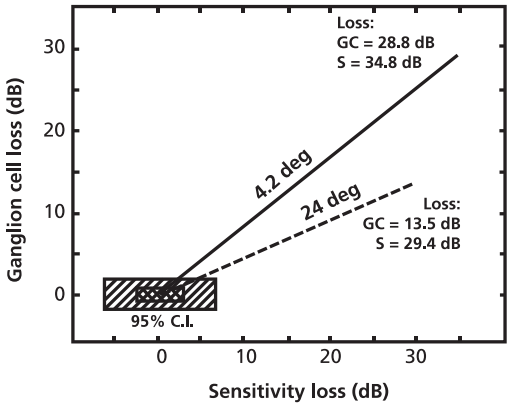

Figure 2. Primate retinal ganglion cell loss correlated with visual sensitivity plotted on log-log axes to produce a linear relationship, with retinal eccentricity taken into account (after Harwerth et al. 2004).

Early results, plotted on log-linear axes (Figure 1), were taken in partial support of previous work, in that visual sensitivity loss appeared small and constant until around 30–50% of RGCs were lost, after which a more steady relationship between RGC and visual sensitivity loss appeared (Harwerth *et al.* 1999). Whilst the results appear to suggest either that clinical perimetry is insensitive to early neural loss, or that there is some redundancy in the visual system such that a proportion of RGCs can be lost before vision is affected, account needs to be taken of the log-linear scaling used, which emphasises larger losses to the detriment of smaller, early losses.

In later work, Harwerth *et al.* (2004) addressed the issue of variability in the structure–function relationship by taking into account retinal eccentricity as a factor in expected RGC axon count, eliminating a proportion of the scatter in the model. The issue of scaling was also addressed, finding that plotting RGC loss on a logarithmic scale (dB) produced a linear relationship between RGC loss and visual sensitivity loss (also measured in dB), with varying gradient depending on retinal eccentricity (Figure 2) (Harwerth *et al.* 2004, 2005).

The structure–function model derived from primate data was then applied in a reanalysis of the data from the study by Kerrigan-Baumrind *et al.* (2000). The model was used to predict RGC axon counts from measured visual sensitivities of glaucoma patients, and the results compared to postmortem histological cell counts. The results showed that clinical perimetry is, in fact, a direct expression of glaucomatous neural loss and as such a good measure of stage of disease (Harwerth and Quigley 2006). The application of the model to human data revealed a slightly less precise relationship between structure and function. This may be due to the elapsed time between the visual field being measured and the patient's death and subsequent histological RGC count, but may also be due in part to reduced variability in visual field measurements obtained from well-trained primates compared to clinical measurements obtained from patients without extensive perimetric experience (Harwerth *et al.* 1993).

The spatial structure–function relationship

In order to establish a relationship between functional measures and currently available in vivo measures of retinal and optic nerve head structure it is essential first to establish a proper spatial relationship between these. That is, to establish a relationship between the locations tested in clinical perimetry and the corresponding areas of RNFL and optic nerve head.

One such model, which has since been widely used, was derived from fundus photographs obtained during routine clinical examination of normal-tension glaucoma patients (Garway-Heath *et al.* 2000). Photographs featuring visible, well-defined RNFL bundle defects (so-called 'wedge defects') or prominent nerve fibre bundles were selected, and the defects manually traced back to the optic nerve head. A scaled HFA 24-2 visual field matrix was then superimposed on to the photographs and the relationship of visual field test locations to the optic nerve head circumference was estimated by observing the proximity of test locations to nerve fibre defects/bundles. The resulting map divided the optic nerve head into six sectors, each with a corresponding area of visual field test locations (Figure 3).

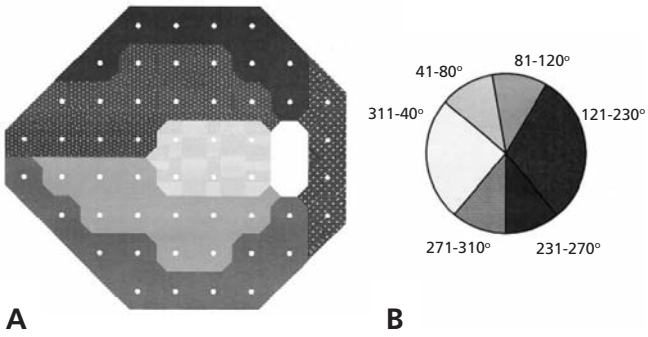

Figure 3. Visual field divisions (A) and corresponding optic nerve head sectors (B) (after Garway-Heath et al. 2000).

This model has been incorporated, with some small modification, into the HRT software and used in several studies aiming to correlate neuroretinal rim measurements or peripapillary RNFL thickness (measured by OCT) to visual field sensitivities. The model does have some clear limitations. It assumes a consistent retinotopic organisation of RGC axons travelling in bundles, which is not well established. For example, it may be possible that there is some tendency of RGC axons to move between adjacent bundles, and some controversy exists over the relative position of optic nerve head insertion of long (peripheral) and short (central) RGC axons which may affect the resultant spatial map (Fitzgibbon and Taylor 1996; Minckler 1980; Ogden 1974, 1983; Radius and Anderson 1979). The relatively large sectors used in the map were a product of large between-subject variability, much of which can be explained by variations in optic nerve head position (Garway-Heath *et al.* 2000). The map has not been verified by examination of corresponding visual field data and therefore remains an essentially theoretical, albeit useful, representation of the spatial structure–function relationship. These limitations increase variability and, therefore, limit the precision of subsequent in vivo quantitative studies which make use of the spatial model.

Later work by Gardiner *et al.* (2005) used cross-sectional HFA 24-2 visual field and HRT structural data in an attempt to refine the spatial map. The resultant map divided the optic nerve head into 36 10° sectors (thereby increasing resolution) and assessed the correlation of each sector to each visual field test location (Gardiner *et al.* 2005). However, the correlations were mostly very weak, with the mean best correlation found for each optic nerve head sector being 0.28 (range 0.12–0.52). This, combined with the increased complexity of the map, means it has not been widely adopted and as such the map produced by Garway-Heath *et al.* (2000) is still the most often used.

In vivo measurements

Garway-Heath *et al.* (2002) compared visual sensitivity at HFA 24-2 visual field test locations and pattern electroretinogram response amplitudes to checkerboard stimuli to optic nerve head neuroretinal rim thickness as measured by HRT using the spatial map proposed earlier by their own group (Garway-Heath *et al.* 2000). They found a weak but significant linear relationship between optic nerve head neuroretinal rim area and both pattern electroretinogram amplitudes and visual field sensitivity values when plotted on linear scales (Garway-Heath *et al.* 2002). The study provided further evidence that structural and functional changes in glaucoma occur concurrently, and there is no RGC functional reserve.

Studies using OCT to measure RNFL thickness have also used the above spatial models to correlate sensitivity at HFA 24-2 visual field test locations in the arcuate areas to RNFL thickness in corresponding parapapillary areas (Figure 4). In agreement with the primate and human studies previously mentioned, a linear model was found to be the best fit to the data obtained from human glaucoma patients (Hood and Kardon 2007; Hood *et al.* 2007). In using OCT data however, it must be remembered that the entire RNFL thickness is measured, which contains not only RGC axons, but also glial cells, capillaries and efferent fibres. As such there is a certain minimum thickness in every patient, beyond which no amount of glaucomatous damage will thin the RNFL. This minimum thickness manifests itself as the intercept with the *y*-axis (RNFL thickness), or as the asymptote to the curve when results are plotted on log-linear axis (Hood and Kardon 2007; Hood *et al.* 2007).

In vivo structural measurements using OCT have also been taken in primates and correlated with visual field sensitivity measurements from behavioural perimetry, as described above. The linear model was again the best fit to the data, which in this case were presented as percentage loss, thus avoiding the above issue of residual measured thickness (100% loss does not mean zero thickness) (Harwerth *et al.* 2007). This study shows good agreement with those on human data, confirming the validity of the primate model used.

These linear models, whilst being the best fit to the data, do not account for all of the variability in the studied populations, ie the data are still quite scattered around the regression line. Much of the scatter is probably due to the variability in the measurements taken, especially perimetry where response variability has been shown to be inversely correlated to sensitivity (ie as the sensitivity worsens, variability increases) (Henson *et al.* 2000).

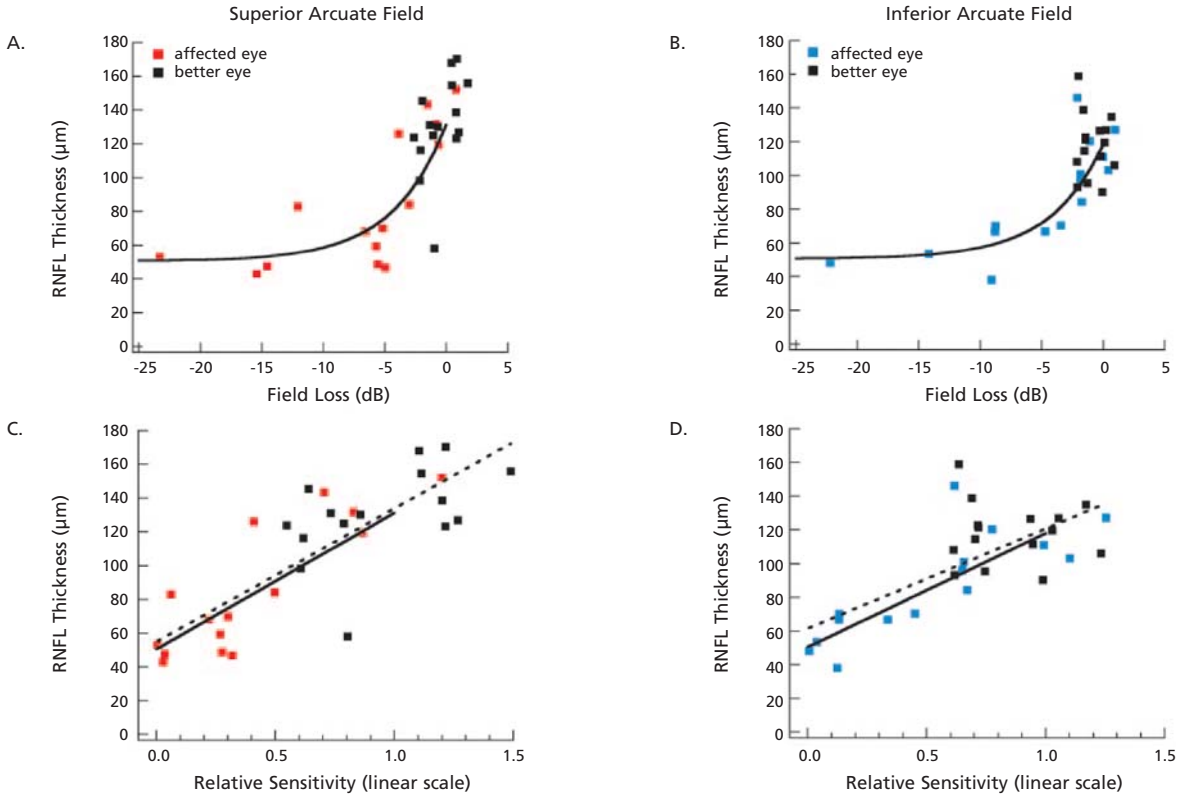

Figure 4. Visual sensitivity versus retinal nerve fibre layer (RNFL) thickness for superior and inferior arcuate areas of the visual field, plotted on log-linear (A, B) and linear axes (C, D) (after Hood et al. 2007).

Another potential method of improving the model for in vivo measurements is to improve the precision of the spatial models used, as variability within these is reflected in the results of any quantitative study using them.

Disease detection

It is a commonly held belief that structural damage precedes functional loss in glaucoma, but current evidence suggests that the two occur concurrently throughout the progression of the disease. That being the case then, the question, 'can change be detected earliest with structural or functional measures?' becomes purely a question of instrumentation and interpretation, and is perhaps more appropriately phrased as 'can statistically significant change be detected earliest with structural or functional measures?'

First, the issue of statistical significance of a test result must be understood. Usually we say a result is statistically significant when $P < 0.05$, meaning that the probability of the obtained result occurring in the healthy population is less than 5%. When the test results in the healthy population are normally distributed (equally spread about the mean), the test result must be more than ±1.96 standard deviations away from the mean to achieve statistical significance at this 5% level. This range of 1.96 standard deviations either side of the mean is known as the central 95%.

Consider then, two tests of a single variable, carried out on the same population of subjects. Even though the 'true values' are the same for both tests, if one test produces more variable results than the other, the central 95% is wider and so the spread of results considered 'healthy' is greater. It follows that, for a subject tested who was originally around the mean in this measure, but has since decreased by a given amount, the test with less variable results could detect a statistically significant deviation from 'healthy' as the result would fall outside the central 95%, whereas in the more variable test the subject could still fall within the central 95% of normal test results and be classed as 'healthy' (Figure 5). This could be considered the case in glaucoma, where, for example, RNFL thickness measured by OCT and visual field sensitivity measured by perimetry are both surrogate measures of neural loss, but have different degrees of variation in the healthy population.

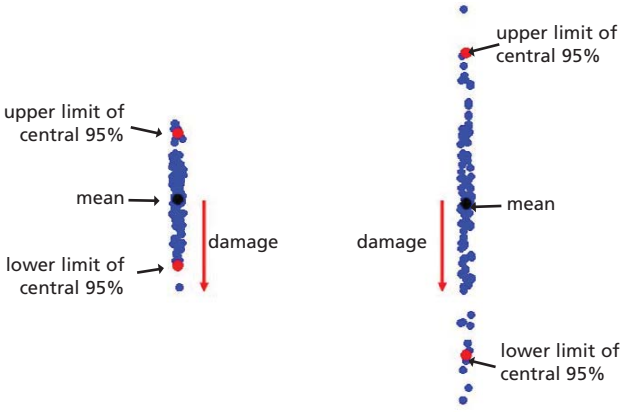

Figure 5. Illustration of 100 measures of healthy patients (blue dots) tested by two methods. In the test on the left the amount of damage results in a statistically significant defect, whereas in the test on the right the same damage starting from the same point is still within the variability in the healthy population and so is undetected by the test.

Figure 6 uses the example of OCT and perimetry to highlight further the effect of changing variation in the healthy population on disease detection. In Figure 6A, more people have statistically normal visual field sensitivity and statistically abnormal RNFL thickness than vice versa and, so of those where only one of the tests detects the disease, OCT detects glaucoma more often. However, in Figure 6B, the variability of test results from the healthy population is changed such that the opposite is true.

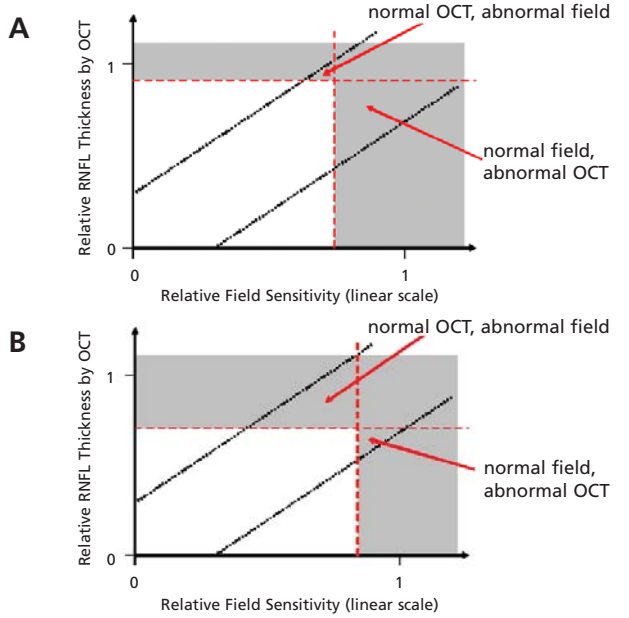

Figure 6. Effect of changing variability of test results from the healthy population on disease detection by two tests. Dotted grey lines represent upper and lower limits of test results; shaded grey areas represent central 95% of test results for visual field and retinal nerve fibre layer thickness (see text for further explanation).

In real patients then, the current evidence suggests that, whilst all patients beyond a certain stage of disease are detected by either test, in the case of early disease where the two tests disagree, more patients will show statistically significant RNFL loss on OCT and statistically normal visual field sensitivities than vice versa (Figure 7), but it is important to remember that there are still many patients for whom perimetry detects statistically significant glaucomatous damage before RNFL thickness measurement (Hood and Kardon 2007).

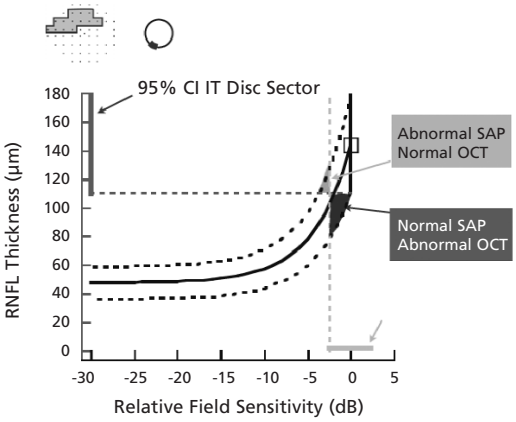

Figure 7. Relative sensitivity in the superior arcuate area of standard automated perimetry versus optical coherence tomography (OCT) for detection of glaucoma. Dark grey shaded area shows that more patients have normal perimetry and abnormal OCT than vice versa (smaller, light grey shaded area) (after Hood and Kardon 2007).

As a slight aside, another issue to consider when attempting to answer the question of which occurs first is that of selection bias. Many trials and studies of new instruments take a 'high-risk' population such as those with ocular hypertension, and exclude all those within the population with statistically significant visual field loss from the study. The remaining subjects are then tested with the new instrument, which detects statistically significant structural damage in some, and thus concludes that the new instrument detects glaucoma earlier than perimetry. This conclusion is true for some subjects, but it is important to remember that there are likely to be some subjects with statistically significant visual field loss who were excluded from the study, whom the new instrument does not correctly classify, ie the study population is biased in favour of the new instrument.

The topic of correctly classifying early glaucomatous eyes in a cross-sectional sample has frequently arisen in the literature in recent years with the arrival of new instruments such as the GDx, HRT and OCT, and this topic has many parallels within optometric and ophthalmic practice. It is, however, important to remember that this is an inherently poor method of glaucoma detection. The wide and overlapping variation in both structure and

function of normal and glaucomatous eyes means that there is no cut-off point beyond which we can say that a patient has glaucoma. Glaucoma is a continuous, progressive disease and as such it is better to look for significant change within a subject in multiple visits over time. Obviously, this has its practical implications and so sensitive and specific methods are needed to detect glaucoma in a single visit, but this is not ideal.

It is worth briefly noting that, late on in the progression of the disease when structural tests approach their baseline ± measurement error, functional tests may become better for monitoring.

Future Directions

Improvements in the structure–function relationship model in the form of reduced scatter and more precise estimates of the slope may come via improved precision in spatial structure–function maps and reduced variability in testing methods, especially perimetry where patient factors as well as physiological and technological factors affect results. Psychophysical developments in visual field testing, whether through new techniques which selectively examine certain neural pathways, or through improved testing of peripheral locations by altering the stimuli used, may also help to reduce scatter in structure–function models (Anderson 2006).

Another area which has been given little consideration in the past is that of spatial types of glaucomatous optic nerve head and visual field damage. It is widely accepted that glaucoma is not actually one disease, but rather a group of diseases with similar characteristics, and so it follows that these slightly different diseases may have slightly different patterns of structural and functional damage. It has already been demonstrated clinically that many glaucoma patients can be placed into categories based on optic nerve head appearance; these patients then have other similar disease characteristics such as spatial patterns of visual field loss to those within the same category (Broadway *et al.* 1999). Similar work has been carried out with visual field data to attempt to classify patients based on spatial patterns of sensitivity loss (Brusini and Johnson 2007; Keltner *et al.* 2003). These may be important findings as it is possible that these distinct disease types may have different underlying mechanisms which progress and respond to treatments differently. Unfortunately it is difficult to know exactly how many categories there should be, and it seems likely that if these distinct disease mechanisms exist, many patients will be affected by more than one. Broadway *et al.* (1999) found it impossible to classify the majority of their patients into one of their four optic nerve head categories. Whilst more categories may help somewhat, it may be more likely that increased understanding of these types could come through objective statistical mapping techniques of optic nerve head (Yan *et al.* 2005) and visual field data (Twa *et al.* 2008).

In terms of detection of early glaucomatous damage in a single patient visit, a major weakness of current methods is in extracting individual results from the variability of a group. It would be better to assess a patient's results against that patient's own variability, but this is not possible with current visual field reliability indices as these do not accurately predict variability across multiple tests (Bengtsson 2000). For imaging techniques it may be possible to use image quality and variation in repeated images taken within the same session to alter confidence interval estimates, potentially improving early disease detection where good-quality images can be obtained.

Conclusions

Current evidence suggests that glaucomatous structural change and functional loss occur concurrently and linearly throughout the progression of the disease. There appears to be little or no redundancy in the visual system in the form of any RGC functional reserve, such that small losses of RGCs cause small losses in visual sensitivity; however these are not necessarily noticed by the patient or detected by current methods.

The matter of early detection methods in glaucoma is dominated by statistical issues. Which tests are more sensitive to early damage depends on the relative variation of results in healthy controls, and the initial (predisease) structural and functional status.

In many patients, structural measures achieve statistical significance before functional measures, but in some patients the opposite is true. It is, therefore, optimal to measure both structure and function whenever possible.

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Multiple Choice Questions

This paper is reference C-11799. Three points are available for optometrists. Please use the inserted answer sheet. Copies can be obtained from Optometry in Practice Administration, PO Box 6, Skelmersdale, Lancashire WN8 9FW. There is only one correct answer for each question.

1. The majority of cases of primary open-angle glaucoma in the UK are detected by:
(a) General medical practitioners
(b) Specialist glaucoma screening programmes
(c) Optometrists during routine eye examinations
(d) The Hospital Eye Service

2. Which of these methods can be used to provide objective measures of ocular structures relevant to glaucoma detection and monitoring?
(a) Scanning laser polarimetry
(b) Optical coherence tomography
(c) Confocal scanning laser ophthalmoscopy
(d) All of the above

3. Which method did Quigley *et al.* (1982) use to measure the visual fields of their subjects?
(a) Manual kinetic perimetry
(b) Short-wavelength automated perimetry
(c) Frequency-doubling perimetry
(d) Standard automated threshold perimetry

4. During behavioural perimetry, to be regarded as a true positive the monkey's response must be within what time period of stimulus presentation?
(a) 5.5s
(b) 3.7s
(c) 1.4s
(d) 0.9s

5. In Harwerth's behavioural structure–function model, the rate of change in sensitivity with respect to ganglion cell loss depends on:
(a) The retinal eccentricity tested
(b) The scaling used to plot the model
(c) The duration of the perimetric test
(d) All of the above

6. Garway-Heath *et al.* developed their spatial structure–function map using:
(a) Fundus photographs
(b) Optical coherence tomography sectoral retinal nerve fibre (RNFL) layer measurements
(c) Sectoral Moorfields regression analysis
(d) Histological ganglion cell counts

7. The retinal nerve fibre layer contains:
(a) Glial cells
(b) Retinal ganglion cell axons
(c) Capillaries
(d) All of the above

8. In end-stage glaucoma the RNFL thickness reaches a minimum which is:
(a) Zero due to loss of all retinal ganglion cell axons
(b) Not zero due to some residual unaffected retinal ganglion cell axons
(c) Not zero due to the presence of other unaffected RNFL components
(d) Not zero due to measurement error

9. Response variability in perimetry:
(a) is unaffected by sensitivity
(b) is increased in patients with a visual field defect
(c) is decreased in patients with a visual field defect
(d) cannot be measured in patients with a visual field defect

10. Which is correct in glaucoma based on current evidence?
(a) Structural damage precedes functional damage
(b) Functional damage precedes structural damage
(c) Structural and functional damage occur at the same time
(d) There is no relationship between structural and functional damage

11. Which is most correct regarding the early clinical detection of glaucoma?
(a) Perimetry always detects glaucomatous damage before RNFL thickness measurement
(b) RNFL thickness measurement always detects glaucomatous damage before perimetry
(c) Either method may detect glaucomatous damage before the other
(d) Both methods detect glaucomatous damage at the same time

12. Beyond a certain stage of disease glaucomatous damage can be detected by:
(a) Either structural or functional methods
(b) Structural measures only
(c) Functional measures only
(d) Neither structural nor functional methods

13. Which of the following tests is the most commonly used functional test in glaucoma diagnosis and monitoring?
(a) High-pass resolution perimetry
(b) Short-wavelength automated perimetry
(c) Standard automated perimetry
(d) Multifocal visual evoked potentials

14. Which of the following statements regarding statistical significance is incorrect?
(a) If $P<0.05$ the probability of the obtained result occurring in the healthy population is less than 5%
(b) If $P<0.05$ the probability of the obtained result occurring in the healthy population is less than 0.5%
(c) If results are normally distributed a test result must be more than ± 1.96 standard deviations from the mean to achieve statistical significance at the 5% level
(d) The range of 1.96 standard deviations either side of the mean is known as the central 95%

15. How many glaucoma suspect eyes were examined in Quigley's (1982) study which led to the belief that 50% of optic nerve fibres have already been lost before a visual field defect can be detected?
(a) 3
(b) 30
(c) 300
(d) 3000

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Current Trends in Macular Hole Management

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Introduction

A macular hole is defined as a full-thickness defect of the foveal retina from the internal limiting membrane to the outer segment of the photoreceptor layer. Although the first reported case, described by Knapp in 1869 (Mireskandari *et al*. 2004), was ascribed to a traumatic aetiology, practitioners have recognised that this condition occurs more commonly in patients without any antecedent injury. As such, macular holes are characterised as idiopathic full-thickness macular holes to differentiate them from their traumatic counterpart.

The differential diagnosis of full-thickness macular holes includes lamellar holes, epiretinal membranes and cystoid macular oedema. Lamellar holes represent partial-thickness defects of the retinal layers within the macular area. Fluid-filled cysts forming within the outer plexiform and inner nuclear layers of the retina are the characteristic, pathological changes seen in patients harbouring cystoid macular oedema. It is noteworthy that these cysts may coalesce to form a lamellar hole. Epiretinal membranes occur due to proliferation of retinal glial cells through breaks in the internal limiting membrane. The appearance of these membranes ranges from a heightened irregular light reflex at the macula (cellophane maculopathy) to marked retinal wrinkling and distortion of the blood vessels (macular pucker).

Idiopathic macular holes are relatively common, with a prevalence of approximately 3 per 1000 people (Ezra *et al*. 1998). In one population-based study, the investigators revealed that macular holes were as common as glaucoma and diabetic retinopathy (Rahmani *et al*. 1996). Furthermore, idiopathic holes have a predilection for women over the age of 65 years (Evans *et al*. 1998). In view of the afore-mentioned epidemiological and demographic characteristics, optometrists are likely to be the first practitioner that such patients may consult. It is therefore important that optometrists are conversant with the proposed pathogenesis, methods of examination and surgical treatment of this macular malady. Moreover, knowledge of the various surgical stratagems with their attendant prognostic outcomes will provide guidance with regard to the suitability and timing of referral of such patients.

Pathophysiology

Although the vitreous was conjectured to be involved in the pathogenesis of macular holes in 1924 (Lister 1924), a classification system was only described six decades later implicating both anteroposterior and tangential vitreoretinal traction as the main causative factors relevant to their evolution (Johnson and Gass 1988). Notwithstanding the fact that the classification system is employed by the vitreoretinal community worldwide, the exact mechanism of vitreofoveal traction leading to the formation of idiopathic macular holes remains elusive (Bainbridge *et al*. 2008).

Contraction of the prefoveal vitreous cortex following invasion of underlying retinal Müller cells has been proposed as a mechanism for tangential traction (Gass 1988). Following perifoveal vitreous separation, any abnormal, persistent vitreoretinal attachment that is subject to dynamic tractional forces may induce significant anteroposterior traction, leading to hole formation (Gaudric *et al*. 1999). Posterior vitreous detachment (PVD) plays an instrumental role in the creation of macular holes, as evidenced by the enlargement of existing holes (Ezra 2001) and the increased risk of hole formation in the contralateral eye of affected individuals immediately following a PVD (Lewis *et al*. 1996). As a result, it was considered that the presence of a PVD in the contralateral eye confers immunity against acquiring a macular hole. However, there have been two case reports in which macular holes developed 2 and 3 years after successful rhegmatogenous detachment repair in which PVDs were present at the time of their initial retinal insult (Sheth and Bainbridge 2008). This suggests that anteroposterior traction is not a sine qua non of the formation of macular holes.

In a reappraisal of his original theory, Gass hypothesised that the foveolar receptor cells are bound together by a group of Müller cells which he termed the 'Müller cell zone', the disruption of which may lead to a foveal schisis or cyst (Gass 1999). Avulsion of the cystic roof results in a fully detached operculum that remains suspended on the posterior vitreous cortex. Clinicopathological studies have demonstrated that the opercula mainly consist of vitreous

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cortex and glial remnants (Gass 1998). Photoreceptor bodies have also been detected in under half of opercula (Ezra *et al*. 1997). Following disruption of the Müller cell zone, the photoreceptor layer undergoes centrifugal retraction leading to a full-thickness retinal dehiscence (a split). The resultant hole allows the overlying syneretic (liquefied) vitreous fluid to gain entry to the adjacent subretinal tissue, thus augmenting the elevated edges of the hole (Tornambe 2003).

The macular stages are described as follows:

- Stage 1a: macular cyst or impending macular hole. Ophthalmoscopically, it appears as a yellow spot. This is due to elevation of the fovea from vitreous traction with a concomitant increase in visualisation of xanthophyll pigment.
- Stage 1b: the xanthophyll pigment appears to adopt a doughnut-shaped yellow ring as the foveal tissue elevates. Dehiscence (a split) of deeper retinal layers at the umbo (depression in the foveola conferring to the foveolar reflex) may occur as a result of persistent traction within this area.
- Stage 2: defined as a full-thickness macular hole $< 400\mu m$ in size.
- Stage 3: defined as a full-thickness macular hole $> 400\mu m$ in size associated with posterior cortical vitreous adhesion or traction.
- Stage 4: represents a full-thickness macular hole whose size dimensions are equal to its stage 3 counterpart in which a PVD is complete.

In the Eye Disease Case-Control Study (Chew *et al*. 1999), the investigators reported the risk of developing a hole in the second, hitherto unaffected eye to be 4.3% for 3 years or less of follow-up rising to 7.1% after 6 years or more of follow-up. In view of the fact that a macular hole develops before the onset of a PVD in the majority of cases, it is not unreasonable to assume that the risk of developing this macular malady in the fellow, unaffected eye harbouring such a detachment is negligible.

Clinical Examination

The symptoms associated with idiopathic macular holes are multifarious and may include metamorphopsia, blurring of central vision and (if the hole is large) a central scotoma. Interestingly, it is not uncommon for the patient to be asymptomatic and the hole detected following occlusion of the unaffected eye during a routine optometric eye examination.

The decline in visual acuity in patients harbouring a macular hole is positively correlated with the amount of retinal tissue lost. Notwithstanding this fact, patients may experience a substantial degree of visual dysfunction which is disproportionate to the size of the lesion. This disparity is attributed to the concomitant cuff of subretinal fluid and its attendant photoreceptor atrophy. Patients harbouring a stage 1 hole are affected the least and maintain excellent visual acuities. Central, full-thickness holes, by contrast, confer the worst visual acuity scores. Although the typical level of acuity in such patients is 6/60, it is not uncommon for the level to be substantially worse than this.

Signs

True holes may be differentially diagnosed from masquerading lesions such as epiretinal membranes with pseudoholes (Figure 1), lamellar holes and cystoid macular oedema (Figure 2) from their fundal biomicroscopic appearance alone. A typical full-thickness hole is characterised by an oval or round lesion with yellow dots at its base (Klein's retinal tags) and a cuff of presumed subretinal fluid (Martinez *et al*. 1994). Figure 3

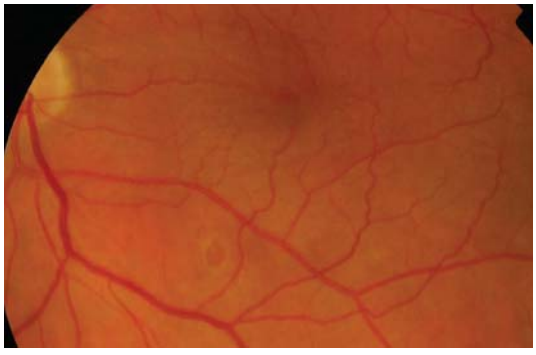

Figure 1. Pseudomacular hole secondary to epiretinal membranes.

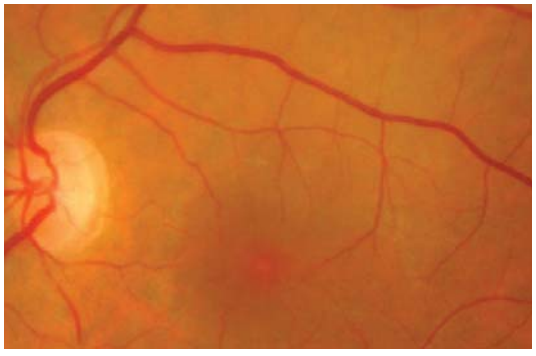

Figure 2. Cystoid macular oedema.

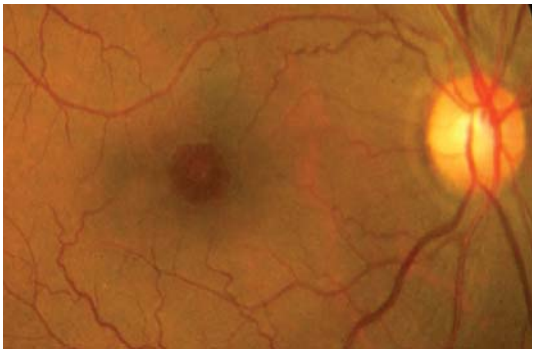

Figure 3. Stage 4 macular hole.

demonstrates a stage 4 macular hole. The presence of a prefoveal opacity or pseudo-operculum provides further support to the diagnosis. Klein's retinal tags may represent lipofuscin-laden macrophages of the underlying retinal pigment epithelium.

Occasionally, differentiating true holes from their imitators is difficult and necessitates the employment of additional tests. The Watzke–Allen slit beam test is one such test that may easily be incorporated in the optometric eye examination (Watzke and Allen 1969). This involves projecting a narrow slit beam of light through either a fundus contact lens or non-contact, fundus biomicroscopy lens on to the fovea. A reported break in the beam of light equates to a positive test and is due to a lack of retinal tissue in the projected area. It was once considered that a narrowing or distortion is not diagnostic. Indeed, the presence of a reported break has been utilised as entry criteria for inclusion into major macular hole studies, including the Vitrectomy for Macular Hole Study Group (Freeman *et al*. 1997; Kim *et al*. 1996). However, one significant study demonstrated that the majority of patients who harbour macular holes as identified by ocular coherence tomography (OCT) did not report a break in the beam of light (Tanner and Williamson 2000). Another study demonstrated that, in 22 patients confirmed as harbouring a full-thickness macular hole via a retinal nerve fibre analyser, 8 reported a thinning of the slit beam (Asrani *et al*. 1998). Tanner and Williamson (2000) conjectured that the reason for bowing or thinning of the slit beam observed in the majority of their macular hole cohort was due to the displacement of photoreceptors towards the rim rather than total loss of retinal tissue per se. Thus, patients who report thinning of the light have functioning foveal tissue in the rim of the hole.

Tanner and Williamson (2000) proposed two reasons why patients reported a break in the slit beam. First, holes of longer duration would allow time for the displaced photoreceptors to undergo secondary degeneration; and, second, hole formation was attributable to an alternative mechanism in this cohort. The former postulation is unlikely as there was no statistical difference in the mean duration of symptoms in both groups.

Notwithstanding the questionable validity of the Watzke–Allen test as a stand-alone diagnostic tool, the author recommends its utilisation during the optometric assessment of these patients, since distortion or a break definitely signifies macular pathology. It is a simple, non-invasive test to execute that can be readily incorporated into the eye examination. Whether the presence of a break serves as a useful, preoperative, prognostic factor for macular surgery is a question that remains unanswered (Tanner and Williamson 2000).

An alternative to the Watzke–Allen test, albeit one that is unavailable in optometric practice, is to aim a 50μm argon laser beam on to the lesion. Patients are invited to describe what they see. A positive test occurs when the laser beam is not detected when projected on the area of interest but appears when projected on normal retinal tissue.

The use of Amsler grid tests, although sensitive for macular lesions, is not specific for macular holes. Thus, their use is limited in the ophthalmological work-up of macular holes. However, these tests are readily available in optometric practice and, if a small scotoma is detected, would raise the optometrist's suspicion of a hole being present when the fundal appearance is equivocal.

Imaging

OCT remains the non-invasive diagnostic test par excellence in both the diagnosis and staging of macular holes due to its ability to evaluate the vitreomacular interface (Hee *et al*. 1995). The test also allows the practitioner to differentiate macular holes (Figures 4 and 5) from similar ophthalmoscopic lesions such as lamellar holes (Figure 6) and cystic changes (Figure 7). Although this method of examination is not commonly utilised in optometric practice, its use is routine in both the pre- and postoperative assessments of macular holes in ophthalmology clinics. Some authors have utilised this instrument in order to study whether or not the preoperative findings correlate with functional outcomes following surgery

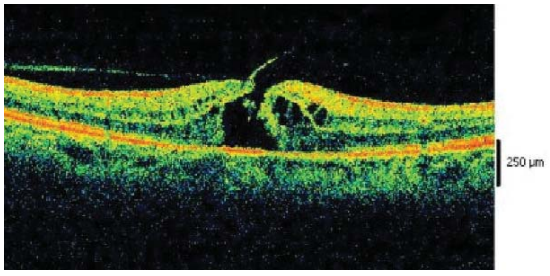

Figure 4. Ocular coherence tomography image of a stage 2 macular hole. Note full-thickness defect centrally (approximately 200µm in diameter) with cystic spaces to the right of the defect. Hyaloid still attached to the lid of the hole.

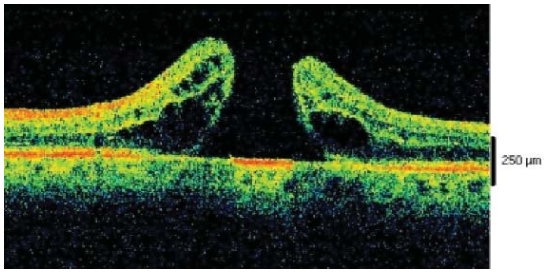

Figure 5. Ocular coherence tomography image of stage 4 macular hole. Full-thickness defect > 400µm and thick, everted edges.

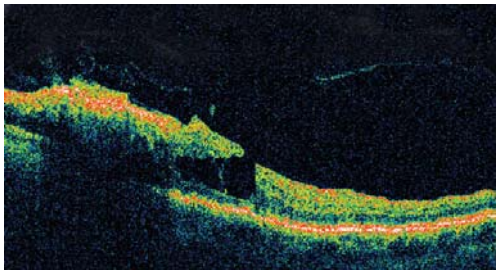

Figure 6. Ocular coherence tomography image of a lamellar hole secondary to vitreomacular tractional syndrome. Note residual retinal tissue underneath lesion, differentiating it from a full-thickness hole and overlying retracted hyaloid.

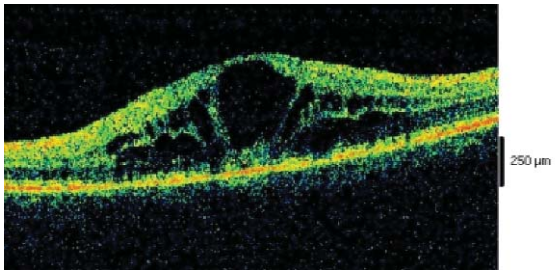

Figure 7. Ocular coherence tomography image of cystoid macular oedema. Multiple cystic lesions both centrally and paracentrally. Note residual retinal tissue underneath central cyst and marked increase in retinal thickness.

(Haritoglou *et al*. 2007; Kusuhara *et al*. 2004). In a refinement of OCT measurements, Ruiz-Monero *et al*. (2008) concluded that a macular hole whose minimum diameter was < 311µm conferred a better prognosis following surgery. Moreover, a tractional hole index (THI), defined as the ratio of the diameter of the hole at its base to its minimum diameter, > 1.41 served as a good prognostic indicator for surgery (Ruiz-Moreno *et al*. 2008). The THI represents the ratio between the anteroposterior and tangential, vitreomacular, tractional forces. A lesion whose diameter at any point equals that of its base would have been afflicted by significant tangential forces. The inference of this finding supports the theory that such forces are the main progenitors of macular holes. Owing to the small cohort and conflicting results from other OCT parameter studies, further studies are warranted to establish whether the afore-mentioned measurements are necessary.

Occasionally, fluorescein angiography has been used to differentiate the lesion from cystoid macular oedema or choroidal neovascular membranes. However, its use as a diagnostic tool has been surpassed by the afore-mentioned, non-invasive OCT. Typically, full-thickness holes produce a non-expanding window defect (hyperfluorescence) in the early stages of the angiogram with no leakage or accumulation of dye. Dye exhibiting a 'flower-like' or petaloid appearance at the end stages of the angiogram is pathognomonic of cystoid macular oedema (Figure 8). Distortion of the retinal vasculature is the classic picture seen with epiretinal membranes on fluorescein angiography (Figure 9). The fact that pseudoholes secondary to epiretinal membranes may also produce a similar angiographic picture to their true counterpart underscores the limited use of this technique.

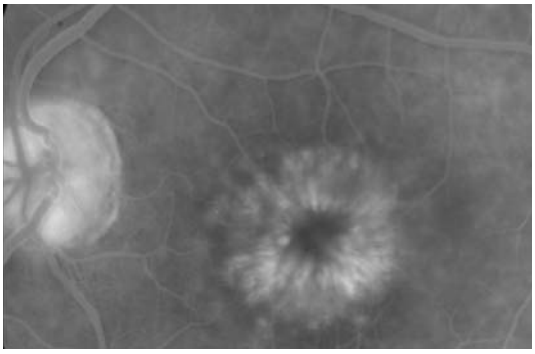

Figure 8. Fluorescein angiogram of lesion shown in Figure 2 demonstrating petaloid appearance.

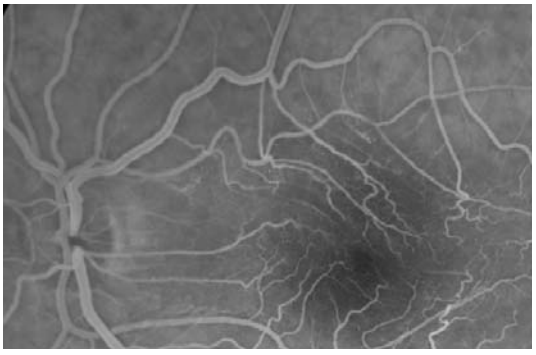

Figure 9. Fluorescein image of the lesion shown in Figure 1 demonstrating distortion of retinal vasculature.

Surgical Management

Prior to 1991, macular hole management was a low priority amongst retinal specialists. Today, the vitreoretinal community is very active in conducting research in this subject area. The main drive to this metamorphosis was the results from Kelly and Wendel's (1991) surgical technique to correct macular holes which resulted in anatomical closure and improvement of two Snellen lines of acuity in 58 and 42% of their patients respectively.

The principles of macular hole surgery are to relieve the anteroposterior and tangential tractional forces at the fovea (achieved via a vitrectomy and peeling of the internal limiting membrane and any associated epiretinal membrane) in addition to promoting apposition of the macular hole edges via an intraocular tamponade with either a gas or oil (Bainbridge *et al*. 2008). It has been suggested that the edges of the hole juxtapose as a result of the formation of a fibrin plug and intraretinal desiccation (Tornambe 2003). The latter is achieved through blockade of vitreous fluid through the hole. The techniques for achieving this will be discussed later.

The appositional process has been demonstrated microscopically and involves preretinal glial cell proliferation composed of glial cells and Müller cells (Madreperla *et al*. 1994; Miller *et al*. 1986). In order that these glial cells may form a membrane to conjoin the edges of the lesion, it is important that there is a paucity of vitreous fluid within the vicinity and that the edges of the hole are not everted. Both of these findings have been negatively correlated with glial cell migration and successful hole closure (Schubert *et al*. 1997). Thus it is reasonable to assume that one of the requirements to achieving successful hole closure is to keep the macula dry.

In order to ascertain whether surgical intervention for stage 1 holes served to prevent the development into its full-thickness counterpart, a multicentre clinical trial was undertaken by the Vitrectomy for Prevention of Macular Hole Study Group. The investigators concluded that the surgical complication risk outweighed the benefits and, as such, recommended monitoring only (de Bustros 1994). This is perfectly reasonable since most stage 1 holes resolve spontaneously.

Although the surgical outcomes of stage 2 holes are favourable, both the Moorfields Macular Hole Study (MMHS) and Vitrectomy for Macular Hole Study (VMHS) groups demonstrated an indubitable benefit of surgery for stage 3 and 4 holes (Ezra and Gregor 2004; Freeman *et al*. 1997). In the MMHS, the overall anatomical closure rate was 81 versus 11% in the observation group. In the VMHS, the anatomical closure at 6 months was 69% in those who underwent surgery compared to 4% randomised to observation alone.

Vitrectomy

Meticulous removal of the posterior cortical vitreous via a pars plana vitrectomy is an essential step in relieving the tractional forces exerted on the fovea. Traditionally, this has been achieved using the 20-gauge instrumentarium. This is the largest of the vitrectomy systems. An increasing number of surgeons are utilising the smaller 23- and 25-gauge systems. The 25-gauge system represents the smallest of the transconjunctival vitrectomy systems. Although the 25-gauge instrumentarium has been employed in macular hole surgery, proponents of the 23-gauge system argue that, owing to higher flow rates and its greater tensile strength, a faster vitrectomy with a reduced risk of instrument breakage during ocular movements can be achieved with this system (Lott *et al*. 2008).

Whereas the 20-gauge system requires sutures, its smaller counterparts do not. Sutureless surgery affords distinct benefits to both the patient and the surgeon. Benefits to the patient include a reduction in inflammation (with a concordant reduction in recovery time) together with a reduced likelihood of postoperative suture-related astigmatism. A reduction in operative time may be achieved with this technique, thus benefiting the surgeon. The advantages notwithstanding, the majority of vitreoretinal surgeons in the UK are employing the 20-gauge system (Khan *et al*. 2009).

Removal of the internal limiting membrane

Peeling of the internal limiting membrane has been advocated as an essential component of macular hole surgery with regard to achieving successful hole closure (Tognetto *et al*. 2006) and as a preventive measure for the reopening of holes (Kumagai *et al*. 2004). Notwithstanding, the exact mechanism underlying the success remains equivocal. Some investigators argue that it is a result of a concomitant reduction in vitreoretinal, tangential traction (Tognetto *et al*. 2006) while others support the notion that glial proliferation is stimulated, resulting in an increased macular hole closure rate (Uemoto *et al*. 2004).

Although internal limiting membrane peeling appears to improve the anatomical closure rate in most studies, some have reported it to be negatively correlated with visual outcome. It has been suggested that excessive attempts to remove the membrane may enhance anatomical closure through the promotion of glial cells at the expense of damage to the inner retinal elements (Smiddy *et al*. 2001). Since this is a challenging manoeuvre, most surgeons employ an intraocular dye in order to enhance visualisation of the internal limiting membrane, thus reducing the inadvertent damage to the underlying nerve fibre layer.

The dyes currently in use are trypan blue and indocyanine green (ICG). Concerns have been raised regarding the use of ICG owing to its ability to induce phototoxic injury to the retinal pigment epithelial cells (Sippy *et al*. 2001). In order to reduce the risk, the concentration of the dye is reduced to 0.05%. In a small randomised control trial comparing trypan blue with ICG 0.05%, there was no statistically significant difference in visual outcome between the two groups. However, there was a higher rate of small, persistent scotomata in the ICG group (Beutel *et al*. 2007).

Intraocular tamponade

An intraocular tamponade is inserted after performing a vitrectomy and internal limiting membrane peel and has several putative roles. To recapitulate, it may facilitate apposition of the neurosensory retina and prevent intraretinal hydration by preventing subretinal migration of fluid from the vitreous (Tornambe 2003). Notwithstanding, the exact mechanism by which the tamponade achieves closure of macular holes is debatable.

Some investigators support the theory that the bubble has a 'waterproofing' effect (Tornambe 2003) while others maintain its positive effects are via direct mechanical pressure (Thompson *et al*. 1996).

Proponents of Tornambe's 'hydration hypothesis', whereby the reduction in intramacular fluid via the retinal pigment epithelial pump is an essential component to successful anatomical closure, support the notion that the endotamponade should be large enough so as to ensure desiccation of the macular area. Failure to prevent the newly formed postvitrectomy aqueous humour from coming into contact with the hole during a critical postoperative contact period may interfere with the afore-mentioned reparative process. It is for this reason, in addition to the supposition that the perpendicular forces may either displace subretinal fluid away from the macula and provide counterpressure to the tractional forces (Berger and Brucker 1998), that face-down or prone posturing has been advocated for many years. The latter concept is often referred to as the 'flotation force effect'.

It is worthy of note that the flotation force effect may only be present if the gas bubble is immersed in fluid. Furthermore, a bubble that is above a fluid level exerts no buoyancy forces (Stopa *et al*. 2007). The main forces present are those at the gas–retina interface which are not dependent on posture. The corollary of this is that, in an eye which has a large gas fill in the immediate postoperative period (with a paucity of newly formed vitreous fluid), any buoyancy forces are negated. With these forces negated, there is a concomitant decline in the need for prone posture.

Silicone oil, which is hydrophobic and significantly less buoyant than its gaseous counterparts, lies anterior to the fovea in the upright position, as confirmed by OCT (Kokame and Yamamoto 2004). The fact that it has been effectively employed as an endotamponade in patients with macular holes not maintained in the face-down position (Goldbaum *et al*. 1998) provides further evidence to refute the flotation effect theory.

As mentioned previously, glial proliferation is an essential component to the repair process. It has been conjectured that the endotamponade, although not stimulating the glial cells directly, may serve as a scaffold along which these cells may proliferate and form a bridging preretinal membrane required to reapproximate the edges of the hole (Smiddy *et al*. 2001).

The intraocular tamponade utilised may be a gas or silicone oil. The gases commonly used are sulphur hexafluoride (SF6), hexafluorohexane (C2F6) and octafluoropropane (C3F8). The latter occupies the vitreal cavity for the longest period, with complete absorption by 8 weeks. The typical maximum absorption times for C2F6 and SF6 are 4 and 2 weeks respectively. Air affords the shortest absorption time (less than 1 week). Although longer-acting gases have been advocated, their possible

advantages are often counterbalanced by the adverse visual effects when performing activities of daily living. Furthermore, patients must not travel by air during this time period. This is due to the fact that the lower atmospheric pressure at high altitude causes the gas bubble to expand. This, in turn, may lead to a significant increase in IOP and an increased risk of developing a central retinal artery occlusion.

Traditionally, patients were instructed to adopt the prone posture immediately after surgery in order to facilitate the endotamponade. Typically, patients are requested to maintain prone posture for at least 50 minutes per hour of the day for at least a week. Not only is this difficult for patients to execute for the instructed time, Verma *et al*. (2002) discovered that such positioning was only maintained in an average of 38% of patients in their study group without adversely affecting visual and anatomical outcomes. More importantly, prone posturing is in itself not without its complications. Both ulnar neuropathies (Holekamp *et al*. 1999) and acute intraocular pressure spikes (Gupta 2009) have been reported. It also causes a deterioration in the patient's quality of life and, as such, often precludes some patients from undergoing surgery. Finally, for those who are still eligible, work becomes impossible, thus incurring significant economic implications.

Numerous studies pervade the literature supporting the obviation of prone posturing in patients undergoing macular hole surgery. Anatomical closure rates with the 20-gauge instrumentaria combined with phaco-emulsification of the crystalline lens and vitrectomy (phacovitrectomy) vary between 85 and 96.7% (Madgula and Costen 2008; Simcock and Scalia 2001; Tornambe *et al*. 1997; Tranos *et al*. 2007). Similar success rates were achieved without combined phacoemulsification in patients instructed not to adopt the prone posture (Tranos *et al*. 2007).

The author conducted a non-randomised, observational, retrospective trial during the period of September 2007–September 2008 at the Calderdale Royal Hospital, Halifax. Data were collected from 40 eyes from 39 consecutive patients who underwent transconjunctival, sutureless, 23-gauge vitrectomy, phacoemulsification, internal limiting membrane peel and intraocular gas tamponade (16% C2F6) for stage 3 and 4 idiopathic macular holes by a single surgeon. Macular holes were flat-closed in 37 (92.5%) eyes at the first attempt. The remainder were eventually closed with silicone oil without the need for face-down posturing. Postoperatively, 55% of patients achieved greater than two lines of improvement in Snellen visual acuity and 30% of patients achieved one line of improvement in Snellen visual acuity. The remainder of the patients' visual acuities remained stable. There were no intraoperative complications.

As mentioned previously, silicone oil has also been used as the tamponade, especially in those patients who are unable to maintain the prone posture. Providing there is an optimal fill in the vitreal cavity, successful closure can be obtained with success rates of 80–97% (Goldbaum *et al*. 1998; Karia *et al*. 2001). However, anatomical closure does not necessarily correlate with an improvement in visual acuity and this may reflect outer retinal toxicity via the oil (Bainbridge *et al*. 2008). Indeed, the manufacturers of the new high-density oils recommend that they are removed within 3 months to reduce the likelihood of inducing toxic effects.

Complications

Intraoperative complications include retinal detachments, surgically induced retinal tears and failure to close the hole at the first attempt. There is also a small, but nevertheless significant, risk of acquiring a retinal detachment in the postoperative period.

Postoperative intraocular pressure may increase secondary to gas bubble extrusion into the anterior chamber or blockage of the anterior-chamber angle by silicone oil. Conversely, the eye may become hypotonous (intraocular pressure < 6mmHg). This is more likely following sutureless surgery in the presence of a wound leak.

Cataract

Most intraocular procedures are cataractogenic. Indeed, the fact that cataract is the most common postoperative complication of macular hole surgery (Ellis *et al*. 2000) warrants further discussion. Indeed, this fact was eloquently highlighted in Thompson *et al*'s (1995) study in which 71% of eyes that underwent such surgery required cataract extraction within the first 2 years following their initial procedure. It is also noteworthy that the incidence of posterior subcapsular lens opacities was higher in those patients who did not posture compared to those who did following macular hole surgery in Tranos *et al*'s (2007) study. Two further points regarding cataract surgery performed as a separate procedure merit discussion. Firstly, due to a paucity of vitreous support in a vitrectomised eye, there is an increased risk of insult to the lens zonules and posterior capsule. Secondly, there are numerous studies that highlight the increased reopening rate of macular holes following separate cataract extraction (Duker *et al*. 1994). It is for these reasons that

an increasing number of surgeons are combining macular hole surgery with phacoemulsification (phacovitrectomy). It may also be possible that a combined procedure affords a more prodigious gas fill which, as alluded to earlier, may enable preservation of a dry macula without the need for prone posturing.

The posterior capsular opacification rate within 3 months of the procedure in our cohort was 12.5%. Simcock and Scalia (2001) noted their rate to be slightly higher, at 30%. Others have found lower rates. It has been purported that the underlying mechanism is related to the extended time that the gas is allowed to make contact with the posterior capsule. The same authors emphasised that, although a combined posterior capsulotomy may reduce the risk of this complication, this benefit may be negated by the increased risk of the gas bubble entering the anterior chamber.

Eckardt *et al*. (2008) have recently demonstrated that macular holes remain closed after 24 hours of short-acting endotamponades such as air. Although further studies are warranted before this technique is widely employed, the use of these more ephemeral agents may reduce the rate of posterior capsular opacification.

Surgical Outcomes

Anatomical closure and visual improvement rates can be expected in 90 and 70% of surgically treated idiopathic macular holes respectively either with or without postoperative prone posturing. It is noteworthy that successful anatomical outcome does not always equate to an equivalent improvement in visual acuity. Visual recovery has a relatively protracted time course and improvements have been reported years later (Leonard *et al*. 1997).

Unsurprisingly, stereoacuity has been found to be reduced with unilateral macular holes. One study has shown a demonstrable improvement in stereopsis following surgical correction of unilateral holes (Hikichi *et al*. 2001). In order to assess the practical effects of reduced stereopsis, Mireskandari *et al*. (2004) examined the stereoacuity and motor fusional reserves and assessed the effects of activities pertaining to daily living such as driving or pouring liquid into a glass relevant to this visual function, on patients receiving macular hole surgery without lens extraction. Interestingly, although patients were aware of a unilateral visual defect, only one from their cohort of 17 actually reported a subjective awareness of difficulty in performing binocular tasks The authors conjectured that this may be due to the relatively long timescale before surgery (mean 14.5 months) allowing a relatively protracted period of adaptation. Notwithstanding such a slight improvement in stereoacuity in half of their postoperative, macular hole cohort, the investigators reported substantially reduced stereoacuities compared to patients artificially rendered monocular through spectacle lens blur. This suggests that the blur associated with retinal pathology has greater ramifications for binocular function than induced blur. Furthermore, the deleterious effects on binocular function may be positively correlated with the duration of the retinal pathology. This has been demonstrated in patients with long-standing keratoconus (Sherafat *et al*. 2001). If this is the case, then earlier surgical intervention may lead to an increase in binocular restoration. Further studies are warranted to investigate this postulation and this should involve patients receiving combined phacoemulsification since lens opacities, whether pre-existing or surgically induced, are a significant, confounding variable to such an investigation.

Historically, visual acuity measurements and rates of anatomical closure have been the major outcome parameters utilised by clinicians in assessing the success of macular hole surgery. Moreover, preoperative visual acuity is often the only subjective measure that surgeons utilise prior to counselling patients for surgery. An informative measure is to assess the visual quality of life. One study evaluated the success of macular hole surgery in 59 patients utilising the National Eye Institute 25-item Visual Function Questionnaire (VFQ-25) conducted before and after surgery (Hirneiss *et al*. 2007). The authors concluded that surgery leads to an improved quality of life despite the fact that the fellow eye is unaffected. Moreover, they noted that a low preoperative VFQ-25 score and visual acuity scores were correlated positively with an improved benefit of surgery.

Referral Guidance for the Optometrist

This article has highlighted the incontrovertible benefits of macular hole surgery. As a result of refinements to the surgical technique, one can expect anatomical closure at the first attempt in over 90% of patients. Despite a paucity of prospective, randomised control trials comparing the anatomical and functional outcomes of patients undergoing surgery with and without prone posturing using the same surgical techniques and endotamponade, there is increasing evidence supporting the abrogation of this positioning in the first few days postoperatively (Gupta 2009). However, optometrists should execute a degree of caution when mentioning this fact to patients as a significant number of surgeons nationwide are still advocating prone posturing (Khan *et al*. 2009). It is therefore advised that optometrists liaise with their local ophthalmologists in order to establish their modes of practice.

It should also be borne in mind that surgical outcomes are dependent on both the stage and duration of symptoms but not the age of the patient (Bainbridge *et al*. 2008). In one review of surgical outcomes in macular hole surgery, closure rates within a year of onset of symptoms compared to those waiting more than a year were 94 and 47.4% respectively (Jaycock *et al*. 2005). The likelihood of achieving a good visual result is greatest if surgery is conducted within 6 months following the onset of visual symptoms. That said, there are reports in the literature that demonstrate positive visual outcomes beyond this timescale (Stec *et al*. 2004). It is the author's opinion that patients who have harboured holes for 18 months or more are unlikely to achieve an improvement in visual acuity postoperatively. As alluded to earlier, establishing the timescale of hole formation is inherently difficult. For some, it is the result of a serendipitous finding by the optometrist during a routine eye examination. Others are unable to ascertain accurately the onset of their symptoms. It is for this latter reason that macular studies are subject to recall basis when attempting to address the longevity of macular holes.

In conclusion, patients diagnosed with, or suspected of harbouring a macular hole should be routinely referred if they are willing to have surgery or if the diagnosis is in doubt. Lesions resembling choroidal neovascular membranes should be referred within 1 week. Annual examinations are recommended for patients who have acquired a hole in one eye and in whom a PVD has not occurred in their fellow eye. They should be advised to attend their practitioner sooner if they develop adverse visual symptoms.

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Multiple Choice Questions

This paper is reference C-11797. Three points are available for optometrists. Please use the inserted answer sheet. Copies can be obtained from Optometry in Practice Administration, PO Box 6, Skelmersdale, Lancashire WN8 9FW. There is only one correct answer for each question.

1. What is the approximate prevalence of idiopathic macular holes?
(a) 1 per 1000 people
(b) 2 per 1000 people
(c) 3 per 1000 people
(d) 4 per 1000 people

2. Which of the following is true regarding macular holes?
(a) The majority are traumatic in origin
(b) Males are more frequently affected
(c) They are always unilateral
(d) Patients can be asymptomatic

3. The disruption of which group of retinal cells can result in a foveal schisis or cyst?
(a) Bipolar cells
(b) Müller cells
(c) Amacrine cells
(d) Photoreceptor cells

4. You notice a macular hole with yellow deposits at its base. There is a Weiss' ring in the vitreous. What is the stage of hole?
(a) Stage 2
(b) Stage 4
(c) Stage 3
(d) Stage 1

5. With regard to the theories underlying the pathophysiology of holes, which statement is false?
(a) Unaffected eyes with posterior vitreous detachment are incapable of acquiring a hole
(b) The exact mechanisms are not completely understood
(c) Tractional forces have been implicated both tangentially and in an anteroposterior direction
(d) Vitreous fluid access has also been suggested as a risk for hole progression

6. All of the following lesions may imitate macular holes except which one?
(a) Epiretinal membrane
(b) Klein's retinal tags
(c) Cystoid macular oedema
(d) Lamellar holes

7. Which one of the following would be the single most reliable test in establishing the diagnosis of an equivocal macular hole that could be available in optometric practice?
(a) Watzke–Allen test
(b) Visual acuity
(c) Amsler chart
(d) Ocular coherence tomography

8. In what percentage of patients did Kelly and Wendel achieve a two-line improvement in visual acuity following macular hole surgery?
(a) 42%
(b) 50%
(c) 58%
(d) 65%

9. Which of the following hole measurements confers the worst prognosis?
(a) A minimum diameter of 280μm
(b) A base diameter of 400μm and minimum diameter of 380μm
(c) A base diameter of 400μm and minimum diameter of 200μm
(d) A base diameter of 550μm and minimum diameter of 380μm

10. Regarding macular hole surgery, which of the following statements is true?
(a) Anatomical closure is observed in approximately 50% of patients
(b) It is unsuitable for stage 1b holes
(c) A gas is utilised as the intraocular tamponade in all cases
(d) Successful closure is negatively correlated with increasing patient age

11. Regarding endotamponade, which of the following statements is false?
(a) It may prevent intraretinal hydration
(b) It may serve as a scaffold for proliferating glial cells
(c) In general, the gases used to achieve anatomical closure occupy the vitreal cavity for 2–8 weeks
(d) Long-acting gases must be removed surgically to expedite visual recovery

12. An 80-year-old female who underwent unsuccessful macular hole surgery to her right eye presents with a 1-day history of blurred vision in her left eye. Her acuities are R 6/60 and L 6/24. Other findings are: Watzke–Allen negative, metamorphopsia on Amsler and an ill-defined lesion in the left macular area. She does not want further surgery. What is the best management?
(a) Monitor only as patient does not want surgery
(b) Refer within 1 week
(c) Routine referral
(d) Refer to a colleague with a special interest in low-vision aids

13. Which of the following statements regarding complications of macular hole surgery is false?
(a) Cataract is the commonest postoperative complication
(b) Posterior capsulotomies are performed to prevent posterior capsule opacification following phacovitrectomy
(c) Occasionally retinal detachment can occur postoperatively
(d) Gas bubble extrusion into the anterior chamber can cause an increase in intraocular pressure

14. Which of the following statements regarding macular hole surgery is incorrect?
(a) Prone posturing is no longer required to achieve anatomical and functional successes
(b) Peeling of the internal limiting membrane is associated with an improvement in anatomical closure rates
(c) Binocular function maybe improved following surgery
(d) Radial nerve palsies have been associated with prone posturing

15. Which of the following statements is correct?
(a) In the UK, sutureless vitrectomy is commonly employed
(b) The anatomical closure rate is double the rate of visual improvement postsurgery
(c) The success rate is inversely proportional to the timing of the hole
(d) Detection of a hole requires an urgent referral

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Intraocular Scatter and Visual Performances

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Introduction

In the last two decades, techniques to measure and correct for refractive errors have reached unsurpassed levels (spectacles, contact lenses, intraocular lenses, laser surgery) to the point where customised correction of ocular aberrations can be foreseen. In this context, aberrometers are now becoming a popular tool for assessing the optical quality of the eye. Aberrations are however only one of the two basic optical phenomena affecting our vision. Even in an aberration-free eye, there will be inhomogeneities within the eye that will scatter light (Figure 1). This scattered light will not contribute towards the 'normal' formation of the image and thus will degrade vision. In a young and healthy eye, ocular scatter is low, but in some circumstances (eg old age, cataract, post refractive surgery) its consequences on the quality of vision can be significant. The purpose of this article is to review what is known about intraocular scatter, its link with visual performance and the information that can be extracted from scatter and glare measurements.

What is Light Scatter and What are the Sources of Scatter in the Human Eye?

As light passes through the structures of the eye, it can be deviated from its trajectory due to the presence of inhomogeneities. If the angle of deviation is less than 90° and the light reaches the retina, we refer to it as forward scatter (FWS). If the angle is larger, ie if the light is scattered in the opposite direction, then we refer to it as backscatter (BWS). FWS directly affects the quality of the retinal image, in contrast to BWS.

The amount of light scattered is proportional to the intensity of the incoming light and its angular distribution depends on the scattering source (size, shape, refractive index, spatial distribution). In the normal human eye, FWS represents the 1–2% of incident light that falls outside the point it should reach on the retina. This scatter is mainly due to the cornea, lens and fundus and their respective contributions are approximately 30%, 40%, 30% (Vos 1984). Scatter by the fundus is mainly sideways and is modulated by the pigmentation. In addition, the iris and outer coats are not entirely opaque and their contributions might be relatively significant for lightly pigmented eyes. Scatter by floating particles in the vitreous is negligible.

What is the Difference Between Scatter and Glare?

As defined previously, scatter is a purely optical phenomenon that depends on the wavelength of the incident light, and the geometrical and optical characteristics of the scattering structure. Glare, on the other hand, refers to a subjective sensation, as defined by the Commission Internationale de l'Eclairage (CIE): http://www.cie.co.at/index_ie.html): 'visual conditions in which there is excessive contrast or an inappropriate distribution of light sources that disturbs the observer or limits the ability to distinguish details and objects'.

The CIE differentiates between two types of glare: discomfort and disability (though in the literature other differentiations have been made: distractive glare, dazzling glare, adaptation glare). Disability glare has the effect of reducing some aspect of visual performance, such as contrast sensitivity, while discomfort glare provokes a disagreeable sensation but does not necessarily reduce performance.

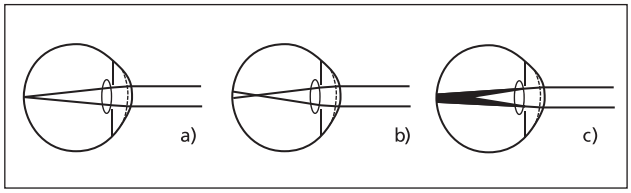

Figure 1. The two optical phenomena affecting our vision: aberrations and scattering. (a) Emmetropic eye; (b) myopic eye: the light is not focused in the correct plane; (c) intraocular scatter: light is correctly focused but spread by inhomogeneities.

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With regard to discomfort glare, it is clear that scatter will be only one possible factor next to others, such as increased sensitivity to normal straylight or dynamic of adaptation to changing ambient light levels. In contrast, scatter is the main cause for disability glare (Michael *et al*. 2008), but the two should not be seen as synonymous. Disability glare can be influenced, for instance, by aberrations and its consequences will depend on the spatial frequencies of the object observed (Aguirre *et al*. 2007). Neural inhibition may also play a role when the glare source is close to the line of sight (Vos 2003a).

To clarify further the relation between scatter and disability glare, it is worth discussing the relation between scatter and the notion of veiling luminance. When a glare source is in the field of view, the light from this source will be scattered over the retina, thus reducing the contrast threshold. It is then usual to consider the problem in terms of a veiling luminance, ie the luminance of a uniform background that could replace the glare source and produce the same reduction in contrast threshold. Some sophisticated expressions, developed by the CIE, can be used to calculate this veiling luminance, as a function of different parameters (eg illuminance in the plane of the pupil due to the glare source, its eccentricity, scattering properties of the eye). These expressions are empirical and only describe the subjective relationship between glare source and veiling luminance.

Early Research on Scatter and Glare

Glare has long been recognised as a cause of visual problems. Vos in a review (2003b) referred to Goethe's description of subjective haloes, and suggests that this was an early postulation of a neural basis for glare. Purkinje was credited with ascribing glare solely to light scatter, whilst Helmholtz reported two possible mechanisms: a nervous and a physical explanation. However, it is really at the beginning of the 20th century that scientists began to investigate systematically and separate the optical (ie scatter) and neural nature of disability glare (Cobb 1911; Stiles 1929).

What is Rayleigh or Mie Scatter?

The names of these two physicists are often encountered when reading about light scattering. Mie scattering refers to an analytical solution of Maxwell's equations for the scattering of electromagnetic radiation by spherical particles. Rayleigh scattering refers to a reduced version of the Mie solution which is only valid when the wavelength of light is very small with respect to the scattering particles. Because Rayleigh and Mie scattering refer to particles of different size, the associated scattering profiles are different. With Rayleigh scattering, BWS and FWS components are approximately equal, whereas FWS dominates with Mie scattering (Figure 2). In addition, Rayleigh scattering presents strong wavelength dependence ($1/\lambda^4$) when Mie scattering is almost wavelength-independent. A good example is the blue colour of the sky. When looking away from the sun, the sky appears blue and not black because shorter wavelengths (blue) are more scattered towards the observer than longer wavelengths (red). The phenomenon is even more dramatic at sunset when the light path is much greater, hence creating a wider gradient between blue and red light. If the composition of the atmosphere is changed, for instance, if the air is saturated with large particles such as water droplets, then Mie scattering dominates and the phenomenon appears wavelength-independent, eg white clouds, fog, white colour of cigarette smoke after inhalation.

Within the eye, due to the variety of scattering sources (in terms of size, refractive index difference with the surrounding media) the total intraocular light scatter distribution is complex and can be seen as a combination of Rayleigh–Mie scattering.

What is the Relation Between Forward and Backward Scatter?

In the human eye, back-scattered light is not well correlated with forward scattered light (de Waard *et al*. 1992). Depending on the source of scatter there may be a good relation (Yager *et al*. 1993), but, in general, there is usually more FWS than BWS (Atchison and Smith 2000; Bettelheim and Ali 1985). This is probably because the sources of scatter tend to be significantly larger than the wavelength (ie Mie scattering dominates: Atchison and Smith 2000; Wooten and Geri 1987) and the fact that intraocular scatter is complex, with light possibly scattered several times.

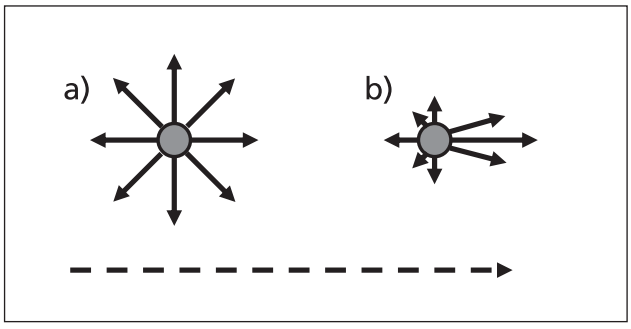

Figure 2. (a) Rayleigh scattering; (b) Mie scattering. The direction of incident light is depicted by the dashed arrow.

This absence of correlation is unfortunate because FWS is difficult to measure objectively and it is clinically the most significant. FWS affects directly the quality of the retinal image when BWS mainly reduces the amount of incident light. The image observed in a slit-lamp biomicroscope is a combination of BWS and specular reflection (depending on the method of illumination used) and thus cannot be used to assess the amount of FWS.

What are the Consequences of Forward Light Scatter on Visual Performances?

Contrast sensitivity

As stated previously, light propagating in the eye will be scattered by inhomogeneities and this scatter can be backward (and observable through a slit-lamp biomicroscope) or forward. BWS will mainly reduce the amount of light reaching the retina. In contrast, by spreading the light on the retina, FWS will reduce the contrast (both chromatic and monochromatic). This is illustrated in Figure 3 and is the main reason, with glare, why scatter degrades visual performance.

Although there is a good correlation between FWS and contrast sensitivity loss, the contrast sensitivity loss is not directly proportional to the amount of scatter and thus cannot be seen as a good measure of it. Furthermore, contrast sensitivity loss will vary with the spatial frequencies of the observed target (Aguirre *et al*. 2007; Barbur *et al*. 1999). The best correlation between reduction of contrast sensitivity and scatter will be obtained in the presence of a glare source (eg use of the Pelli–Robson chart together with the Brightness Acuity Tester (Marco Ophthalmic, USA): Elliott and Bullimore 1993).

Night driving

Driving at night involves challenging lighting conditions (poor illumination, numerous glare sources) where contrast sensitivity is particularly important. In addition, glare has been reported as a risk for road accidents (see van den Berg *et al*. 2003; Babizhayev 2003 for review). Since glare is directly related to scatter, testing the amount of intraocular light scatter could provide useful information on the night-driving ability of patients who are at risk of suffering from disability glare (eg patients over 50, patients with early signs of cataract).

In this context several tests have been developed, usually based on a measure of mesopic contrast sensitivity or glare sensitivity. The best-known ones are perhaps the Nyktotest (Rodenstock, Germany) and Mesotest (Oculus Optikgerate, Germany) that are designed to simulate night-driving conditions. However, if they do provide additional information with respect to a simple visual acuity test, the added value is difficult to appreciate as these tests have been shown to have a limited applicability (van Rijn *et al*. 2002, 2005). Driving is a complex activity, involving not only visual factors (eg visual acuity, scatter, dynamic of adaptation to changing light levels) but also motor and cognitive skills, which partly explains why straylight measurements, or contrast sensitivity tests with glare, do not correlate well with driving performance (Drum *et al*. 2007).

Currently, there is no agreement on which test or combination of tests is best suited to assess night-driving ability, and how to interpret the information in terms of threshold. Further research is required to develop a test that would become a standard part of the driving licence assessment. In the meantime, existing tests (eg Brightness Acuity Tester with Pelli–Robson chart, C-Quant (Oculus, Germany)) can provide useful information providing practitioners are aware of their limitations.

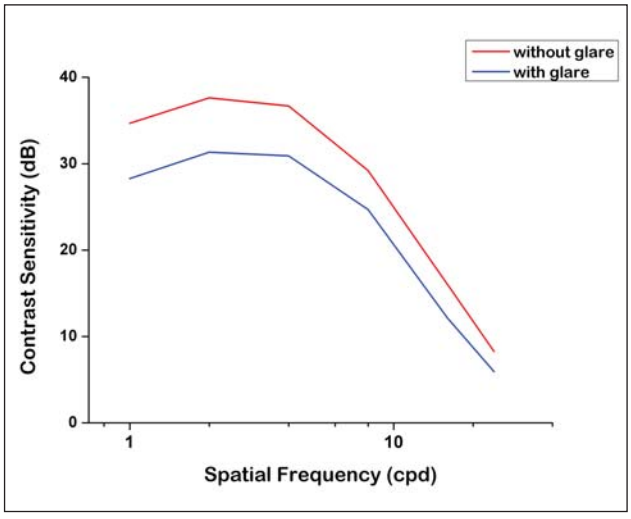

Figure 3. Changes in contrast sensitivity provoked by a glare source.

Haloes and corona

In some circumstances, and particularly when observing a small bright source, intraocular light scatter can give rise to two entoptic phenomena: bloom and flare. Bloom is when objects appear as if they were glowing, and corresponds to the disability glare previously discussed. The second phenomenon, flare, encompasses ciliary corona and lenticular halo (Hemenger 1992; Mellerio and Palmer 1972; Simpson 1953). The ciliary corona corresponds to a pattern of thin lines radiating from the centre of the source. It is caused by small variations (less than 2μm) in the refractive index of the ocular media (Ritschel *et al*. 2009; van den Berg *et al*. 2005) and is independent of pupil size.

Further away from the light source, the lenticular halo can be observed. It appears as concentric coloured rings (from blue in the inside to red towards the periphery). This phenomenon is due to the structure of the lens. Basically, the lens is composed of fibres oriented radially, similarly to the spokes of a wheel. At the centre of the lens, the fibres are densely packed and the refractive index appears relatively uniform but, towards the periphery, the spacing between the fibres increases and they act as a radial diffraction grating (Hemenger 1992; Mellerio and Palmer 1972). Since the lenticular halo is an entopic phenomena, the apparent size of the halo is constant and independent of the distance between the observer and the source.

A similar phenomenon occurs in corneal oedema (Caldicott and Charman 2002). The transparency of the cornea is related to its highly organised structure where collagen fibrils behave as a three-dimensional diffraction grating. If this structure is disrupted, a diffraction pattern similar to the lenticular halo can be observed. The two phenomena can be differentiated using a stenopaeic slit (Emsley–Finchman test). If the shape of the halo varies when the slit is passed in front of the eye, then it means the diffractive structure selected by the source varies, and this change corresponds to the varying orientations of lens fibres. The corneal halo is not affected by the position of the slit.

How Can we Measure Scatter?

Straylight has been studied for many years, and many techniques have been developed to try to quantify it (Aslam *et al*. 2007, Cobb 1911). The main difficulty is how to assess objectively and accurately FWS. BWS is directly observable but, as we have previously stated, its measurement provides little information on FWS, which affects the quality of the retinal image.

Until the mid-1980s, most techniques were based on a measure of visual acuity or contrast sensitivity in the presence of a glare source, from which light scatter had to be deduced, often requiring complex theoretical assumptions. For these reasons, none was accepted as a gold standard for straylight measurement. This was eventually deemed to be the straylight meter (van den Berg and Ijspeert 1992) that provided a measure of scatter rather than a measure of its consequences on visual performances.

The basics of the straylight meter are the following. The patient looks at the centre of a ring of light-emitting diodes (LEDs) (Figure 4). The image of the ring is formed on the retina of the patient, and, in the case of a perfect eye, this image would be perfect with no light in the centre of the ring. In the presence of scatter, some light will be scattered in the middle of the ring. Consequently, the amount of light in the centre of the ring is directly proportional to the intraocular scatter. Since the ring can be equated to a small source of eccentricity θ, the use of various rings allows measurement of the angular dependence of the intraocular scatter. In practice, there is also a spot source in the centre of the ring and the two sources are flickering in counterphase. The task of the observer is to adjust the luminance of the spot to cancel the flickering, ie to match the veiling illuminance produced by the scattered light. For this reason, this technique is referred to as 'direct compensation'.

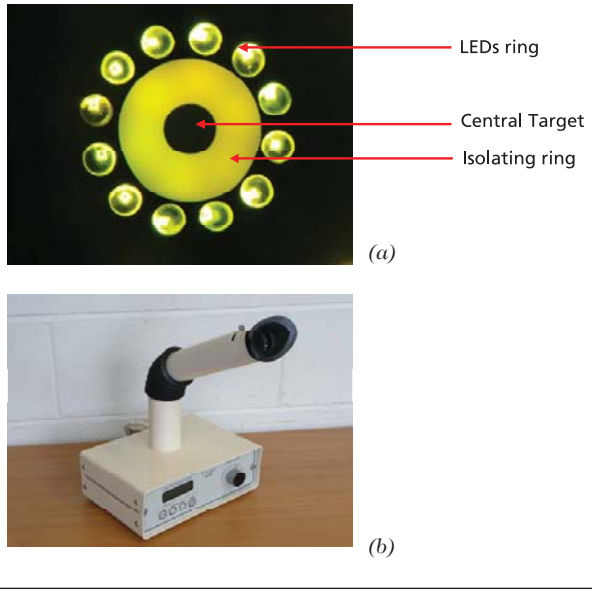

Figure 4. (a) Straylight meter; (b) arrangement of the central test patch and straylight sources inside the van den Berg straylight meter. LEDs, light-emitting diodes.

Despite its apparent simplicity, the use of the straylight meter can be confusing for untrained subjects and the measurements depend strongly on their cooperation and understanding of the task. To tackle these shortcomings, a technique was developed, called compensation comparison (Franssen *et al*. 2006). The commercial instrument based on this technique is the C-Quant. The new technique is different in that the central field is split into two halves (Figure 5) and the patient is submitted to a series of 25 choices (ie 'which half of the central field flickers the most?'). Because of this forced-choice method and the improved design, the C-Quant is easier to use than the straylight meter, the repeatability has been increased and an estimation of the reliability of the measure is provided.

Although the C-Quant is probably the best commercial instrument available to assess intraocular scatter, it presents various limitations. The measurement is still subjective and only one parameter is provided. This parameter does not provide any insight on the source of scattering. Poor subject positioning may allow errors of measurement to occur (Coppens *et al*. 2006a).

In this context, several attempts have been made in the last decade to develop a completely objective technique. Among these attempts we can list: a method based on the assumption that scattering in the eye depolarises light (Bueno *et al*. 2004); a method based on the analysis of Tscherning aberrometer images; and two approaches based on the use of Hartmann Schack (HS) aberrometers. The first one is based on a study of HS images (Donnelly and Applegate 2005; Fujikado *et al*. 2004; Mihashi *et al*. 2006). In this method, a degree of scatter is associated with the spread of light behind each lenslet of the HS. The second method is based on the comparison of two different measurements of the point spread function (PSF), one sensitive to scattering (double-path system), the other not (usually computed from the wavefront recorded by the aberrometer: Cox *et al*. 2003; Shahidi and Yang 2004). All these methods present different drawbacks (fundamental limitations, range of applications, expensive hardware) and, to our knowledge, none has formed the basis of any commercial instrument, with the exception of the OQAS (Visiometrics, Spain) (Gell *et al*. 2004), whose measure of scatter is based on the recording of the double-path PSF.

The OQAS seems thus to be an important milestone in the development of an instrument which provides a rapid, repeatable, accurate and objective measure of intraocular scatter. Although it is, with the C-Quant, the best alternative in the absence of an ideal instrument, the comparison between the two is difficult. The two measurements are based on different parts of the PSF (large angle scatter with the C-Quant, small angle scatter with the OQAS) and thus will relate differently to real-life performance.

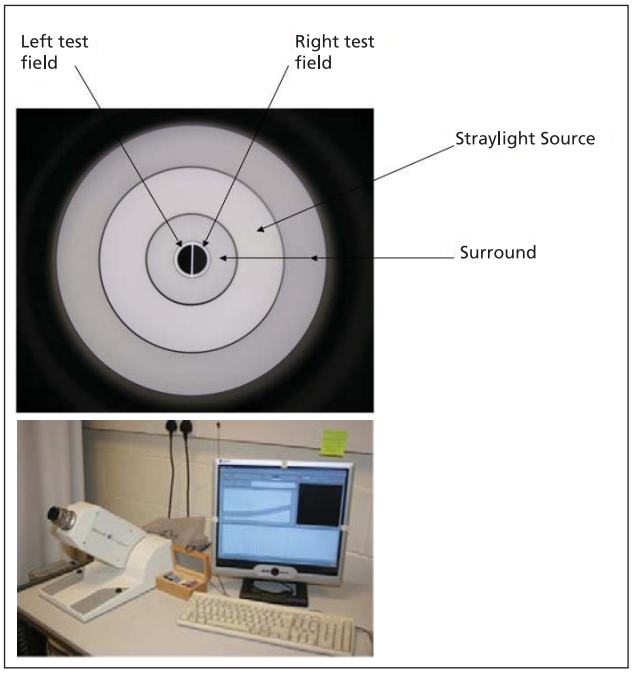

Figure 5. C-Quant instrument (bottom) and layout of stimuli for the compensation comparison method (top).

What is the Relation Between Intraocular Light Scatter and the Following Parameters?

Pathologies

The transparency of the cornea and lens is related to their structures, and, consequently, any pathology affecting their integrity (postsurgical changes, inflammation, scarring, swelling) will be a potential source of scattering. Increased level of scatter has been reported with corneal dystrophies (van den Berg *et al*. 1993), keratoconus and refractive surgery: photorefractive keratectomy (Neeracher *et al*. 2004; Veraart *et al*. 1995), laser in situ keratomileusis (LASIK), wavefront-guided LASIK (Schallhorn *et al*. 2008) and intra LASIK (Krueger *et al*. 2008), although some of these results were disputed (Nagy *et al*. 2002). Contradicting results were also reported with the use of contact lenses and corneal grafts. With respect to contact lenses, a possible source of scatter could be the presence of subclinical levels of oedema (Elliott *et al*. 1991). Regarding corneal grafts, a possible explanation for the discrepancy between studies is the fact that different methods and different groups were used (Brahma *et al*. 2000; Hindman *et al*. 2007; Patel *et al*. 2008a, b).

Scatter is also well known to be an issue associated with cataracts (de Waard *et al*. 1992; Franssen *et al*. 2006; van den Berg *et al*. 2007). Different types of cataracts will involve different scattering sources, eg inhomogeneities created by localised changes in protein concentration, development of protein aggregates (Bettelheim 1985; Hemenger 1992; Whitaker *et al*. 1993; Yaroslavsky *et al*. 1994), degradation of the cytoplasm within the lens fibres, disorganised fibre membranes and multilamellar bodies (Gilliland *et al*. 2004), which, in return, can affect differently the scatter profile. Cortical cataract will give rise to Mie scatter, due to the large size of the disruption, and will be pupil-dependent such that vision may improve in the presence of a glare source, while a dilated pupil such as when driving at night will give rise to symptoms of glare. Nuclear cataract will give rise to both Rayleigh and Mie scatter and will be troublesome regardless of pupil size. There is a significant correlation between visual acuity and straylight in this type of cataract (Strobel *et al*. 1990). Posterior subcapsular cataract will give rise to Mie scatter and will be most troublesome in small-pupil conditions such as in the presence of a glare source or near-vision tasks. Posterior subcapsular cataract has been shown to be the most significant cause of glare sensitivity (Lasa *et al*. 1992). Interestingly, surgery fails to bring BWS level back to normal (van den Berg *et al*. 2003) and this scatter will also increase due to posterior capsule opacification (Meacock *et al*. 2003). The amount of scatter can then be reduced by Nd:YAG capsulotomy but will still remain higher since in normal individuals, even when the posterior chamber is absolutely clear.

In addition to the cornea and lens, increased scatter can also be associated with pathologies of the uvea (uveitis and the associated aqueous flare), iris (eg iris hypopigmentation: van den Berg and Spekreijse 1986) and has been reported in patients with retinal disorders (Grover *et al*. 1998; Shahidi *et al*. 2005). In this last case, the consequences of the retinal image degradation due to scatter will be all the more dramatic since the overall vision performances are reduced by the pathology.

Age

Scatter increases with age, with a sharp rise from 40 years old (Hennelly *et al*. 1998; Ijspeert *et al*. 1990; van den Berg 1995: Figure 6). This is mainly due to increased scatter from the lens, where the continual growth and deposition of yellow lens proteins (Monnier and Cerami 1981) mean that the healthy eye has increasing Rayleigh light scatter with age. In addition there is also a small amount of scattering due to the increase in presence of the fluorescent compounds found in the lens (Elliott *et al*. 1993). The contribution of the cornea stays relatively constant.

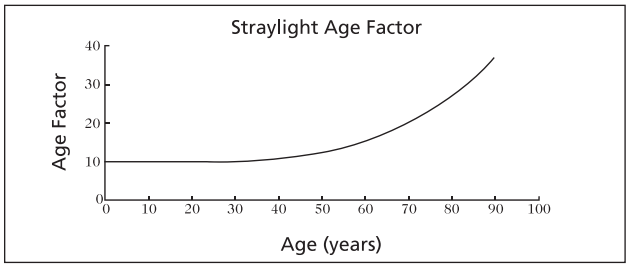

Figure 6. Age dependence of straylight.

Refractive correction

The amount of straylight produced by spectacle lenses that are reasonably clean and in good condition is not significant with respect to FWS (van den Berg *et al*. 2003). Extremely dirty or scratched glasses can produce straylight levels comparable with those of a healthy eye but then the problem can be easily addressed. For this reason, wearing refractive corrections when measuring FWS or glare sensitivity is not an issue providing the lenses are clean. Scatter/glare measurements are usually quite robust with respect to refractive errors but asking patients to remove their spectacles could only reduce the accuracy and repeatability of the measure. In a similar context, studies have shown that ocular lubricants such as artificial tears have no influence on scatter (Veraart and van den Berg 1992).

Among the different devices that exist for correcting refractive errors, it is worth mentioning multifocal lenses. These lenses are designed to have multiple focal points so, in one way, these lenses behave as a source of intraocular scatter. Even with new-generation lenses designed to reduce haloes (eg improved edge design with intraocular lenses, smoother transition between refractive zones), the superposition of a sharp image together with a blurred one will cause problems of straylight effects (glare, blur circle) for some patients.

Point spread function and aberrations

The PSF of the eye represents the image of a point by the eye. The more different it is from a point, the worse the optical quality of the eye. The optical phenomena affecting the size of the PSF are diffraction, aberrations and scattering (Figure 7). Aberrations and diffraction dominate the light distribution in the centre of the PSF (few arcminutes) whereas scatter represents the outer skirts. No information on scatter can be deduced from measurements of aberrations. In particular, aberrometers based on a Hartmann–Shack principle provide an estimate of the PSF that does not take into account the scatter and hence will overestimate the optical quality of the eye.

Figure 7. Simulation of the image degradation due to aberrations and scattering. (a) Original image; (b) blurred image; (c) degradation due to straylight only.

Wavelength

There has been some confusion as to whether there is a wavelength effect on intraocular straylight, brought about in part by confusion between excised lens studies, general scatter theory and psychophysical studies. Early workers assumed that Rayleigh-type scatter would be important and therefore a strong λ^4 dependence would be present (where λ is the wavelength of light).

In a recent study, Coppens *et al*. (2005, 2006b) examined straylight in distinct groups (age and pigmentation), across wavelengths ranging from 457 to 625nm. They found a λ^4 effect in highly pigmented young observers but not in lightly pigmented or older subjects. One explanation is that all eyes have strong wavelength dependence, but long-wavelength straylight is increased in lightly pigmented eyes, whilst lens aging increases straylight across all wavelengths.

As a result of the presence of differing mechanisms at work in differing groups (Whitaker *et al*. 1993), straylight can be seen, in the most part, as independent of wavelength (Vos 2003a; Wooten and Geri 1987).

Pupil size

If the source of scatter within the eye is homogeneous (eg the whole crystalline lens), then the pupil should have no influence (assuming no contribution through the iris). The physics of scattering does not change and the ratio between incident light and scattered light stays unaffected. This was confirmed experimentally by Franssen and coworkers (2007) who found that, for normal pupils, in the range 2–7mm, straylight weakly depends on pupil size. Consequently, photopic measurements should hold for scotopic/mesopic conditions. For accurate measurements however, pupil size needs to be taken into account, as with smaller pupil size the straylight caused by the translucency of the globe increases, and the properties of the scattering source can vary.

What can be done to Reduce Problems Associated with Intraocular Scatter?

The solution, if any, will depend on the source of scattering. For instance, scatter associated with posterior capsular opacification will be easily addressed with Nd:YAG laser posterior capsulotomy. In the same way, scatter associated with the healing process after corneal laser surgery will reduce with time or drugs. On the other hand, because the source of scatter is basically physiological discontinuities within the eye at an extremely small scale, there is no way to correct for it as is possible to do with aberrations. Another approach is then to use filters to reduce the sources of glare: antireflection coatings, polarising filter, tinted lenses.

Optically, tinted lenses can be useful as they reduce the amount of light entering the eye and possibly block harmful wavelengths. The predominant cause of disability glare however is FWS caused by Mie scatter, and therefore the reduction of the wavelength-dependent but isotropic Rayleigh scatter would be of limited help (Hayashi and Hayashi 2006). Filtering light with ultraviolet A blocker could however be of some benefit with regard to blue/green autofluorescence of the crystalline. This phenomenon is found in healthy eyes (Sparrow *et al*. 1992) but increases with age and is greatest in those with nuclear or mixed cataract. It was also reported in people with diabetes. As an increase in lens fluorescence and a decrease in lens transmittance can be delayed by good metabolic control, the determination of lens fluorescence could provide information about the long-term control of diabetes (Larsen *et al*. 1989).

Pharmacologic miosis can also help to reduce glare problems in cortical cataracts and the influence of aberrations.

Concluding Remarks

Although scatter is not synonymous with glare, glare is a direct consequence, with possibly a major influence on visual performance. Optometrists should thus, ideally, introduce a measure of scatter when examining 'at-risk' patients (eg patients over 55, patients with cataracts and history of corneal dystrophy) and more particularly patients complaining of difficulties with looking against the light, hazy vision, haloes at night, glare sensitivity or reduced (chromatic) contrast sensitivity.

Unfortunately, there is currently no instrument to measure accurately and objectively the angular distribution of intraocular FWS, no model to relate these measurements accurately to the physiological source of scatter, and no good metric to relate it to real-life visual performance.

Various tests however exist: straylight meter, C-Quant, OQAS, mesopic visual acuity, low-contrast acuity with glare. They have been reviewed extensively in the literature (Aslam *et al*. 2007; Drum *et al*. 2007; van den Berg *et al*. 2003). The Brightness Acuity Tester, used on medium setting, together with a Pelli–Robson or Mars contrast sensitivity chart, provides relatively good reliability and discriminability (Elliott and Bullimore 1993), together with a simple procedure and low cost. The straylight meter and its more modern version, the C-Quant, are often seen as the gold standard due to their superior repeatability and discriminative power, but the correlation between the parameter they measure and visual impact, or other vision tests, is not well established.

Despite their limitations, these instruments have their place in clinical practice. Patients complaining of poor vision often have difficulties in describing their impairment and such instruments could help in understanding these complaints and assist in making appropriate recommendations (eg against driving at night, or on indoor lighting). These instruments are also of importance to refine cataract referral, especially in patients with good high-contrast visual acuity, or for postsurgical evaluation of corneal procedures, such as LASEK, Descemet's stripping endothelial keratoplasty (DSEK) or penetrating keratoplasty.

Recently, the number of publications on measuring scatter and glare and developing an objective technique has increased significantly. This is possibly because of the good level of correction achieved for refractive errors, and problems of increased scatter associated with the development of corneal laser surgery. This rise in the number of studies has already produced interesting improvements (eg OQAS) and will hopefully be soon translated into a valid technique and commercial instrument.

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Multiple Choice Questions

This paper is reference C-11796. Three points are available for optometrists and dispensing opticians. Please use the inserted answer sheet. Copies can be obtained from Optometry in Practice Administration, PO Box 6, Skelmersdale, Lancashire WN8 9FW. There is only one correct answer for each question.

1. Which of the following tests provides an objective measure of intraocular scatter?
(a) Pelli–Robson contrast sensitivity chart
(b) Miller–Nadler glare test
(c) Brightness Acuity Tester
(d) C-Quant

2. Which of the following measurements does not provide a direct measurement of intraocular scatter?
(a) C-Quant
(b) Mesostest
(c) OQAS
(d) Straylight meter

3. A glare source, situated at 10° of the line of sight, produces 200lux in the plane of the pupil. What is approximately the corresponding veiling luminance?
(a) 20lux
(b) 10cd/m^2
(c) 20cd/m^2
(d) 40cd/m^2

4. Which of the following would not be associated with increasing intraocular scatter?
(a) An increase in aberrations
(b) A reduction in contrast sensitivity
(c) A degradation of the modulation transfer function
(d) An increase in glare sensitivity

5. In a young normal eye, the contributions to intraocular scatter for the cornea, lens, fundus and vitreous are respectively:
(a) 30% – 40% – 30% – 0%
(b) 30% – 70% – 0% – 0%
(c) 25% – 25% – 25% – 25%
(d) 50% – 40% – 5% – 5%

6. Which of the following statements regarding intraocular scatter is incorrect?
(a) With Rayleigh scattering forward and backward scatter are approximately equal
(b) Forward scatter is usually greater than backward scatter due to the large sizes of the scatter sources compared to the wavelength of light
(c) Backward scatter will reduce the contrast of the retinal image
(d) Loss of contrast sensitivity varies with the spatial frequency of the target

7. The veiling luminance produced by a glare source describes:
(a) The luminance produced by the glare source in the plane of the retina
(b) The luminance of a uniform field which when placed in the plane of the target would lead to the same contrast reduction as the glare source
(c) The luminance of a uniform field which when placed in the plane of the retina would lead to the same contrast reduction as the glare source
(d) The illuminance of a uniform field which when placed in the plane of the target would lead to the same contrast reduction as the glare source

8. Which of the following statements is true in the healthy eye?
(a) Scatter increases with the wavelength of incident light
(b) Scatter increases with the size of the pupil
(c) Scatter increases with the pigmentation of the iris
(d) Scatter increases with the age of the patient

9. Which of the following statements regarding disability glare is incorrect?
(a) It will reduce some aspect of visual performance
(b) It provokes a disagreeable sensation but does not reduce performance
(c) Light scatter is the main cause
(d) Its consequence depends on the spatial frequencies of the object observed

10. The Stiles–Holaday equation is only valid for the eccentricity of the glare source up to what maximum angle?
(a) 10°
(b) 20°
(c) 30°
(d) 40°

11. Which of the following statements regarding Rayleigh or Mie scatter is incorrect?
(a) Mie scattering refers to scattering of electromagnetic radiation by spherical particles
(b) Rayleigh scattering is valid when the wavelength of light is large with respect to the scattering particles
(c) Total intraocular light scatter is a combination of Rayleigh–Mie scattering
(d) Front scatter dominates with Mie scattering

12. Which of the following statements regarding light scatter and the point spread function (PSF) are incorrect?
(a) Light scatter does not affect the PSF
(b) Aberrations and diffraction dominate the light distribution in the centre of the PSF
(c) No information on scatter can be deduced from aberration measurements
(d) Hartmann–Shack aberrometers produce a PSF that do not take light scatter into account

13. Which of the following would not be expected to reduce glare symptoms?
(a) Nd:YAG laser posterior capsulotomy
(b) Corneal healing following laser treatment
(c) Aberration-controlled contact lenses
(d) Pharmacological miosis

14. Which of the following statements regarding night driving is incorrect?
(a) Contrast sensitivity is particularly important
(b) Glare is a risk factor for road accidents
(c) Early cataract can increase disability glare
(d) There are currently no tests available to simulate night driving conditions

15. Which of the following statements regarding forward scatter and refractive correction is incorrect?
(a) Scratched lenses produce straylight levels comparable to a healthy eye
(b) Spectacles should never be worn when measuring forward scatter
(c) Some patients will experience more glare with multifocal lenses
(d) Ocular lubricants have no influence on scatter

Multiple Choice Answer Sheet

Instructions

The MCQs for the review papers in this issue are adjacent to each paper. The answer sheet is divided into sections with the title of the paper clearly marked at the top of each section.

Indicate your answer by filling in the relevant answer box, e.g. ●. You do not have to answer the questions for every paper.

Make sure you fill in your address and, if appropriate, College membership number. Return this sheet to *Optometry in Practice* at the following address: **CET Editor, Optometry in Practice, PO Box 6, Skelmersdale, Lancashire WN8 9FW**

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| D | — | | | | |

1. Visual Acuity and Visual Satisfaction

	a	b	c	d
Question 1	0	0	0	0
Question 2	0	0	0	0
Question 3	0	0	0	0
Question 4	0	0	0	0
Question 5	0	0	0	0
Question 6	0	0	0	0

We value your feedback on this article. On a scale of 1 of 3, where **1= poor, 2 = average, 3 = good,** please rate this paper for:

Interest [ ]
Relevance to your work [ ]

2. Current Trends in Macular Hole Management

	a	b	c	d
Question 1	0	0	0	0
Question 2	0	0	0	0
Question 3	0	0	0	0
Question 4	0	0	0	0
Question 5	0	0	0	0
Question 6	0	0	0	0
Question 7	0	0	0	0
Question 8	0	0	0	0
Question 9	0	0	0	0
Question 10	0	0	0	0
Question 11	0	0	0	0
Question 12	0	0	0	0
Question 13	0	0	0	0
Question 14	0	0	0	0
Question 15	0	0	0	0

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Interest [ ]
Relevance to your work [ ]

3. Intraocular Scatter and Visual Performances

	a	b	c	d
Question 1	0	0	0	0
Question 2	0	0	0	0
Question 3	0	0	0	0
Question 4	0	0	0	0
Question 5	0	0	0	0
Question 6	0	0	0	0
Question 7	0	0	0	0
Question 8	0	0	0	0
Question 9	0	0	0	0
Question 10	0	0	0	0
Question 11	0	0	0	0
Question 12	0	0	0	0
Question 13	0	0	0	0
Question 14	0	0	0	0
Question 15	0	0	0	0

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Interest [ ]
Relevance to your work [ ]

4. The Structure–Function Relationship in Glaucoma: Implications for Disease Detection

	a	b	c	d
Question 1	0	0	0	0
Question 2	0	0	0	0
Question 3	0	0	0	0
Question 4	0	0	0	0
Question 5	0	0	0	0
Question 6	0	0	0	0
Question 7	0	0	0	0
Question 8	0	0	0	0
Question 9	0	0	0	0
Question 10	0	0	0	0
Question 11	0	0	0	0
Question 12	0	0	0	0
Question 13	0	0	0	0
Question 14	0	0	0	0
Question 15	0	0	0	0

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Relevance to your work [ ]

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Answers to multiple choice questions for Volume 10 Issue 2 will appear in the next issue of *Optometry in Practice.*

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