Movement detection threshold and ocular hypertension

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CLINICAL STUDY

Abstract

Purpose **To determine whether patients with ocular hypertension (OH) have elevated oscillatory movement displacement thresholds (OMDT) indicative of early neural damage.**
Methods **Evidence of early neural loss was sought using OMDT. The OMDT of 29 normotensive individuals were compared with those obtained from 44 untreated age-matched OH eyes (20 male, 24 female).**
Results **A mean OMDT at 15 sec arc at 40 years of age was obtained in normotensive individuals, the age trend increasing by about 4 sec arc per decade. About one-third of all ocular hypertensives (13 cases; 6 male, 7 female), who were dismissed without treatment, exhibited abnormal OMDT. An equal proportion of abnormal thresholds were observed in individuals in each decade, although the age trend diverges from that established for normotensives with increasing age. Mean OMDT for ocular hypertensives (37.1 sec arc) were significantly different ($t = 2.7$, $p < 0.007$) from the mean obtained from normotensives (22.2 sec arc).**
Conclusion **The results emphasise the need for more rigorous differentiation of OH using psychophysical techniques indicating early neural damage.**

Key words Ganglion cells, Glaucoma, Psychophysics

Glaucoma continues to represent the commonest cause of irreversible worldwide blindness[1] and attention remains focused on early diagnosis and treatment to minimise visual morbidity. Hence the management of patients with ocular hypertension (OH) and primary open angle glaucoma (POAG) remains a major part of the work of general ophthalmologists.

OH is defined as intraocular pressure (IOP) above the normal range, but with normal optic disc appearance and visual field indices. Generally patients with IOPs consistently above 21 mmHg are labelled as having OH. OH presents a continuing clinical challenge because of its relative frequency and increased risk of developing POAG. IOP continually exceeding 22 mmHg, without glaucomatous damage, is found in 1.6% of the population over 30 years of age and in 10.5% in the 70–79 year age group.[2] In such groups the risk of developing POAG over a 5 year interval varies from 3% to 5%, compared with 0.5% to 1.25% in a normotensive population.[3] OH management remains controversial. Prophylactic treatment may prevent significant optic nerve atrophy occurring before visual field losses are detected. Conversely many with OH never develop optic nerve damage during their lifetimes and neither surgical nor, necessarily protracted, medical treatment is completely free of risk. Early detection of glaucomatous atrophy in those with OH remains problematic, given that limited longitudinal studies have observed great variability in the initial appearance and progression of optic disc and nerve fibre layer abnormalities in such patients.

A number of studies have advocated techniques for early detection of glaucoma,[4–6] but particular drawbacks may limit their clinical usefulness. This includes conventional automated perimetry, which has a lack of sensitivity.[7] It would be of considerable benefit to detect early neuronal loss in glaucoma. Oscillatory movement displacement threshold (OMDT), the smallest movement of a given stimulus which gives rise to the perception of movement, offers an ability to detect early neural damage. Such thresholds fall into the category of hyperacuities and have been used to describe visual deficits in diabetes mellitus, optic neuritis, adult anisometropic and strabismic amblyopia, childhood amblyopia, and glaucoma even in cases where visual acuity was normal.[8–12] The present study compared OMDT in OH patients with thresholds obtained from normal controls.

Subjects and methods

Informed consent was obtained from all subjects in accordance with the tenets of the Declaration of Helsinki and the guidelines of the local ethics committee. OH patients were recruited from the University Hospital glaucoma clinic. The normal subjects were hospital employees or friends of the OH patients.

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Presented in part at the Royal College of Ophthalmologists Annual Congress, Glasgow, May 1998

Received: 15 June 2000
Accepted in revised form: 20 October 2000

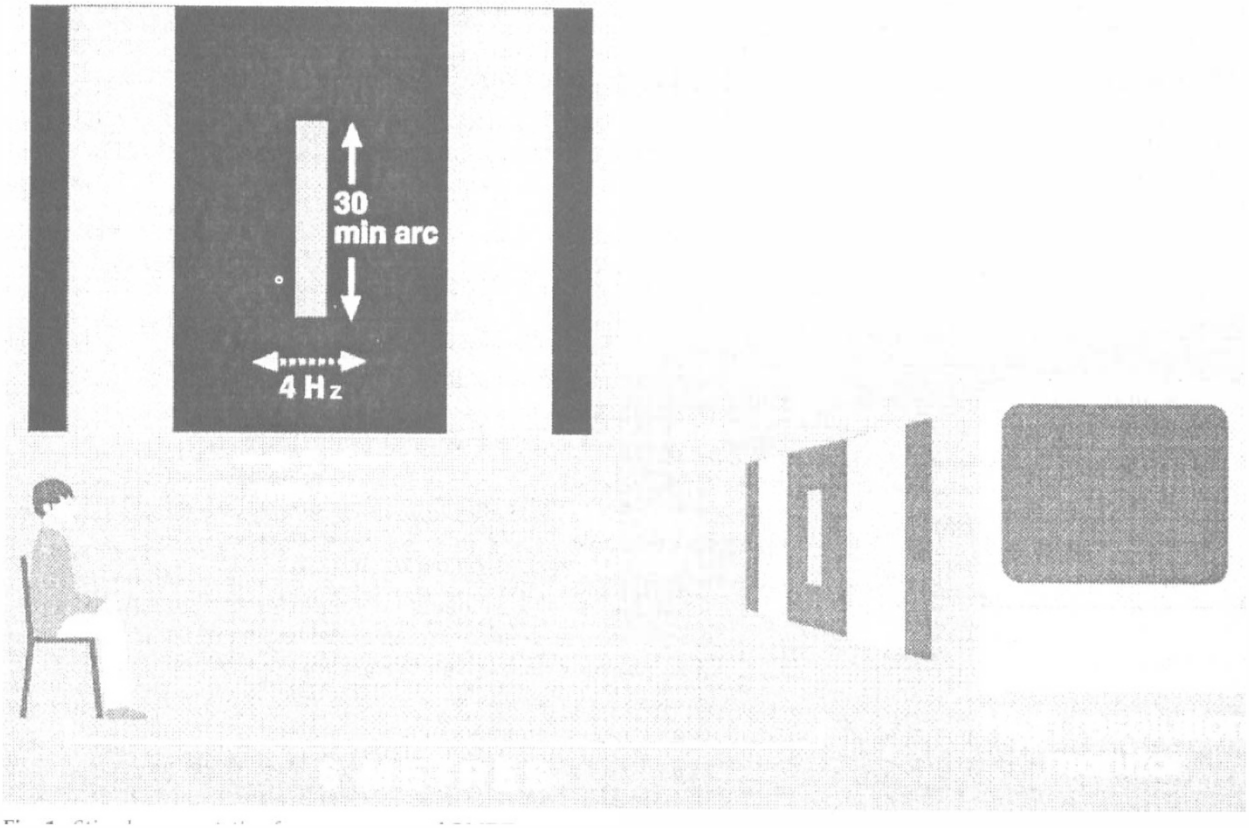

Fig. 1. *Stimulus presentation for measurement of OMDT.*

Patients

Forty-four OH patients, who were not on any form of treatment, and 29 age-matched normotensive patients, were evaluated. All patients had a corrected logmar visual acuity of at least 0.7 and no history of ocular or neurological disease, except OH in the experimental group. Patients were included in the OH group if the IOP exceeded 21 mmHg on two or more occasions using Goldmann contact tonometry, and two or more normal Humphrey Field Analyzer (HFA, Humphrey, Dublin, CA) examinations using the criterion of no points of the full threshold test on Program 24–2 depressed by more than 5 dB compared with age-matched controls. All subjects had comprehensive eye examinations to document anterior segment, open iridocorneal angles (Shaeffer grade III–IV), posterior pole, macular region and normal optic nerve head according to established criteria.[13] There were no refusals to participate.

Stimuli

One examiner (T.J.B.) performed determination of OMDT. The subject viewed an oscillating stimulus and two stationary reference lines generated using an Innisfree 'Picasso' CRT Image Synthesiser onto a Kikusui Cos 117 CRT (P31 phosphor, frame rate 200 Hz). The test stimulus, which could be made to oscillate at 4 Hz, subtended 30 arc min vertically by 5 arc min horizontally at a 6 m viewing distance. The stationary reference bars, which were of equivalent luminance (135 cd/m^2), subtended 1° vertically by 14 arc min horizontally. Screen background luminance was 15 cd/m^2 giving a Michaelson contrast of 80%. Stimulus configuration when the central oscillating bar was stationary is shown in Fig. 1.

Procedure

One eye was tested in each patient. If both eyes fulfilled the study criteria then one eye was selected at random. Subjects adapted to the dimmed ambient light levels for 7 min prior to OMDT determination, which utilised a two-alternative temporal forced-choice staircase, producing a 71% correct detection level of the psychometric function. The initial displacement amplitude was 32 arc sec and increased by steps of 16 arc sec following an incorrect response. With two correct responses the displacement amplitude was similarly reduced. Step sizes were progressively reduced to 8 and 4 sec arc, each trial being ended after six reversals. Threshold was estimated from the mean of the last four reversals. Test duration was 3–5 min.

Statistics

Analysis of variance (ANOVA) was used for statistical analysis unless stated otherwise.

Results

Comparison of the two groups reveals that ocular hypertensives have significantly greater OMDT than the control group ($t = -2.7$, $p < 0.008$; $F = 10.1$, $p < 0.000001$). The mean OMDT for the control group was 22.2 sec arc

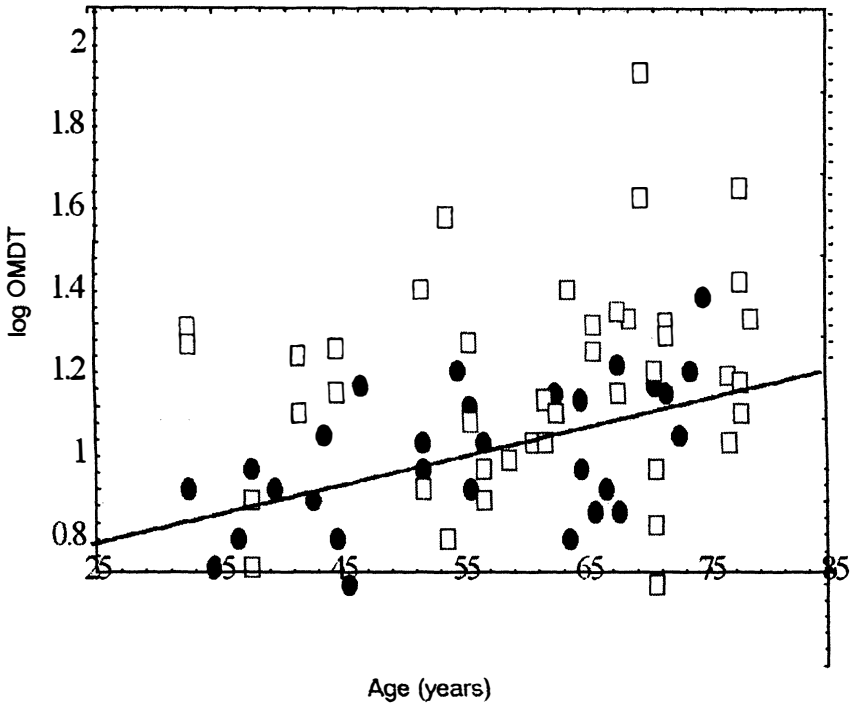

Fig. 2. *The variation in log OMDT with age for the normal (black ovals) and ocular hypertension (white rectangles) groups.*

(Lg 1.3) with a range of 10–50 sec arc (Lg 1.0–1.7) and standard deviation of 8.9 sec arc (Lg 0.2). This compares with a mean OMDT for the OH group of 37.1 sec arc (Lg 1.5) with a range of 11.2–176.8 sec arc (Lg 1.000–2.3) and standard deviation of 28.3 sec arc (Lg 0.3). The results are shown graphically in Fig. 2. This shows a trend line of age versus OMDT, the abnormal observers being at least 2 standard deviations above the mean. By this method, 13 of 44 (29.5%) OH individuals were abnormal. There is a general tendency for OMDT to increase with age – an observation that has been made before.[14] As anticipated, the two groups had significantly different IOPs ($t = -13.3$, $p < 0.000001$; F 60.9, $p = 0.000001$). A comparison of the IOP of control subjects with the OH group showed that the mean IOP of the 29 normal control subjects (14 males, 15 females) was 19.9 mmHg (range 18–22 mmHg, standard deviation 0.4–mmHg) compared with 27.1 mmHg (range 22–34 mmHg, standard deviation 2.9 mmHg) for the 44 OH (20 male, 24 female) subjects. There was no significant difference in the mean age of the two groups ($t = 1.7$, $p = 0.1$; $F = 1.0$, $p = 0.9$), although the mean age of the OH group was slightly higher at 61.3 years (range 33–79 years, standard deviation 13.0 years). The mean age of the control group was 56.1 years (range 33–75 years, standard deviation 13.1 years).

Discussion

OMDTs have been demonstrated to be versatile in detecting early neuronal loss in a variety of ocular disorders. Results obtained for the OH group indicate that OMDT were significantly elevated when compared with a normal control group, suggesting early neuronal dysfunction in about 30% of such patients. This may not be surprising since it is known that long-standing OH can progress to POAG[15] and a significant proportion of ganglion cells are lost before visual field deficits are evidenced by automated perimetry.[16] The IOP in such individuals may have been elevated for a longer period of time, showing greater variability, or they may have had a greater susceptibility to optic nerve head or nerve fibre layer damage. A more plausible explanation is that, in the OH group, the higher IOP causes retinal ganglion cell dysfunction without necessarily producing cell loss. This could lead to an abnormality rate (30%) far in excess of the known conversion rate. This group of patients did not receive treatment, but continue to be monitored closely in the clinic.

Cleary identifying individuals who are susceptible to developing field loss is of importance and highlights the need for evaluating alternative tools for early detection of glaucoma. Psychophysical testing,[17–19] electrophysiological testing,[20–22] and histological examination[13] have all been used for this purpose. OMDT represent a valuable technique in the assessment of OH. A recent study reviewed various psychophysical techniques with the conclusion being that the application of a range of different psychophysical and electrophysiological techniques, rather than just one, may enhance our ability to detect early glaucomatous damage.[23] However, all these techniques remain experimental detection methods and the time and expertise required for their operation limit their use in the clinical setting. Furthermore, other methods such as ophthalmological evaluation of the optic disc are an easier and yet reliable way of studying various optic nerve head parameters in the early detection of glaucoma.[24] The most comprehensive method of early detection would probably include a combination of structural and functional correlates. However, in a previous study there was no correlation between optic

disc parameters and peripheral displacement thresholds in ocular hypertension.[25] A relationship may be found in the future with more sophisticated imaging technology.

Conflicting evidence may be found concerning which particular ganglion cells are most susceptible to glaucomatous damage.[25] This concept is interesting because if it can be demonstrated which type of ganglion cell is the first to be damaged in glaucoma a psychophysical test could be designed to specifically detect preferential loss of function in that cell type. There is a danger in the concept of selective loss being confused with selective test. Just because a selective test is being used to detect, for example, magnocellular loss, it does not mean that those cells are being lost preferentially, simply because this test can only specifically isolate magnocellular cell function. Using such a selective test one does not know what is happening to the other ganglion cell types. However, non-selective cell loss does not undermine testing for those groups of cells which exhibit reduced redundancy.[25]

In conclusion, OMDT represents a robust psychophysical method to detect early glaucomatous loss and may have a part to play in monitoring OH patients. It is a method that can be used in the presence of media opacities and is a relatively simple test to operate. A long-term follow-up study is necessary to establish whether an abnormal OMDT is a marker for conversion from OH to glaucoma. Very importantly, it may provide a marker for early damage, which may be reversible. Researchers in the field of early detection must begin to consider what tests or combination of tests, psychophysical or otherwise, can reliably complement present techniques of diagnosis of glaucoma in the clinical setting.

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