

Case Record 8

Ocular Hypertension



January 2012

Dr Peter Frampton
DOptom MSc FCOptom
BAppSc(Optom)(AUS) DipTp(AS)
DipTp(SP) DipTp(IP)



A personal view: Community OHT Management and NICE

The publication of the NICE guidelines on glaucoma (NICE 2009) created enormous debate and not an insignificant level of conflict within and between health care professionals.

In the author's opinion the NICE guidelines (2009) constitute a balanced collation of our current understanding of risks, conversion rates and management options for COAG and OHT. The NICE recommendations, based largely on the evidence from the major longitudinal studies, synthesised by the European Glaucoma Society (2003), are not meant to be exclusive. Nowhere in the NICE document is it suggested that optometrists are outside the loop and must refer everyone with tensions over 21mmHg, even in the complete absence of other observable signs of risk or frank disease.

What NICE do state is that at diagnosis the patient should be offered GAT, Pachymetry, Gonioscopy, threshold fields and optic nerve assessment (NICE 2009). The guidelines strive to target the 9.5% of OHTs at risk of conversion (Brandt et al 2001, EGS 2003), while ensuring the differential diagnosis of those at risk of acute angle closure. Interpretation comes with the term 'diagnosis'. Does that imply an ophthalmologist? If it does then optometrists return to being screeners, reporting abnormalities without interpretation. This represents a great opportunity; we can meet the challenge and take optometry forward or be left behind. Optometrists and undergraduates need to be pro-actively encouraged to embrace the required skills of gonioscopy, pachymetry and perhaps even applanation tonometry. We need to create clinicians, not recipe followers.

Significantly, the NICE document specifically qualifies the guidelines by stating that they do not override a clinician's responsibility to make decisions appropriate to each patient.

December 2011

Salient information taken from electronic records

DATE: 3/12/11

Mr

Age : 50.

Address

Presenting Symptoms

Routine eye exam. Becoming more dependent on near Rx and inconvenience increasing at work. Distance vision fine . No diplopia. No HAs.

POH

Reading Specs only. No previous ocular surgery or treatments.

FOH

Glaucoma - father.

General Health and Medications

Non-smoker. Allergic to Penicillin. No Hayfever

No medications : General health good but has not seen GP for many years.

No previous history of general or ocular medication use or surgery.

Refraction

R +0.50/-0.25x85 (6/4.8-) Add +1.75 N5

L +0.25/-0.25x90 (6/4.8-) Add +1.75 N5

Phorias Dist- 3Exo Near Orthophoroc

<u>Tensions (GAT)</u> (11.05am)	R 26	L 25	<u>Glaucoma Screen</u>
<u>Pachymetry</u>	575µm	560µm	Full (attached)
<u>Pupils</u>	E&A D,C& N		

Slit Lamp

VH 3 Angles open. (Gonioscopy not considered in this case), Iris configuration Flat. Corneas clear.

Dilated Fundsocopy (0.5% Tropicamide)

Right and Left Discs VCD 0.1 Neural rims healthy and uniform (ISNT conforms) No barring, no bayoneting, No PPA. (Photographed)

No RNFL defects noted

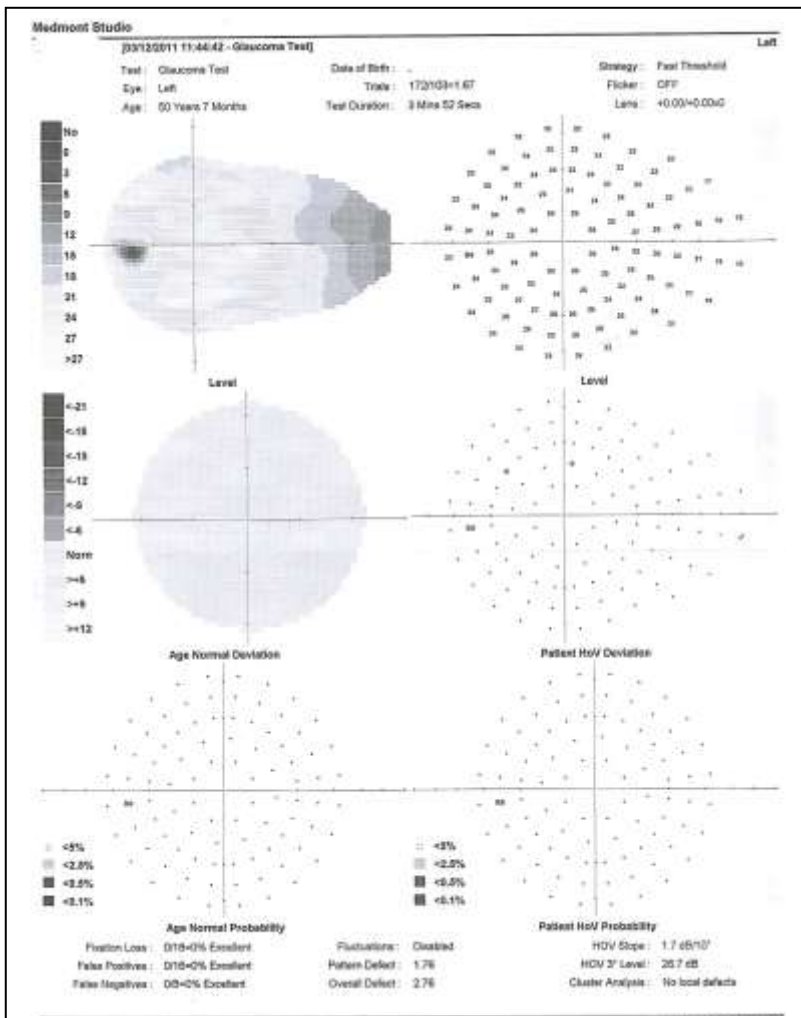
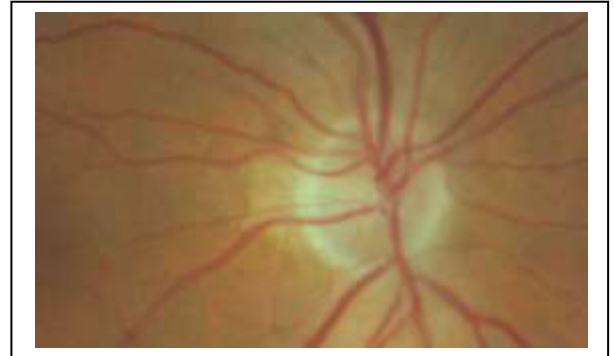
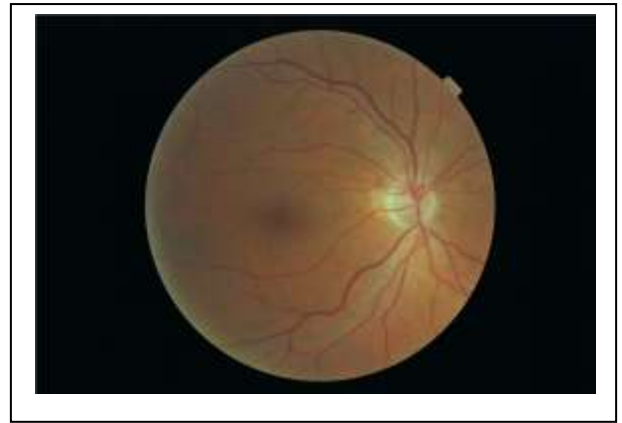
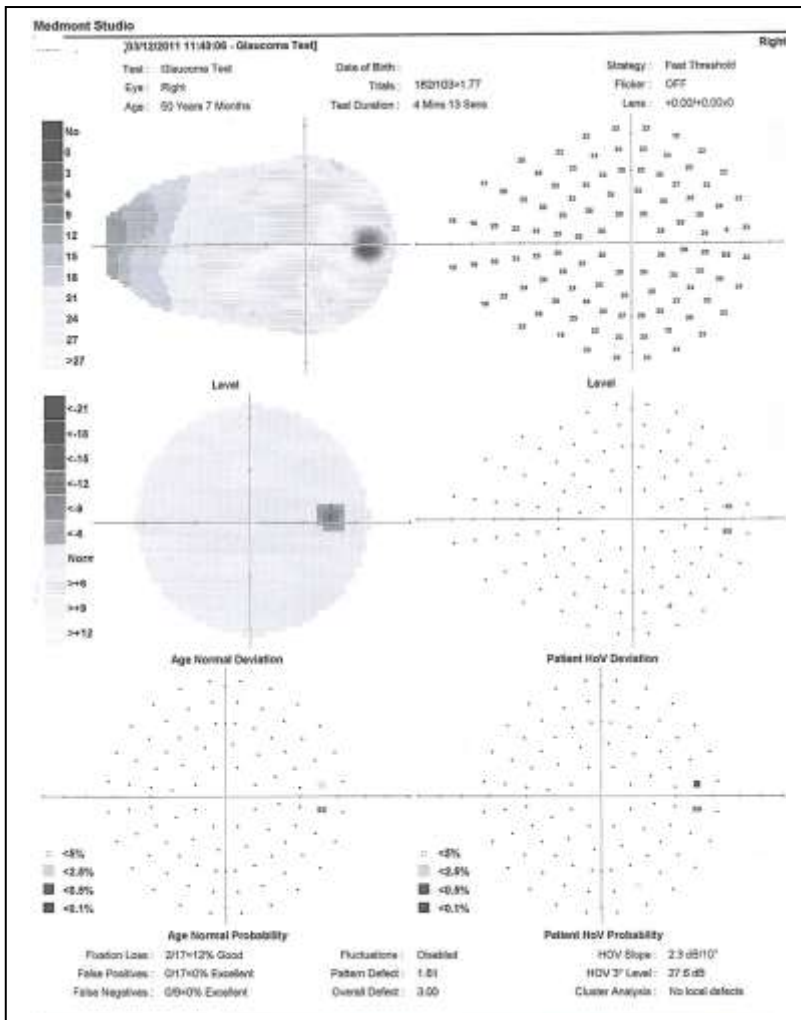
Advice and CMP

Advised on OHT and increased risk of converting. Need to monitor regularly since FH of glaucoma and close to NICE prophylactic treatment guideline.

Also suggested a well man clinic check to ensure no undetected vascular risk factors that may influence management.

Date : 4/12/11 (11.00am)

GAT repeated and confirmed R 24, L 24



The Ocular Hypertensive Treatment Study (OHTS) (Kass et al 2002) categorically showed that hypotensive medication halved the conversion rate of OHTs to glaucoma; at least over a 5 year period. Such a dramatic percentage drop in conversion would seem to support the conservative concept of prophylactic treatment.

However, as early as the 1960s, evidence was presented showing that only a minority of ocular hypertensive (OHT) patients develop glaucomatous optic nerve (ON) damage (Palmberg 2002). Further, the 50% reduction in conversion observed in the OHTS represented a drop from 9.4% to 4.4% (EGS 2003) of the sample group of over 1600 participants (Feuer et al 2002). Over 90% of untreated patients did not convert while half the treated patients went on to be classified as POAG despite treatment (EGS 2003, Kass et al 2002).

The OHTS Manual of Procedures (2001) reported, at that time, equal opinion for and against the effectiveness of treatment. The same manual estimated the annual cost of glaucoma medication in the US to be \$300 million with little evidence of societal health benefits. The document further considered the adverse drug effects on individuals as well as costs in time and lost productivity to the community. Since OHT has a 10 to 15 times greater prevalence than POAG (Bell and Charlton 2011) there is a clear need to identify and treat those ocular hypertensives at higher risk of conversion without inappropriately treating those at low risk.

Gordon et al (2002) concluded that Baseline Age, Horizontal and Vertical CD ratios, Pattern Standard Deviation (PSD), IOP and Central Corneal Thickness (CCT) are strong predictive factors of conversion. The European Glaucoma Society (2003) synopsis of numerous OHTS papers suggests treatment should be offered to moderate risk patients based on age, medical status, life expectancy and treatment benefit.

The general medical status of individual patients needs to be pro-actively investigated by Optometrists wishing to be involved in community monitoring of 'at risk' patients. While more emphasised for normal tension glaucoma (NTG), pressure independent factors, especially the vascular supply to the optic nerve are important in the evolution and progression of glaucoma (EGS 2003). Drance et al 2001 and Anderson et al 2003, considering NTG only, identified disk haemorrhages and migraine, and probably all vasospasm or vascular dysregulation, as clearly identifiable predictive markers for progression. Diastolic Perfusion Pressure (pressure gradient between IOP and Diastolic BP. $\text{Diastolic Perfusion Pressure} = \text{Diastolic BP} - \text{IOP}$), is a marker of general vascular dysregulation, and should be above the critical level identified by Tielsch and Quigley (1995) of 50mmHg.

The NICE guidelines (2009), as well as considering the investigative techniques that should be offered a patient diagnosed with OHT, also highlight a knowledge of current systemic and topical medications and drug allergies and intolerances as vital to the correct management of a patient. These must become standard enquiries with all patients presenting for examination.

The patient involved falls within an 'at risk' group. Repeatable tensions of 25mmHg, a CCT of less than 590µm, age and a family history of glaucoma could suggest prophylactic treatment with a β-Blocker (OHT pathway Appendix 1). β-blockers are more cost effective, for IOP >21-25mmHg and CCT between 555 and 590µm, than prostaglandin analogues (NICE 2009), but are known to cause cardiovascular and pulmonary side effects (BNF 2011, Camras 1996, Geiser, Juzych, Robin and Schwartz 1996). Younger patients are less likely to manifest chronic breathing or heart maladies as was the case for the presenting patient. If treatment was considered however, blood pressure and a general health screen would be prudent as he reported not having had a health check for many years.

The tensions recorded are marginal for NICE recommended prophylactic treatment, regardless of CCT. The fields were full, with excellent reliability and global indices, Cluster Analysis, Age Normal Deviation and Pattern HoV Deviations. Gonioscopy was not considered necessary; no suggestion of angle congestion within the anterior chamber was found and the angles were estimated as Grade III for each eye (van-Herrick's), only angles less than III have been found to be closable (Palmberg 1996).

The decision not to treat does necessitate meticulous follow-up (Bullimore 2002). The European Glaucoma Society (2003), reporting results from the Early Manifest Glaucoma Trial, also suggest that patients should be monitored more closely in the first few years than is commonly done. NICE (2009) did not identify any studies indicating a recommended review period for patients with OHT, treated or untreated. The recommend review for treated OHTs at high risk but at target pressure is 6 to 12 months.

The patient was advised on the clinical considerations and a six month recall uploaded.

REFERENCES

1. Anderson D, Drance S and Schulzer M for CNTGS. (2003). Factors that Predict the Benefit of lowering Intraocular Pressure in Normal Tension Glaucoma. *American journal of Ophthalmology* 136(5): 820-829
2. Bell J and Charlton J. (2011). Ocular Hypertension. Accessed Emedicine/Medscape <http://emedicine.medscape.com/article/1207470>.
3. Brandt JD, Beiser JA, Kass MA, Gordon MO and the Ocular Hypertensive Treatment Study (OHTS) Group. (2001). Central Corneal Thickness in the Ocular Hypertensive Treatment Study (OHTS). *Ophthalmology* 108 (10) : 1779-1788.
4. BNF 62 (September 2011). British National Formulary. Accessed www.bnf.org
5. Bullimore M. (2002). To Treat or Not To Treat? That is the Question. *Optometry and Vision Science* 79(12) : 741-742.
6. Camras CB. (1996). Comparison of latanoprost and timolol in patients with ocular hypertension and glaucoma. *Ophthalmology*, 103, 138-147.
7. Drance S, Anderson D and Schulzer M for the CNTGTS. (2001). Risk Factors for Progression of Visual Field Abnormalities in Normal-Tension Glaucoma. *American Journal of Ophthalmology* 131 : 699-708.
8. European Glaucoma Society. (2003). Terminology and Guidelines for Glaucoma Edition II. Dogma. Accessed www.eugs.org
9. Feuer W, Parrish R, Schiffman J, Anderson D, Budenz D, Wells M, Hess D, Kass M, Gornon M and the OHTS Group. (2002). The Ocular Hypertension Treatment Study: Reproducibility of Cup/Disc Ratio Measurements Over Time at an Optic Disc Reading Centre. *American Journal of Ophthalmology*; 133(1): 19-28.

10. Geiser SC, Juzych M, Robin AL and Schwartz GF. (1996) Clinical Pharmacology of Adrenergic Drugs. In Ritch R, Shields MB and Krupin T (eds). *The Glaucomas (second edition) Vol III Glaucoma Therapy*. Mosby. USA.
11. Gordon M, Beiser J, Brandt J, Heuer D, Higginbotham E, Johnson C, Keltner J, Miller P, Parrish R, Wilson R, Kass M for the Ocular Hypertension Treatment Study. (2002). The Ocular Hypertensive Treatment Study: baseline factors that predict the onset of primary open angle glaucoma. *Arch Ophthalmology*; 120(6): 714-720.
12. Kass M, Heuer D, Higginbotham E, Johnson C, Keltner J, Miller J, Parrish R, Wilson M and Gordon M. (2002). The Ocular Hypertension Treatment Study: A Randomized Trial Determines that Topical Hypotensive Medication Delays or Prevents the Onset of Primary Open Angle Glaucoma. *Arch Ophthalmology*; 120: 701-713.
13. NICE. (2009). Glaucoma, Diagnosis and Management of Chronic Open Angle Glaucoma and Ocular Hypertension. National Collaborating Centre for Acute Eye Care. Accessed <http://www.nice.org.uk/nicemedia/live/12145/43887/43887.pdf>
14. Ocular Hypertension Treatment Study (OHTS). (2001). Manual of Procedures Version 3.0. accessed <http://www.vrcc.wustl.edu/mop/mop.htm>
15. Palmberg P. (1996). Gonioscopy. In Ritch R., Shields M.B. and Krupin T. *The Glaucomas (Second Edition) Vol I Basic Sciences* . Mosby. USA.
16. Palmberg P. (2002). Answers from the Ocular Hypertension Treatment Study. *Arch Ophthalmology*; 120: 829-830.
17. Tielsch J and Quigley H. (1995). Hypertension, Perfusion Pressure, and Primary Open-angle Glaucoma. *Arch Ophthalmology*, 113: 216-221.

APPENDIX 1
NICE 2009 OHT Pathway

OHT pathway (monitoring and treatment for people with OHT and people with suspected COAG who have high IOP)

