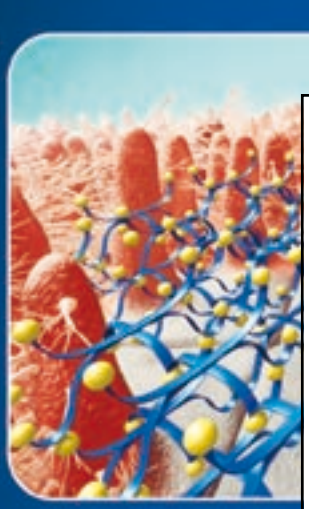
SYSTANE®: A Revolution in Dry Eye Therapy



Dry eye causes destabilisation of the tear film which leaves the cornea vironment and can cells[1]

YSTANE contacts the eye -like protective layer e tear film[2] l repair processes to occur[2]

Use SYSTA for ALL Dry

- Soothing relie
- Superior and l efficacy to Re

OFFICE USE

Job Number: _____

Designer: _____

CAMPUS GRAPHICS
La Trobe University

APPROVAL

PLEASE check this proof carefully for errors and omissions.
Your signature below constitutes acceptance and full responsibility for all errors, omissions and legal and ethical compliance in this document. CAMPUS GRAPHICS will not accept liability for errors overlooked at this stage of proofing. Author corrections will be charged at our hourly rate plus materials.

OK AS IS

____ / ____ / ____
your signature/date

OK WITH CORRECTIONS
NO FURTHER PROOF REQUIRED

____ / ____ / ____
your signature/date

CORRECTIONS REQUIRED
SHOW NEW PROOF

____ / ____ / ____
your signature/date

This proof is not submitted for color approval, print quality, or paper approval.
This proof must be approved 10 working days prior to print deadline.

Initial and return ASAP or Fax: 9479 1655

Start with Systane, Stay with Systane

PBS Information: Restricted Benefit 1 + 5 repeats

1. Korb DR *et al.* 2002. The Tear Film: Structure, function and clinical examination. Butterworth Heinemann, Oxford, UK. 2. Meadows DL. *Rev Ophthalmol* 2003; April (pt 2 of 2): 10. 3. Christensen MT *et al. Curr Eye Res* 2004; 28: 55-62. 4. Tauber S. *Ophthalmology Management* May 2003. 5. Christensen MT *et al.* 23rd Biennial Cornea Research Conference, ARVO 2003.

Australian
Orthoptic
Journal

2007 Volume 39 (1)

The official journal of the Orthoptic Association of Australia Inc
ISSN 0814-0936

The Australian Orthoptic Journal is peer-reviewed and the official biannual scientific journal of the Orthoptic Association of Australia Inc. The Australian Orthoptic Journal features original scientific research papers, reviews and perspectives, case studies, invited editorials, letters and book reviews. The Australian Orthoptic Journal covers key areas of orthoptic clinical practice – strabismus, amblyopia, ocular motility and binocular vision anomalies; low vision and rehabilitation; paediatric ophthalmology; neuro-ophthalmology including nystagmus; ophthalmic technology and biometry; and public health agenda.

Published by the Orthoptic Association of Australia Inc. (Publication date: May 2007).

Editors' details: Zoran Georgievski z.georgievski@latrobe.edu.au, Konstandina Koklanis k.koklanis@latrobe.edu.au, Department of Clinical Vision Sciences, La Trobe University VIC 3086 Australia. Fax: +61 3 9479 3692. AOJ@orthoptics.org.au. Designed by Greg Nelson and printed by Campus Graphics, La Trobe University VIC 3086 Australia, www.latrobe.edu.au/campusgraphics Distributed by the Orthoptic Association of Australia Inc, PO Box 1175 Hampton VIC 3188 Australia.

GUIDELINES FOR AUTHORS

It is a condition of acceptance of any article for the Australian Orthoptic Journal that original material is submitted. The cover letter must accompany the submission and state that the manuscript has not been published or submitted for consideration for publication elsewhere.

The types of manuscripts accepted are as follows:

- **Editorials (by invitation)**
- **Original Scientific Research Papers**
- **Reviews/Perspectives**
- **Case Studies**
- **Letters to the Editor**
- **Book Reviews**

MANUSCRIPT SUMBMISSION

Submitted manuscripts must include a title page, abstract (including keywords), the paper itself, any acknowledgements, references and tables and/or figures. Each of these sections should begin on a separate page. Pages should be sequentially numbered. The manuscript submission should be electronic, via email to: AOJ@orthoptics.org.au

Title Page: The title page should include the title of the manuscript and each author's name, academic qualifications and institutional affiliation(s). A 'corresponding author' should be designated and their address, telephone number, fax number, and email address listed. The title page should also include the word count for the abstract and text.

Abstract and Keywords: The abstract should not exceed 250 words. It should be a clear and succinct summary of the paper presented and need not be structured into subsections. However, where appropriate, it should relate to the format of the paper, including aim, methods, results and conclusion. Beneath the abstract, include up to 5 keywords or terms suitable for use in an index or search engine.

Text: Manuscripts should not exceed 3000 words. Where appropriate the structure of the text should be as follows: Introduction, Method, Results, Discussion and Conclusion. Authors should also use subheadings for Case Studies, generally as follows: Introduction, Case Report and Discussion (Conclusion is optional). Case Studies should not exceed 1500 words.

References: References must be numbered consecutively in order of appearance in the text. In text references should be designated a superscript number following all punctuation.

When there are 5 or more authors, only the first three should be listed followed by et al. References to journal articles should conform to abbreviations in Index Medicus. Examples of reference styles are as follows:

Article: Cornell E, Flanagan J, Heard R. Evaluation of compensatory torsion by blind spot mapping. Aust Orthopt J 1996; 32: 13-17.

Book: Kline LB, Bajandas FJ. Neuro-ophthalmology: Review Manual. 5th Ed. Thorofare, NJ: Slack Inc, 2004.

Book Chapter: Murphee AL, Christensen LE. Retinoblastoma and malignant tumors. In: Wright KW, Spiegel PH, editors. Pediatric Ophthalmology and Strabismus. 2nd Ed. New York: Springer, 2003: 584-589.

Tables and Figures: Tables and figures (graphs, illustrations photographs) must be accompanied by a suitable title and numbered consecutively as mentioned in the text. It is preferable if images are supplied as high resolution jpeg, tiff or EPS files.

Acknowledgments: Identify all sources of financial support including grants or sponsorship from agencies or companies. Include any acknowledgments to individuals who do not qualify for authorship.

Conflict of Interest: Authors should declare any financial support or relationships that may pose a conflict of interest or perceived to.

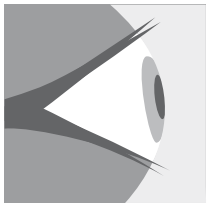
THE REVIEW PROCESS

Manuscripts are reviewed by two referees. The referees are masked to the authors and vice versa. Authors will be notified of the decision once the reviews have been received. Where revisions are required, the author must re-submit within twelve weeks or an agreed timeframe. Revised papers received late will be treated as new submissions.

ENQUIRIES

If you have any enquiries contact the Editors.
Email: AOJ@orthoptics.org.au
Tel: Zoran Georgievski 03 9479 1919
Connie Koklanis 03 9479 1903
Fax: 03 9479 3692.

Australian
Orthoptic Journal
2007 Volume 39 (1)



ORTHOPTIC ASSOCIATION of AUSTRALIA inc

CONTENTS

Editorial

The New Orthoptics

Welcome to the new Australian Orthoptic Journal, the official peer-reviewed scientific publication of the Orthoptic Association of Australia.

This being our first Editorial and indeed as the new Editors of the AOJ, we have paused to reflect on the purpose of our journal for our profession. It is important to revisit such a basic question given that once, certainly when the AOJ was first published in 1966 (and previously as the Transactions of the Orthoptic Association of Australia), the educational and scientific material that was available to members of our profession was much less than what we have access to today. In fact, little under two decades ago there were relatively few sub-specialty journals in the medical field of ophthalmology – for instance, there were none in strabismus, binocular vision etc. (our particular area of clinical and research interest) – but today there seems to be a plethora of sub-specialty journals, including a handful in strabismus and binocular vision. And of course there's the internet; information literally at our fingertips!

Yet, orthoptic journals continue to be published – there are ten around the world (three being English-language publications). All constantly face the challenges that ours does, but we keep on, we persevere. Why? Surely, it's not for the dozen or so papers that each provides us annually? Nor is it principally as a means to publish, since orthoptists' research is increasingly being published in peer-reviewed and Medline-indexed journals.

It is possible that we persevere because we consider a journal to be a diary, an ongoing measure or gauge. Our profession's journal can be considered to be the 'height chart' on the wall for our profession, a permanent record that chronicles our growth year after year, each volume after the other. If we can presume at least part truth in this, we will remind ourselves of how important our journal is to our profession and therefore how imperative it is to keep it going – to persevere. More so today, each volume that is published is indeed a credit to our profession given the 'competition' from not only the increased number of other journals in our field, but the fact that our clinical and research interests have diversified such that we seek to be part of the readership of other journals too. This reflects our profession's progress…

A volume of the AOJ 30 years ago contains a presidential address by Vivienne Gordon entitled, *Orthoptics – The Expanded Role*[1]. An excerpt follows:

> … "Orthoptists provide specialist services in the investigation and treatment of disorders of ocular motility… The expanded role includes visual field testing, glaucoma investigation techniques …areas relating to applied electrophysiology and preventative visual screening. The orthoptist may also assist the ophthalmologist in the management of eye diseases which require special investigation… and in patient education and counselling"…

In 2007, some 30 years on, orthoptists continue to be involved in varied roles, but have also emerged as eye care practitioners who are developing workforce opportunities and are engaging in new models of eye care where orthoptists have pivotal co-management responsibilities for the betterment of patient care. This year also sees the legislative changes that allow orthoptists increased glasses prescribing rights in three states (VIC, SA and NSW). We look forward to being witness to this increase in scope of practice, in growth, and even more so to it being chronicled in the Australian Orthoptic Journal year after year.

Zoran Georgievski & Connie Koklanis
Department of Clinical Vision Sciences
La Trobe University

REFERENCES

1. Gordon VJ. Presidential address - 1977. Orthoptics - the expanded role. Aust Orthopt J 1977;15:3.

Editorial

The Value of Case Studies

This edition of the AOJ contains four case reports, each very different from the others but they all attest to the value of presenting and publishing case studies. These can be of particular value in several ways.

Firstly, publication of rare cases allows for a data base to be developed. Documentation of these cases can provide data for a more comprehensive review of the condition and its occurrence within the population. The very interesting case study by Sommerville McAlester and Kelly (Temperature – A Contributing Factor in a Case of Superior Oblique Palsy) will also alert the readers of th AOJ to possible pathology when they may come across a similar 'throw away line' from a patient and may assist the patient by the clinician's understanding of the possible association with multiple sclerosis. The simple 'ice test' is one that we can readily use in such cases.

The paper by Dirani, Chamberlain, Garoufalis, Chen, Guymer and Baird (Discordant Unilateral Myopia in Adult Female Monozygotic Twins) is another example of the value of recording a rare example of an ocular anomaly. In what appears to be a first report of a significant refractive error in only one of a pair of monozygotic twins, in the absence of any other apparent anomalies, again causes us to reflect on our understanding of human physiology. As the authors postulate, this may have been due to intrauterine factors, however, the presence of strabismus in each of that twin's children only adds to the conundrum (although, of course, we don't know how the father may have contributed his genes to this).

Publication of rare and unusual cases may eventually aid in the understanding of the pathogenesis of these conditions and highlights need for observation and good history taking.

The cases presented by Vassallo, Mancuso and Harper (Two Cases of Valsalva Retinopathy Treated with Nd:YAG Laser Hyaloidotomy) again add to the literature on a particular condition but they also provide a context to learn more about the pathophysiology of the eye, vision and binocular function. As with the Sommerville McAlester and Kelly case study, a simple comment from the patient's history (such as coughing or sneezing) will alert the clinician to the possible causes of the patient's retinopathy. Finally, with the ever increasing areas of practice that orthoptists now need to understand, they can provide the context to help us understand new treatments, such as Nd:YAG laser hyaloidotomy.

The report by Leone, Georgievski and Koklanis (The Speed of Emmetropia) also provides an opportunity to review basic sciences (in this case physiological optics) and normal development by putting these in the context of a case study (a person!) so that they become more relevant and more easily understood. They can also provide evidence that, in many cases, unnecessary treatment may be counterproductive when there is no evidence (in this case no strabismus and only minor anisometropia) that any pathology or potential pathology exists.

I hope that the readers of the AOJ will be alerted not only to the possibility of an unusual pathology from these presentations but, when they see a patient that they find interesting or unusual, they will also consider presenting the case for publication. We can all contribute to our profession in this way.

Associate Professor Elaine Cornell
School of Applied Vision Sciences
University of Sydney

Two Cases of Valsalva Retinopathy Treated with Nd:YAG Laser Hyaloidotomy

Suzane Vassallo, PhD AOBR[1,2]
Catherine Mancuso, BOrth AOBR[2]
C. Alex Harper, FRANZCO[2,3]

[1]Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia
[2]Victoria Parade Eye Consultants, St. Vincent's Medical Centre, Melbourne, Australia
[3]Centre for Eye Research Australia, Melbourne, Australia

ABSTRACT

Activities involving straining can result in a haemorrhagic retinopathy which may cause sudden loss of vision. This is known as Valsalva retinopathy. Though the fundus findings can vary, a premacular haemorrhage is often seen which may resolve spontaneously over several months. Acute management of such cases can be achieved via neodymium (Nd):YAG laser hyaloidotomy. Even in the presence of a large haemorrhage the prognosis is excellent in an otherwise healthy eye. Two cases of Valsalva retinopathy are presented.

Keywords: Valsalva maculopathy, Nd:YAG laser hyaloidotomy, retinal haemorrhage

INTRODUCTION

Valsalva hemorrhagic retinopathy was first described by Duane[1] in 1972. As the description suggests, the retinopathy arises following a Valsalva manoeuvre wherein forcible exhalation occurs against a closed glottis. The manoeuvre causes an increase in intrathoracic and/or intra-abdominal pressure, with an accompanying increase in venous pressure in the upper parts of the body, including the eye[1]. A sudden elevation in intraocular venous pressure may cause normal or abnormal perifoveal capillaries to haemorrhage – immediate and painless loss of vision ensues in an otherwise healthy eye[2]. Vision loss is commonly unilateral[3].

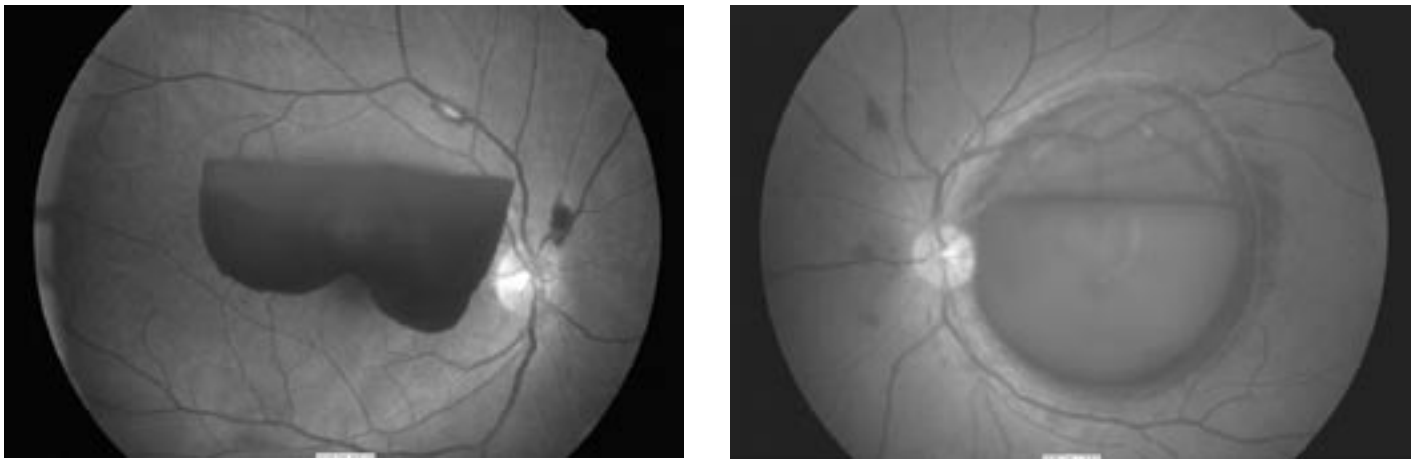
Valsalva retinopathy has been reported to occur during labour[4], heavy lifting (including weight lifting)[3, 5, 6], fiberoptic gastroenteroscopy[7], rigorous sexual activity[8], blowing balloons[9] and colonoscopy[10]. Coughing, sneezing or vomiting are also commonly cited aetiologies[1, 2].

The fundus findings observed with this type of retinopathy varies. The picture may range from one of isolated localised oedema[1], or a small well circumscribed parafoveal preretinal haemorrhage[3], to a massive dome-shaped haemorrhage of up to 5 disc diameters[11] or more[8] in size. The latter may serve to completely obscure the fovea. The size of the haemorrhage seems to correlate with the amount of venous pressure endured during the manoeuvre[1].

The haemorrhage in Valsalva retinopathy may be located between the internal limiting membrane (ILM) and retinal nerve fibre layer (sub-ILM)[12], or between the posterior hyaloid face and the ILM (subhyaloid)[2]. It could also be in both locations[2]. The position of the haemorrhage is clinically difficult to differentiate via slit-lamp biomicroscopy[13], though fine striae and a glistening light reflex on the haemorrhage surface favours a sub-ILM lesion[2, 6, 12]. A more definitive diagnosis appears possible using optical coherence tomography (OCT). Recent findings from two patients[6] highlight that the location of the haemorrhage in Valsalva retinopathy is most likely sub-ILM. While this view has been supported by others[12], the posterior hyaloid face and ILM are not always visible on OCT in the presence of a premacular haemorrhage[7].

Valsalva retinopathy can lead to sudden temporary loss of binocular function. The haemorrhage is usually self-limiting, clearing spontaneously over weeks to months[3, 7]. Intervention in the form of neodymium (Nd):YAG laser[14] or vitrectomy[6] can assist in more rapid visual recovery. While the fundus view can appear striking, a very good prognosis is likely. Two cases of Valsalva retinopathy are presented.

Correspondence: **Dr Suzane Vassallo**
Department of Clinical Vision Sciences, La Trobe University, VIC 3086, Australia.
Email: S.Vassallo@latrobe.edu.au



A **B**

Figure 1. A. Case 1: Right eye fundus at initial presentation B. Case 2: Left eye fundus at initial presentation

CASE 1

A 46-year-old female presented 3-days following sudden loss of vision in her right eye. Upon specific questioning she reported occasional coughing during the 2 weeks prior to her visit, though denied heavy lifting and vomiting. Past ocular history was unremarkable. Her general health, including blood pressure, was normal (105/80 mmHg) and she was not taking any form of medication. Family history was not significant.

Unaided visual acuities were Right (R.) 6/24 Left (L.). 6/18, though each eye improved with pinhole to 6/9, 6/6, respectively. Refraction was not performed at this visit. Intraocular pressure was R. 8 mmHg and L. 9 mmHg. Pupils were equally reactive to light and were dilated with g. tropicamide 1% and g. phenylephrine 10%. Right eye fundus examination revealed a large premacular haemorrhage with foveolar sparing (Figure 1A). The area of the haemorrhage was approximately 5.5 disc diameters and was thought to be subhyaloid. The foveola sparing permitted relatively good visual acuity in this eye (with pinhole), despite the size of the lesion and the patient's presenting complaint. The left eye fundus was normal.

CASE 2

A 28-year-old male Indian student presented with a 4-day history of a red spot obscuring the central visual field in his left eye. Aside from occasional sneezing, he denied any other Valsalva activity. His general health and family history were unremarkable and he was not taking prescription or non-prescription medication. He was a moderate myope (R. and L. -4.00 DS) and prone to developing chalazia.

Corrected visual acuities were R. 6/4 and L. count fingers (CF) when viewing eccentrically with this eye. Intraocular pressures were R. 13 mmHg and L. 14 mmHg. Pupils were equally reactive to light and were dilated with g. tropicamide 1% and g. phenylephrine 10%. In the left eye, fundus examination revealed a large subhyaloid haemorrhage of about 5 disc diameters in area (Figure 1B). Retinal nerve fibre layer haemorrhages were also evident. The right eye fundus was unremarkable.

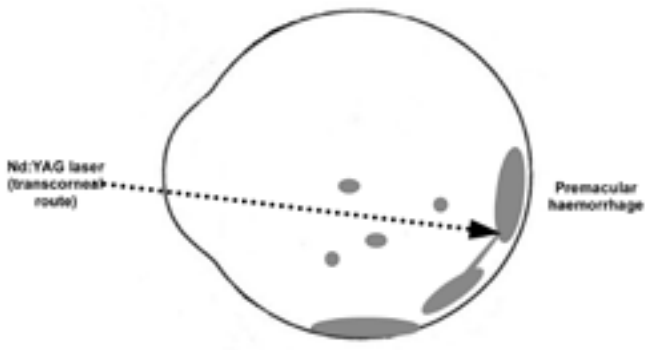


Figure 2. Schematic representation of Nd:YAG laser hyaloidotomy (lateral view). With gravity, blood pools inferiorly in the vitreous where it breaks down over time and is resorbed.

MANAGEMENT

Both cases were managed in the clinic on the day of their presentation with a single shot of Nd:YAG laser (Coherent, Santa Clara, CA) (pulse power 3.2 mJ) to the anterior surface of the posterior hyaloid membrane in an attempt to drain the haemorrhage. The procedure is referred to as a hyaloidotomy or membranotomy. The laser was aimed at the inferior portion of the haematoma, away from the fovea

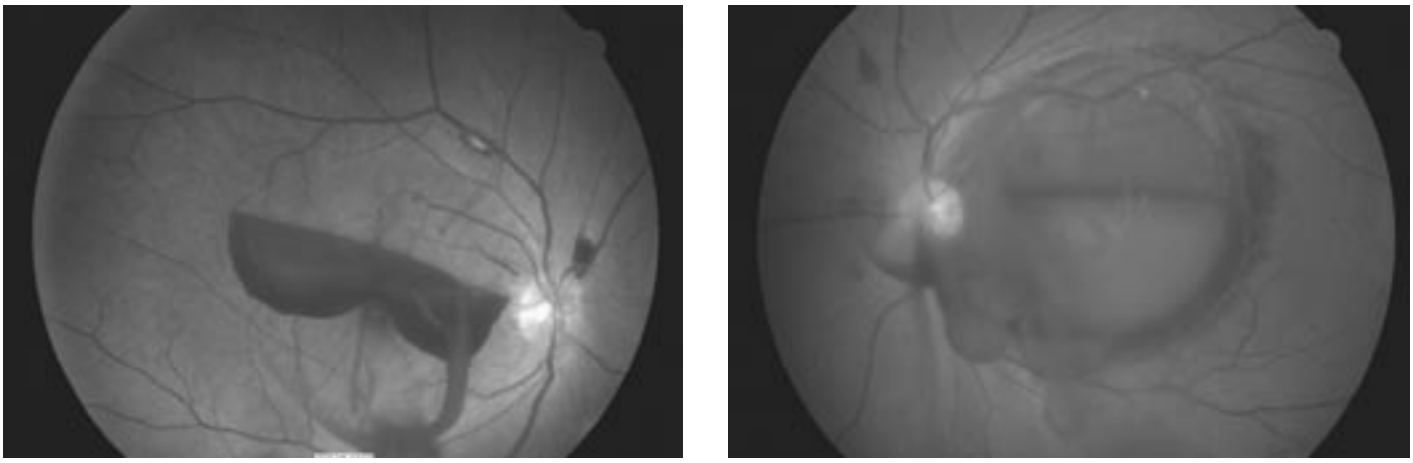


Figure 3. Case 1(left) and Case 2 (right) approximately 15 minutes post Nd:YAG hyaloidotomy. The haemorrhage begins to clear rapidly in both cases.

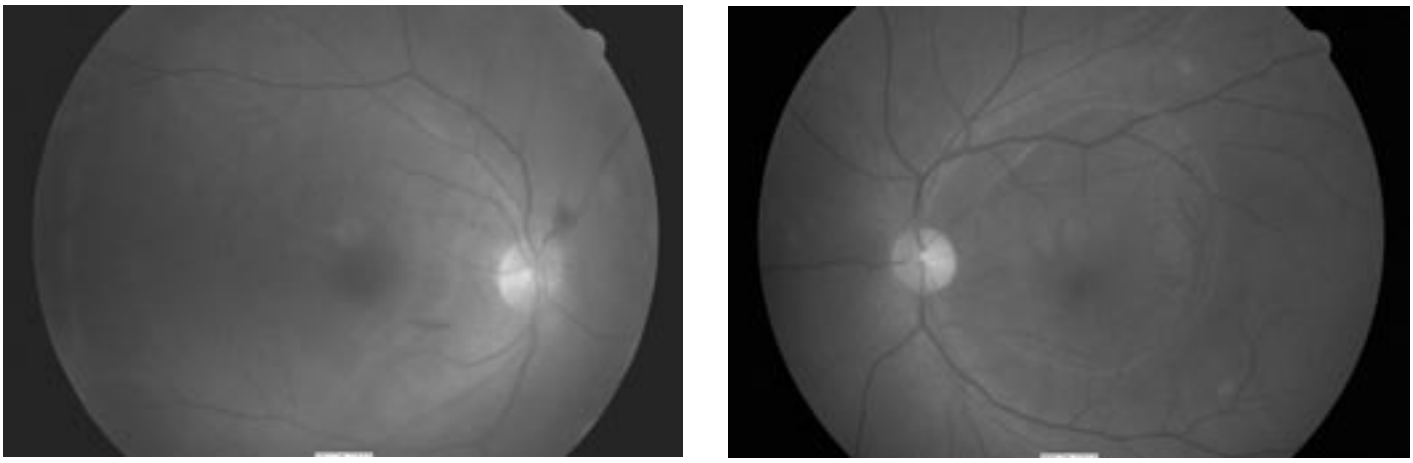


Figure 4. Case 1: One week post Nd:YAG hyaloidotomy. Note the vitreous haze from the haemorrhage dispersion. (Fundus photograph unavailable for Case 2)

Figure 5. Case 2: Seven weeks post Nd:YAG hyaloidotomy. The retinal state has returned to normal. (Fundus photograph unavailable for Case 1).

(Figure 2). In each case, the haemorrhage began to drain immediately post treatment (Figure 3).

Both cases were reviewed one week post laser treatment and both reported an improvement in visual acuity. The fundal view showed vitreous haze, consistent with dispersion of the haemorrhage into the vitreous gel (Figure 4). When compared with the first visit, Case 1 demonstrated clinically stable visual acuities with R. 6/24 L. 6/18, and improvement to 6/5 bilaterally was obtained with refractive correction (R. -3.00/+0.75 x 10° L. -1.50DS). She was monitored for about 3 months post treatment, during which time her right eye vision remained stable. She was then discharged.

Case 2's corrected left eye visual acuity had improved to 6/18- one week post treatment (having initially presented with CF eccentrically). He noted black dots in his left visual field. This continued to clear over subsequent visits. Seven weeks post hyaloidotomy his corrected left eye visual acuity returned to 6/4 and the haemorrhage had completely cleared (Figure 5). Further review was not required.

DISCUSSION

These findings confirm that Nd:YAG laser to the posterior hyaloid face is a safe, rapid and effective intervention in the treatment of premacular haemorrhage arising from Valsalva retinopathy[4, 11, 13-17]. Vision is restored as the blood clears away from the visual axis. The same treatment modality can be used when managing premacular haemorrhages arising from diabetic retinopathy, vein occlusion or retinal macroaneurysm[11]. Although the two cases presented here were not followed beyond 2 to 3 months, those followed for up to 2 years or more have not displayed retinal or choroidal injury from Nd:YAG laser treatment for premacular haemorrhage[13, 18].

Nd:YAG laser photodisruption was first described in the German literature in 1988[17] when multiple laser shots were used to drain the haemorrhage. Since then, the technique has been somewhat modified so that a single laser shot is often used, with 2 to 3 subsequent shots[4, 13, 14] employed

when little drainage is evident from the first[13, 14]. Favardin et al.[15] have used up to 10 shots to clear a premacular haemorrhage arising from diabetic retinopathy.

Nd:YAG hyaloidotomy is considered most effective in treating a premacular haemorrhage of greater than 3 disc diameters in area and of no more than 3 weeks' duration[18]. Treating a large lesion assists in ensuring the laser beam is located away from the fovea – the laser site should be at the inferior margin of the haemorrhage[11, 13, 14]. This reduces the risk of a foveal injury and promotes maximal drainage through the assistance of gravity[16]. The presence of underlying blood is also necessary at the laser site to ensure the retina is protected at this location[11]. A long-standing haemorrhage can be difficult to drain due to blood clotting[15], although a recent report confirms drainage of longstanding altered blood[19]. Others[20] advocate laser treatment for haemorrhages of less than 2 weeks' duration. Once the blood has drained into the vitreous, it breaks down over time and is spontaneously resorbed. It has been suggested that patients be informed to sleep in an upright position to encourage blood settling inferiorly[21].

Conservative management of Valsalva retinopathy involves observation. Generally, the premacular haemorrhage can completely resolve over several months, with full visual recovery in a healthy eye[1-3, 7]. However, such an approach should be reserved for small haemorrhages for these are not suitable for Nd:YAG laser intervention due to the potential for foveal damage[18]. Prompt intervention may be offered for large haemorrhages to speed resolution and restore vision.

Vitrectomy is an option to drain the premacular haemorrhage caused by Valsalva retinopathy[6], however, Nd:YAG laser membranotomy is quick, far less invasive and has fewer potential side effects. The use of vitrectomy in treating premacular haemorrhage due to diabetic eye disease is well established[22].

It has been suggested that patients be informed to exhale during straining or lifting heavy objects so as to prevent a closed glottis and possible retinal damage[5]. It would also be apt to advise patients about the possibility of haemorrhage recurrence where this is considered likely[5]. Finally, reassuring the patient about their prognosis cannot be underestimated[3] and should constitute part of the management approach as well. Many[4, 13, 14] have suggested randomised prospective trials are required to differentiate the mode of treatment which is best for a premacular haemorrhage.

Even though both cases reported were somewhat myopic, there appears no predilection for Valsalva retinopathy with respect to refractive error, age, race or sex[21]. Another aetiology which should be considered when assessing cases of Valsalva retinopathy is the competency of the valve in each internal jugular vein. This is the only valve between the heart and the brain and serves to prevent retrograde venous flow. Diagnosis of a faulty valve is possible via ultrasound investigation[23]. In such cases, patients may not present with a history of a Valsalva manoeuvre per se. These patients would need to be informed about the likelihood of haemorrhage recurrence.

The location of the premacular haemorrhage was thought to be subhyaloid in both cases presented. Striae were not evident on the haemorrhage surface as would be the case for a sub-ILM haemorrhage[2, 6]. Also, it has been postulated that when Nd:YAG laser membranotomy is performed on a sub-ILM haemorrhage, epimacular membrane formation is more likely to result[6]. This was not noted in the cases presented here but perhaps a longer follow-up is required[24]. The rapidity with which the haemorrhage drained is suggestive of a subhyaloid bleed as well[14]. While OCT imaging was not performed in the investigation of these cases, the management options for Valsalva retinopathy would not differ depending upon haemorrhage location[14].

CONCLUSION

Complete resolution of the haemorrhage in Valsalva retinopathy results in an excellent outcome. The findings from the cases presented here support the use of Nd:YAG hyaloidotomy in its management. It appears a safe and effective means of promoting the rapid return of visual acuity to its former state in an otherwise healthy eye. Patients can be reassured about their visual prognosis.

REFERENCES

1. Duane TD. Valsalva hemorrhagic retinopathy. Trans Am Ophthalmol Soc 1972;70:298-313.
2. Gass JDM. Stereoscopic atlas of macular diseases: diagnosis and treatment. 3rd ed. Vol. 2. St. Louis: C.V. Mosby, 1987:564.
3. Chapman-Davies A, Lazarevic A. Valsalva maculopathy. Clin Exp Optom 2002;85:42-45.
4. Ladjimi A, et al. Valsalva retinopathy induced by labour. Eur J Ophthalmol 2002;12:336-338.
5. Romano PE. Exhale while lifting or straining to avoid Valsavla retinopathy or bleeding from stressed retinal vessels. Eur J Ophthalmol 2003;13:113.
6. Shukla D, Naresh KB, Kim R. Optical coherence tomography findings in Valsalva retinopathy. Am J Ophthalmol 2005;140:134-136.
7. Choi S, Lee S, Rah S. Valsalva retinopathy associated with fiberoptic gastroenteroscopy. Can J Ophthalmol 2006;41:491-493.
8. Friberg TR, Braunstein RA, Bressler NM. Sudden visual loss associated with sexual activity. Arch Ophthalmol 1995;113:738-742.
9. Georgiou T, Pearce I, Taylor R. Valsalva retinopathy associated with blowing balloons. Eye 1999;13:686-687.
10. Oboh AM, Weilke F, Sheindlin J. Valsalva retinopathy as a complication of colonoscopy. J Clin Gastroenterol 2004;38:793-794.
11. Rennie CA, et al. Nd:YAG laser treatment for premacular subhyaloid haemorrhage. Eye 2001;15:519-524.
12. Meyer CH, et al. Is the location of Valsalva hemorrhages submembranous or subhyaloid? Am J Ophthalmol 2006;141:231.
13. Çelebi S, Kükner AS. Photodisruptive Nd:YAG laser in the management of

premacular subhyaloid hemorrhage. Eur J Ophthalmol 2000;11:281-286.

14. Raymond L. Neodymium:YAG laser treatment for hemorrhages under the internal limiting membrane and posterior hyaloid face in the macula. Ophthalmology 1995;102:406-411.
15. Farvardin M, et al. Neodymium:YAG laser treatment for premacular hemorrhage. Arch Iranian Med 2005;8:8-13.
16. Durukan H, et al. Photodisruptive neodymium:yttrium-aluminum-garnet laser in the management of premacular subhyaloid haemorrhage. Asian J Ophthalmol 2002;4:10-12.
17. Faulborn J. Behandlung einer diabetischen pramakularen Blutung mit dem Q-switched Neodym:YAG laser. Spektrum Augenheilkd 1988;2:33-35. cited in: Raymond L. Neodymium:YAG laser treatment for hemorrhages under the internal limiting membrane and posterior hyaloid face in the macula. Ophthalmology 1995;102:406-411.
18. Durukan AH, et al. Long-term results of Nd:YAG laser treatment for premacular subhyaloid haemorrhage owing to Valsalva retinopathy. Eye 2006;September 1:doi:10.1038/sj.eye.6702574.
19. Aralikatti AKV, Haridas AS, Smith MA. Delayed Nd:YAG laser membranotomy for traumatic premacular hemorrhage. Arch Ophthalmol 2006;124:1503.
20. Chen Y, Kou H. Krypton laser membranotomy for premacular hemorrhage. Ophthalmologica 2004;218:368-371.
21. Duszak RS. Retinopathy, Valsalva. 2006 Oct [cited 2 Nov 2006]; Available from: URL: http://www.emedicine.com/OPH/topic769.htm
22. O'Hanley GP, Canny CL. Diabetic dense premacular hemorrhage: a possible indication for prompt vitrectomy. Ophthalmology 1985;92:507-511.
23. Nedelmann M, Eicke BM, Dieterich M. Functional and morphological criteria of internal jugular valve insufficiency as assessed by ultrasound. J Neuroimaging 2005;15:70-75.
24. Kwok AK, Lai TY, Chan NR. Epiretinal membrane formation with internal limiting membrane wrinkling after Nd:YAG laser membranotomy in Valsalva retinopathy. Am J Ophthalmol 2003;136:763-766.

Temperature - A Contributing Factor in a Case of Superior Oblique Palsy

Kirsty Somerville McAlester, BMedSci(Orthoptics)Hons
Julia Kelly, DOBA MHSM

Orthoptic Department, Sydney Hospital & Sydney Eye Hospital, Sydney, Australia

ABSTRACT

An unusual case of superior oblique palsy with vertical diplopia, exacerbated by temperature change is presented. The presenting symptoms raised suspicion of a clinical condition akin to that of "Uhthoff's Phenomenon" as seen in multiple sclerosis, however, the increased temperature in this patient led to diplopia not vision impairment. The clinical signs are presented, together with the investigations which were undertaken in order to determine whether the suspected diagnosis of Ocular Myasthenia Gravis (OMG) could be confirmed. The clinical findings are examined in light of current evidence regarding diagnostic tests for OMG. Special attention is given to the 'Ice Test', a simple clinical test that aids in the diagnosis of OMG.

Keywords: Ice Test, Ocular Myasthenia Gravis, superior oblique palsy, temperature.

INTRODUCTION

Superior oblique palsy is recognised as a common ocular motility defect seen in clinical practice.[1,2] A large majority of superior oblique palsies are congenital, however, acquired superior oblique palsies are also frequently encountered.[2] Numerous aetiologies for acquired superior oblique palsies are cited in the literature for example trauma, inflammation, vascular, infection and tumour.[1-3] Ocular myasthenia gravis (OMG), which can mimic any form of pupil sparing, non-restrictive ocular motility defect, is also reported as a possible, yet rare aetiology of superior oblique palsy.[4] However, it is often not possible to identify the aetiology of a superior oblique palsy, with some authors reporting 23% - 38% of cases being idiopathic. [3,5]

This paper presents an unusual case of superior oblique palsy with vertical diplopia, which was exacerbated by increased ambient temperature. The presenting symptom raised suspicion of a clinical reaction that appeared to be akin to that of "Uhthoff's Phenomenon" seen in multiple sclerosis [6], however the increased temperature led to diplopia, not vision impairment. An extensive literature search did not provide evidence of similar cases of superior oblique palsy where increased temperature contributed to decompensation and/or an increase in symptoms. However, several studies have documented increases and decreases in temperature that affect motor and sensory nerve conduction velocity in muscles and have profound effects in neuromuscular conditions such as myasthenia gravis (MG).[7-9]

Correspondence: **Kirsty Somerville McAlester**
Orthoptic Department, Sydney Hospital & Sydney Eye Hospital,
8 Macquarie Street, Sydney, NSW 2000, Australia.
Email: KirstySomerville.Mcalester@sesiahs.health.nsw.gov.au

CASE REPORT

A healthy 53-year-old male presented in mid-summer, complaining of a three-year history of reoccurring, intermittent, vertical diplopia. Three years previously there was sudden onset of diplopia accompanied by loss of depth perception, balance impairment and neck pain. The patient underwent extensive neurological and ophthalmological investigations including MRI, CT scans and a Tensilon test. To the patient's knowledge all results were within normal limits. He was however prescribed a prismatic correction which alleviated the diplopia.

The main complaint on presentation to the orthoptic department was that during periods of hot weather and high temperature an increase in vertical diplopia was experienced, which was no longer controlled by the prismatic correction incorporated in the glasses unless a compensatory head posture (CHP) was used. Interestingly, the patient reported that the prismatic correction was not

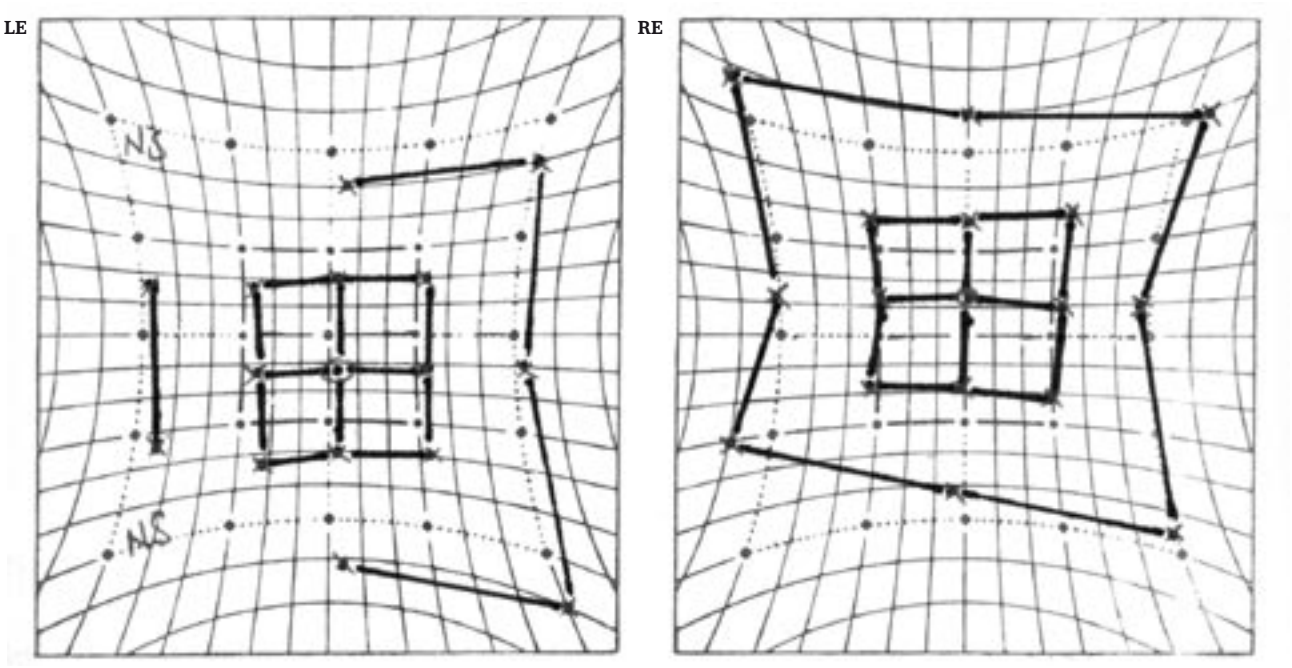


Figure 1. Hess Chart pre ice test

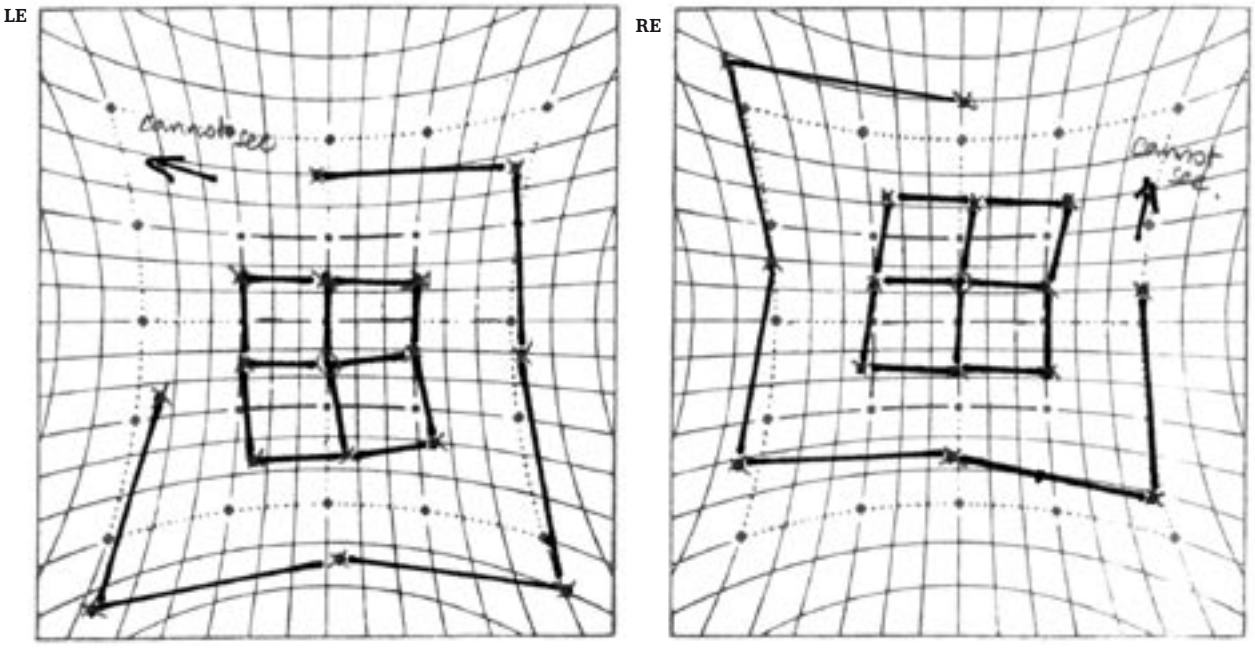


Figure 2. Hess Chart post ice test

required during the winter months or when working in his air-conditioned workshop during the summer months.

Visual acuity was 6/4 in both eyes with –0.25 DS, with normal pupil and funduscopy examinations. A right superior oblique palsy measuring eight prism diopters (PD) in primary position, increasing to 14PD in left gaze was present and confirmed by the Hess chart. The Bielschowsky Head Tilt Test result was positive for a right superior oblique palsy.

Due to the extensive investigation in the past, normal fundoscopy and the fact that the abnormality was confined to the ocular muscles no further investigations were conducted. An additional 3PD Fresnel was added to the 2PD

already incorporated in the distance and reading glasses. This alleviated the diplopia and the need for an increased CHP. On follow up visits the patient reported the prismatic correction was required less frequently during autumn and not at all in the winter.

In the following summer, the patient returned complaining of a one week history of constant diplopia, which was present with and without the prismatic correction. The patient again reported that the increase in diplopia was associated with rising temperature. In an attempt to alleviate the diplopia the patient wore his near and distance prismatic correction simultaneously. This provided a correction of 10PD, which was an increase of 5PD since the previous summer.

Ocular examination remained unchanged apart from the persistence of the right superior oblique palsy. The deviation had significantly increased over the 12-month period, which was evident on the Hess chart.

The possibility of increased temperature as a dissociating factor in superior oblique palsies was not an aetiology we had encountered. However, the patient's persistent history of an increased deviation associated with increased ambient temperature, created suspicion of a reaction similar to "Uhthoff's phenomenon".

A review of the literature revealed several studies demonstrating that increases and decreases in temperature can affect nerve conduction in muscles and can have profound effects in neuromuscular conditions such as myasthenia gravis (MG).[7-9] The physiological mechanism underlying this reaction is that increased temperature has a detrimental effect on neuromuscular transmission and muscle force generation.[10] Whereas lowering the temperature produces improvement in myasthenic muscle function, perhaps by inhibiting acetylcholinesterase function. [11]

Orbital cooling using the 'Ice Test' is an easily administered aid to differential diagnosis in OMG.[10-17] The ice test was introduced in 1979 as a simple office test for differential diagnosis of blepharoptosis in suspected OMG.[12] Although initially introduced for cases of blepharoptosis in OMG, since then several authors have used the test in aiding diagnosis of OMG in suspected ocular motility disorders.[11-14]

An ice pack wrapped in a towel was applied to the patient's right eye for a period of five minutes. On removal, the deviation in the primary position had decreased by 1 PD for distance and 2 PD for near. The Hess following the ice test showed a 10 PD recovery in the field of action of the right superior oblique compared to the pre ice test Hess (Fig 1 and 2). Subjectively, the patient reported a significant improvement in his diplopia, which was maintained for the remainder of the clinical visit.

The improvement in the Hess chart, the decrease in the deviation and the resolution of diplopia in primary position was interpreted as a positive result to the ice test. The patient was referred to a neurologist for investigations of OMG. Results of the acetylcholine receptor antibody assay and single fibre electromyography (SFEMG) of both limb and orbicularis oculi were negative.

As a result of the negative findings the suspected aetiology of OMG was excluded and the patient was diagnosed with acquired idiopathic fourth nerve palsy. The patient has since had a right inferior oblique myectomy, and is now diplopia free post operatively and has remained so for 18 months and through two summers.

DISCUSSION

The results of the OMG investigations in this case were negative, with the patient's ocular alignment restored with surgery. At present the patient remains symptom free. However, is the exclusion of a diagnosis of OMG an example of false negative response to specific OMG testing, particularly in the presence of a positive response to the ice test?

The literature highlights that the presence of negative responses to OMG investigative tests does not rule out the diagnosis of OMG[13]. Verifying the diagnosis of MG can be difficult as the diagnostic tests are not 100% sensitive or specific.[10] Furthermore, it is recognised that one-third of patients improve spontaneously in the early stages of the disease.[19] Many authors confirm that although SFEMG is considered to be the "gold standard" for diagnosis of MG, the test is known to have high false negative rates in OMG.[10,13,17-19]. The results of SFEMG are reported to be positive in 91-100% of patients with generalized MG and can vary from 63% -92% of patients with OMG.[10,17,18] The possibility of a positive response in OMG increases significantly when the orbicularis oculi is tested rather than a limb muscle. [10,13,17,19]

In addition, ACh receptor antibodies are variably elevated in MG and may not be abnormal in 30-65% of patients with OMG.[10,13,19] The significance of this result is illustrated by Ellis et al[13], who reported that if they had relied on the SFEMG test alone, "the true diagnosis of OMG would have been missed in 10 out of 14 patients" in their series. Of particular clinical significance in Ellis et al's study, where the ice test was used in conjunction with other diagnostic tests for OMG, all patients who had a positive response to the ice test had OMG. No patient with OMG had a false positive or paradoxical response to the ice test. In addition, none of the participants in the control group had a positive motility or blepharoptosis response to orbital cooling.[13] This positive response to the ice test is further supported by several other studies.[10,16,17,20]

If our patient did not have OMG how or why did he react to the ice test? Could sensitivity to temperature change be a previously unreported decompensation factor in superior oblique nerve palsies? No similar case study describing changes in ambient temperature as a contributing factor in

the decompensation of superior obliques palsy is reported in the literature. However, Larner and Thomas describe a patient with an isolated superior rectus palsy who had a negative response to the extensive OMG investigations but 'cooling' was observed to clearly have "a marked subjective and objective (Hess chart) effect on eye movements." Larner and Thomas suggested "that the underlying superior rectus underaction was due to MG (certainly very focal and unilateral presentations of MG do occur, albeit rarely) but subsequent investigations and treatment provided no evidence to support this diagnosis."[14]

Irrespective of this grey area, we have incorporated the ice test as an additional diagnostic tool in suspected cases of OMG with positive results. The use of the ice test in the diagnosis of OMG is a proven inexpensive, safe and easily administered diagnostic test, which has a high sensitivity and specificity and is a valuable adjunct to the invasive tests. [13,15,20]

Finally, the patient's accurate report of the impact of temperature changes to his symptoms and deviation should encourage us to include questions relating to temperature in history-taking. Indeed, a study of the patterns of decompensation in ocular muscle palsies compared to the time of year may be an interesting future study.

REFERENCES

1. Rosenbaum AL, Santiago AP. Management of selected forms of neurogenic strabismus. In: Rosenbaum AL, Santiago AP, editors. Clinical Strabismus Management: Principles and Surgical Techniques. Philadelphia: W.D. Saunders Company, 1999:219-223.
2. von Noorden GK. Special Forms of Strabismus. In: von Noorden, editor. Binocular Vision and Ocular Motility: Theory and Management of Strabismus. 5th edition. St. Louis: Mosby, 1996:411-412.
3. von Noorden GK, Murray E, Wong SY. Superior oblique paralysis. A review of 270 cases. Arch of Ophthalmol 1986;104:1771.
4. Rush JA, Shaffrin F. Ocular myasthenia presenting as superior oblique weakness. J Clin Neuroophthalmol 1982;2:125-127.
5. Gunderson CA, Mazow ML & Avilla CW. Epidemiology of CNIV palsies. Am Orthopt J 2001;51:99-102.
6. Kanski JJ. Neuro-ophthalmology In: Kanski JJ, editor. Clinical Ophthalmology. 2nd ed. London: Butterworth-Heinemanne Ltd, 1990:444.
7. Borenstein S, Desmedt JE. Temperature and weather correlates of myasthenic fatigue. Lancet 1974;13:63-66.
8. Borenstein S, Desmedt JE. Local cooling in myasthenia. Arch Neurol 1975;32:152-157.
9. Rutkove SB. Effects of temperature on neuromuscular electrophysiology. Muscle Nerve 2001;24:867-882.
10. Golnik KC, Pena R, Lee AG, Eggenberder ER. An Ice Test for the diagnosis of Myasthenia Gravis. Ophthalmology 1999;106:1282-1286.
11. Sethi KD, Rivner MH, Swift TR. Ice pack test for myasthenia gravis. Neurology 1987;37:1383-1385.
12. Saavedra J, Femminini R, Kochen S, deZarate JC. A cold test for myasthenia gravis [letter] Neurology 1979;29:1075.
13. Ellis FD, Hoyt CS, Ellis FJ, Jeffery AR, Sondhi N. Extraocular muscle responses to orbital cooling (Ice Test) for ocular myasthenia gravis diagnosis. J AAPOS 2000;4:271- 281.
14. Larner AJ, Thomas DJ. Can myasthenia gravis be diagnosed with the ice pack test? A cautionary note. Postgrad Med J 2000;76:162-163.
15. Miller NR. The office diagnosis of myasthenia gravis. Am Orthopt J 2000;50:64-75.
16. Movaghar M, Slavin ML. Effect of local heat versus ice on blepharoptosis resulting from ocular myasthenia. Ophthalmology 2000;107:2209-2214.
17. Kubis KC, Danesh-Meyer HV, Savino PJ, Sergott RC. The ice test versus the rest test in myasthenia gravis. Ophthalmology 2000;107:1995-1998
18. Barton JJ, Foulavand M. Ocular aspects of myasthenia gravis. Semin in Neurol 2000;20:7-20.
19. Howard JF. Myasthenia gravis: A summary. http://www.myasthenia.org/information/summary.htm November 1997
20. Lertchauanakul A, Gamnerdsiri P, Hirunwiwatkul P. Ice test for ocular myasthenia gravis. J Med Assoc Thai 2001;1(Suppl):5131-5136

Discordant Unilateral Myopia in Adult Female Monozygotic Twins

Mohamed Dirani BOrth&OphthSc(Hons)[1,2]
Matthew Chamberlain MBBS [1]
Pam Garoufalis BSc BOrth(Hons)[1,2]
Christine Chen MBBS[1,2]
Robyn H. Guymer MBBS PhD FRANZCO[1]
Paul N. Baird BSc(Hons) PhD[1,2]

[1] Centre for Eye Research Australia, University of Melbourne, Australia
[2] Vision Cooperative Research Centre, Sydney, Australia

ABSTRACT

Purpose: We present the first single case study of extremely discordant monozygotic twins for refraction, which provides an insight into the complexity of myopia.

Method: The twins were recruited through the Australian Twin Registry (ATR). Each individual completed a general questionnaire, vision examination and a blood test. Visual acuity was examined using the LogMAR chart at 3m. Cycloplegic (tropicamide 1%) refraction using the Topcon (KR 8100 model) auto-refractor and ocular dimensions (axial length, keratometry, anterior chamber depth) were obtained using the IOL master (Carl Zeiss P/L).

Results: A pair of female monozygotic twins aged 62 years with highly discordant refraction is reported. One twin member has myopic anisometropia with a difference of 7D while her identical twin has no evidence of anisometropia and is mildly hypermetropic in both eyes.

Conclusion: The twins medical and birth history fail to explain the discordance in refraction between the twin pair. There is no marked difference in environmental exposures and medical history between the twins to explain the discordance in refraction. It is possible that the discordant refraction is a result of inter-uterine trauma to twin one during embryonic development or possible injury of being born second to her twin.

Key Words: Anisometropia, discordant, heritability, myopia, refraction.

INTRODUCTION

In Australia, approximately 1 in 40 live births are twins of which 30% are monozygotic (MZ) [1]. MZ twins (genetically identical) arise from the same fertilised egg (zygote) with division of the zygote usually occurring between the 3rd and 6th day of embryonic development.

Studies have previously reported the occurrence of discordant refraction in MZ twins with a difference between 3.00 to 5.50 diopters (D)[2-3]. However, one study has reported a high discordance of 26D4. Higher degrees of refractive discordance in MZ twins were primarily due to anisometropia, strabismus and myopic retinopathy, where both eyes of the twins are affected. However, there is no reported case of MZ twins where only one twin presents with a single highly myopic eye or where there is no previous ocular or systemic history or signs of pathology in the myopic eye.

Correspondence: **Mohamed Dirani**
Centre for Eye Research Australia, The University of Melbourne,
32 Gisborne St, East Melbourne, VIC 3002, Australia.
Email: m.dirani@pgrad.unimelb.edu.au

METHOD

This twin pair were part of a larger twin study being conducted in Victoria. The twins were recruited through the Australian Twin Registry (ATR). Each individual completed a general questionnaire, vision examination and a blood test. The mono-zygosity of this twin pair was confirmed by standard genotyping using a series of 11 polymorphic markers performed by the Australian Genome Research Facility, Melbourne.

Visual acuity was examined using the LogMAR chart at 3m. Cycloplegic (tropicamide 1%) refraction using the

Topcon (KR 8100 model) auto-refractor was obtained and then checked subjectively. Ocular dimensions (axial length, keratometry, anterior chamber depth) were measured using the IOL master (Carl Zeiss P/L).

RESULTS

Both twins were born 2 weeks premature and weighed approximately 1.6kg at birth each. There was no history of low birth weight complications. This MZ pair of female twins, aged 62 years, both have hypertension that has been treated for the last ten years. Twin one has two children now aged 35 and 39 years and both have a history of strabismus. There was no other significant family history of ocular disorders.

TWIN ONE: VISION EXAMINATION

Uncorrected visual acuity was <6/60 (pinhole = 6/24) in the right eye (RE) and 6/5+ in the left eye (LE). A subjective refraction of -7.00/-1.00 x 85° in the RE and +0.50/plano in the LE improved the visual acuity to 6/9 and 6/4, respectively. From the latter results, twin one can be classified as having anisometropic amblyopia. No distance prescription has been prescribed due to the experience of binocular vertical diplopia (right image higher than left) caused by the myopic anisometropia, although reading glasses (+1.50D) have been worn since the age of 13 years. Ocular motility findings were all within normal limits with no signs of ocular muscle imbalance or deviation.

Ocular Dimensions

A difference in axial length of 3mm was observed with the myopic eye being elongated (25.25mm) compared to the non-myopic eye (22.21mm) (Table 1).

TWIN TWO: VISION EXAMINATION

Uncorrected visual acuity was 6/6 in the RE and 6/6 in the LE. Best-corrected visual acuity was 6/4.8 in the RE (+1.00/-0.50 x 85°) and 6/3.8 in the LE (+0.50). No signs of strabismus or muscle imbalance were detected upon examinations.

Ocular Dimensions

Twin two demonstrated a difference in axial length of 0.01mm (Table 1).

DISCUSSION

We report a case of extremely discordant monozygotic twins for myopic anisometropia. Myopic anisometropia in the right eye was found in twin one while her identical twin was marginally hypermetropic in both eyes. The high myopia found in twin one has been present since early childhood and has remained stable during adulthood. There was no past systemic or ocular history to explain the high myopia found in the right eye of twin one. There have been reported cases of identical twins being discordant for refraction[2-4], although reports of myopic anisometropia are extremely rare in identical twins and the general population.

Table 1. Biometric readings (AL = Axial Length)

	Right Eye AL (mm)	Left Eye AL (mm)
Twin One	25.25	22.21
Twin Two	22.19	22.18

Refractive errors and axial length measurements in the left eye in both twins were highly concordant. However, axial length measurements for the right eye in twin one were approximately 3mm longer compared to twin two. The discordance for refraction between the twins seems to predominantly reflect this increase in axial length of twin one.

Both refraction and axial length measurements have previously been shown to be highly concordant in MZ twins compared to dizygotic (DZ) twins, thus supporting a significant genetic component[5]. However, this case study indicates that MZ twins can also be discordant for refraction and axial length. It is unclear how this difference in refraction could have occurred without the involvement of obvious pathology. There is no significant difference in environmental exposures between the twins to explain the discordance in refraction. Both twins weighed the same at birth, were equally healthy as children and were brought up in a very similar environment. It is possible, however, that the discordant refraction may be a result of inter-uterine trauma to twin one during embryonic development or possible injury of being born second to her twin.

ACKNOWLEDGEMENTS

We would like to acknowledge the ATR for recruiting the twins for this twin study.

REFERENCES

1. Hall JG. Developmental Biology IV: Twinning. Lancet. 2003;362:735-743.
2. Otsuka J. The result of eye and refraction examination of 300 sets of twins. Acta Soc Ophthalmol Jap 1950;42:64:46.
3. Gedda L, Brenci G, Franceschetti A. A study of mirror imaging in twins. Prog Clin Biol Res 1981;69A:167-168.
4. Orth H. Extreme Diskordanz der Refraktion-swerte eineiiger Zwillinge. Klin Monatsbl Augenheilkd 1954;124:304-306.
5. Lyhne N, Sjolie AK, Kyvik KO, Green A. The importance of genes and environment for ocular refraction and its determiners: a population based study among 20-45 year old twins. Br J Ophthalmol 2001;85:1470-1476.

The Speed of Emmetropia

Josephine Leone, BOrth&OphthSc(Hons)[1]
Zoran Georgievski, BAppSc(Orth)Hons[1,2]
Konstandina Koklanis, BOrth(Hons)PhD[1,3]

[1] Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia
[2] Orthoptics, Northern Health, Melbourne, Australia
[3] Department of Ophthalmology, Royal Childrens Hospital, Melbourne, Australia

ABSTRACT

The disruption to the emmetropisation process is commonly cited as one important reason for not prescribing glasses for hypermetropia in infants - if, of course, there are no other indicators present (anisometropia or strabismus). The purpose of this case report is to re-visit what is considered to be a 'normal' amount of hypermetropia in infancy and the issues concerned with refractive correction and prescribing in younger children with hypermetropic errors. Using an illustrative case study, it is also aimed to highlight the speed or rate at which emmetropisation can take place.

Keywords: emmetropia, emmetropisation, hypermetropia, refractive error

INTRODUCTION

Infants are generally born hypermetropic, with a gradual decrease in this hypermetropia during the early years of life.[1-3] The process by which this change in refractive error occurs is known as emmetropisation. When infants or children are found to be hypermetropic, the clinician is often faced with challenging questions on how to manage the refractive error. The principles behind prescribing glasses in young children are different to those of adults and take into consideration cortical plasticity and emmetropisation or the influence of refractive correction on this process. We report a case study of an infant displaying a rapid change in refractive error towards emmetropia from initial higher levels of hypermetropia to highlight and discuss issues concerned with refractive correction and prescribing in younger children with hypermetropic errors.

CASE STUDY

A 5½ month old baby, N.E, presented for routine eye examination, having a family history of anisometropic amblyopia (father). On examination, no manifest deviation was apparent and ocular movements were normal. A fusion response was also demonstrated to a 'prism reflex test'. Retinoscopy following cycloplegia (cyclopentolate 1%) revealed a hypermetropic refractive error, +4.00/-1.00x180º and +3.50/-1.00x180º, in the right and left eyes respectively. However, glasses were not prescribed.

On follow-up examination at 17 months of age, N.E.'s refractive error had decreased significantly. Repeat cycloplegic retinoscopy revealed a refraction of +1.00/-0.50x180º and +0.50/-0.50x180º, in the right and left eyes respectively – a decrease in the hypermetropic and astigmatic refractive error initially present. Specifically, there was a decrease of 2.75 diopters in the spherical equivalence of each eye. The remaining ocular examination, which included a cover test, assessment of ocular movements and the prism reflex test, continued to be unremarkable.

At the final follow-up visit at 3½ years of age, N.E. had only a slight further decrease in his refractive error. Cycloplegic retinoscopy yielded refractive error of +0.75/-0.25x180º and +0.50, in the right and left eyes respectively. Further, N.E. demonstrated stereo acuity of 550'' (Lang stereotest) and visual acuity of 6/9 in each eye (Lea Symbols chart).

DISCUSSION

Low levels of hypermetropia are regarded as normal in infant populations. Studies reporting on the distribution of refractive error in healthy infants have reported that over

Correspondence: **Zoran Georgievski**
Department of Clinical Vision Sciences, La Trobe University, VIC 3086, Australia.
Email: z.georgievski@latrobe.edu.au

90% of preschool children have hypermetropia less than approximately 3D and astigmatism of less than 1.75D.[2-4] The most rapid shift in refractive error occurs within the first year of life, particularly between 3 and 12 months[1,2,5] In fact, Ingram et al[6] reported that if emmetropisation occurred, it was complete in 82% of infants by 1 year of age. In our case, N.E.'s hypermetropic refractive error was of a moderate amount and decreased from a spherical equivalent of +3.50D to +0.75D and +3.00D to +0.25D in the right and left eyes, respectively, in the space of just less than 1 year.

Studies of infants have shown that the emmetropisation process is closely related to the initial refractive error present in the first 6 months of life[7,8]. This is such that higher levels of ametropia demonstrate a more rapid decrease in refractive error relative to those with initially lower levels of ametropia. Saunders et al[8] reported that for each dioptre of hypermetropia present during the first 6 months of life, the level of ametropia present decreased by 0.06D per month between the initial refraction and 12-17 months of age. Mutti et al[7] also reported on this phenomenon, noting that higher levels of hypermetropia were related to faster rates of axial growth. This growth was subsequently effective in decreasing hypermetropia to emmetropia during the first year of an infant's life. Mutti et al[7] observed, however, that this was only effective in infants with up to 5D of hypermetropia. Infants with a hypermetropic refractive error greater than this tended not to emmetropise effectively. Conversely, infants with minimal refractive errors close to emmetropia showed little shifts or in some rare instances became myopic.

The precise mechanisms regulating the process of emmetropisation are not well understood. Experimental studies suggest that 'active' visual feedback related to focusing errors drives shifts in refractive error.[7,9-12] For instance, inducing refractive errors in chicks has been shown to be associated with changes in axial length.[10] Variations to the power of ocular components also play a role in the modulation of refractive error. The power of the cornea and crystalline lens has been shown to be inversely correlated to axial growth, but it is thought that this is most likely related due to the growth of the eye rather than being the mechanism for emmetropisation.[7]

Understanding the emmetropisation process has important clinical implications for the management of refractive errors in infants. Given that mild to moderate amounts of hypermetropia 'emmetropise' in straight-eyed infants, there is little need to prescribe glasses for this population. However, the level of hypermetropia that requires correction is contentious. Despite this, the American Academy of Ophthalmology (AAO)[13] has provided guidelines for prescribing glasses in young children and suggests that the threshold for prescribing glasses for hypermetropia in children up to 1 year of age is 6D. The threshold for children aged up to 2 years is 5D and for children up to the age of 3, 4.5D. Spectacles are recommended if correction improves visual acuity or ocular alignment for children 4 years or above.

The AAO further suggests that hypermetropic correction should be reduced by up to 2D in children (except where esotropia is present and full correction is required for optimal alignment). Under-correction of hypermetropia is often recommended on the basis that the emmetropisation process can be impeded by correcting hypermetropic refractive errors.[6,14] Ingram et al[6,14] reported that the consistent wearing of hypermetropic spectacle correction from the age of 6 months was associated with the maintenance of high levels of hypermetropia. However, the effect of spectacle correction continues to be debated. Atkinson et al[15,16] have, for instance, reported that spectacle correction only has a transient effect on refractive error between 9 and 18 months of age and that treatment does not impede emmetropisation in the longer term.

This paper highlights the issues concerned with prescribing hypermetropic correction in the paediatric population. In the case report presented, it is shown that infants with initial moderate hypermetropia levels, similar to those with mild hypermetropia, do emmetropise during their first year of life. This, alongside various cross-sectional and longitudinal studies,[1,2,4-6] suggest that in the absence of strabismus and/or anisometropia, infants with moderate amounts of hypermetropia can be conservatively managed and monitored by regular cycloplegic refractions, rather than have glasses prescribed. The immediate correction of refractive error should best be avoided. Persistent high levels of hypermetropia beyond the first year may warrant treatment, especially if there is no improvement.

REFERENCES

1. Saunders K. Early refractive development in humans. Surv Ophthalmol 1995;40:207-216.
2. Mayer D, Hansen R, Moore B, Kim S, Fulton A. Cycloplegic refractions in healthy children aged 1 through 48 months. Arch Ophthalmol 2001;119;1625-1628.
3. Kuo A, Sinatra R, Donahue S. Distribution of refractive error in healthy infants. J AAPOS 2003;7;174-177.
4. Ottar W, Scott W, Holgado S. Photoscreening for amblyogenic factors. J Pediatr Ophthalmol Strabismus 1995;32:289-295.
5. Wood I, Hodi S, Morgan L. Longitudinal change of refractive error in infants during the first year of life. Eye 1995;9:551-557.
6. 6. Ingram R, Arnold P, Dally S, Lucas J. Emmetropisation, squint, and reduced visual acuity after treatment. Br J of Ophthalmol 1991;75: 414-416.
7. Mutti D, Mitchell G, Jones L, et al. Axial growth and changes in lenticular and corneal power during emmetropization in infants. Invest Ophthalmol Vis Sci 2005;46:3074-3080.
8. Saunders K, Woodhouse J, Westall CA. Emmetropisation in human infancy: rate of change is related to initial refractive error. Vision Res 1995;35:1325-1328.
9. Hung L, Crawford M, Smith E. Spectacle lenses alter eye growth and the refractive status of young monkeys. Nat Med 1995;1:761-765.

10. Irving E, Callender M, Sivak J. Inducing myopia, hyperopia and astigmatism in chicks. Optom Vis Sci 1991;68:364-368.
11. Shaeffel F, Howland H. Properties of the feedback loops controlling eye growth and refractive state in the chicken. Vision Res 1991;31:717-734.
12. Smith E, Hung L. The role of optical defocus in regulating refractive development in infant monkeys. Vision Res 1999;39:1415-1435.
13. American Association of Ophthalmology. Pediatric Eye Evaluations: Preferred Practice Guidelines. San Francisco: American Academy of Ophthalmology, 2002.
14. Ingram R, Gill L, Lambert T. Effect of spectacles on changes of spherical hypermetropia in infants who did, and did not, have strabismus. Br J Ophthalmol 2000;84:324-326.
15. Atkinson J, Anker S, Bobier W, et al. Normal emmetropization in infants with spectacle correction for hyperopia. Invest Ophthalmol Vis Sci 2000;41:3726-3731.
16. Atkinson J, Braddick O, Bobier B. Two infant vision screening programmes: prediction and prevention of strabismus and amblyopia from photo and videorefractive screening. Eye 1996;10:189-198.

Acquired Colour Vision Assessment – Is Ishihara Really Enough?

Kylie Green, BAppSci(Orth)

Orthoptic Department, Sydney Hospital & Sydney Eye Hospital, Sydney, Australia

ABSTRACT

Purpose: A quality improvement project was conducted to determine the most appropriate colour vision test for investigating acquired colour vision loss. The Ishihara Pseudoisochromatic Plate Test (Ishihara) is commonly used in many clinical settings for detecting acquired colour vision defects. However, the Ishihara is a screening test specifically designed to detect congenital red-green colour vision defects. While optic nerve pathology often causes red-green defects, this is not the same as a congenital loss. There is a strong need for in depth colour vision testing to be routinely utilised in clinical settings as colour loss is often the first sign of pathology.

Method: Thirty patients aged 23-76 with suspected optic nerve or macula pathology who were referred to the orthoptic department for colour vision testing, were assessed with the Farnsworth-Munsell 100 Hue (FM 100), Roth 28 Hue (Roth 28) and the Ishihara. The results of these three tests were compared. A control group consisting of 10 patients with no identified pathology were also tested with the FM 100 to ascertain if reliable results can be achieved despite the length of the test.

Results: Of the thirty patients, only one patient had a defect on the Ishihara. The Roth 28 did detect some losses, however, the defects were minimal and not clustered thus the abnormality could not be classified or monitored. The FM100 detected significant changes or abnormalities in colour vision in half of the 30 patients. The 10 control subjects all fell within the high to normal colour discrimination range on the FM 100 hue, with no abnormal axial patterns reported. The significance of these findings is demonstrated by three case studies, which outline the necessity of utilising the FM 100.

Conclusion: The comparison of all three tests shows that the Ishihara is not sensitive enough to detect acquired colour loss and is a poor substitute. While the Roth 28 does detect some colour changes it does not show enough detail to be a useful diagnostic tool. Colour vision testing is a key diagnostic tool and the correct test should be utilised in all clinical settings to aid diagnosis and to monitor progression or regression of optic nerve and macula pathologies.

Key words: colour vision, Ishihara, Farnsworth-Munsell, optic nerve pathology, acquired colour defects, Roth 28 hue.

INTRODUCTION

Colour vision defects can be acquired and may be among the earliest symptoms of ocular damage from disease or toxicity.[1] It is vital to accurately identify and quantify colour vision changes as this can lead to early diagnosis and also identify changes in disease status.

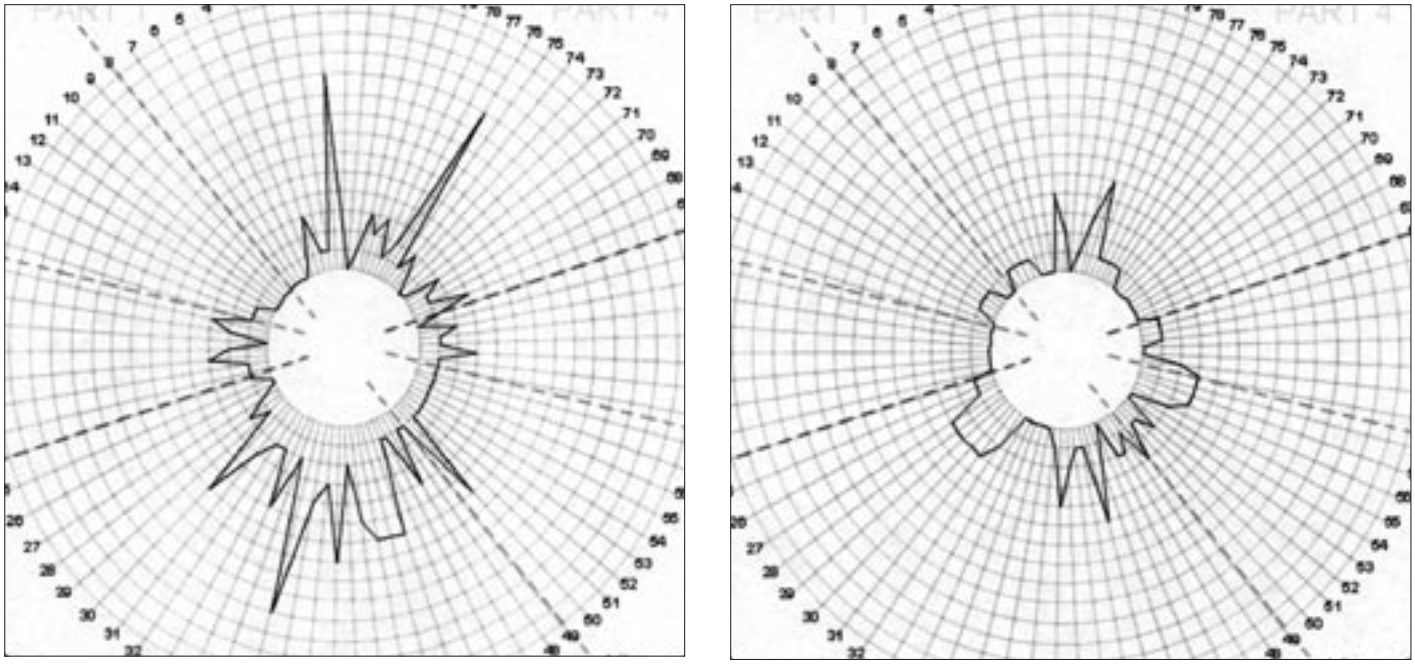
While most clinicians do consider colour vision when assessing patients with suspected optic nerve or macula pathology, many solely rely on the Ishihara, which is quick and easy to administer.[3] However, the Ishihara was designed to detect congenital red-green abnormalities, "acquired abnormalities were not taken into consideration in designing the test".[2] The Ishihara distinguishes normal colour vision from congenital colour vision defects, however, it does not evaluate the colour discrimination abilities of those with normal colour vision.[1] It is therefore inappropriate to use this test when testing patients with suspected acquired abnormalities.

Tests that are designed to detect congenital abnormalities fail to accurately detect acquired colour loss because of the different mechanisms involved in causing each abnormality.[1] Congenital loss is most commonly red-green but can be blue-yellow and is caused by either a missing cone pigment or cones with abnormal absorption.[1,2] Acquired loss, on the other hand, occurs due to changes in pre-receptor filters such as lens opacities, selective damage to specific cone classes or disruption to post-receptor processing.[1,3]

Correspondence: **Kylie Green**
Orthoptic Department, Sydney Hospital & Sydney Eye Hospital,
8 Macquarie Street, Sydney, NSW 2000, Australia.
Email: Kylie.Green@sesiahs.health.nsw.gov.au

Table 1. Verriest's Classification of Acquired Colour Vision Abnormalities

	Visual Acuity	Red-green Loss	Blue-yellow Loss	Typical Pathology
Type 1 (Protan- like)	Moderate to severe loss	Mild to severe loss	Little or no loss	Early destruction of foveal function with poor VA and central scotoma
Type 2 (Deutan- like)	Moderate reduction	Mild to severe loss	Mild loss	Optic nerve disease if VA affected
Type 3 (Tritan-like)	Normal to moderate loss	Less impairment than blue-yellow	Mild to moderate loss which is greater that the red green loss	Optic nerve and macular pathology where VA is preserved



a) b)

Figure 1. FM 100 left eye
a) On Ethambutol TES= 342 b) After ceasing Ethambutol TES = 266

Many clinicians often follow the principles of Kollner's rule which states that "patients with retinal disease develop blue-yellow discrimination loss, whereas optic nerve disease causes red-green discrimination loss".[3] However, it is now known that outer retinal damage can also cause red-green defects and that blue defects can result from optic nerve pathology, as in early glaucoma where blue-yellow discrimination loss is often noticed in the early stages of pathology.[1,3] This is likely to be due to size and concentration of blue-yellow sensitive ganglion cells. As stated by Pacheco, Sahraie and Edgar[3], blue-yellow sensitive ganglion cells or their axons have greater receptive fields, are larger than red-green cells and have a unique morphology and connectivity to second order neurons. Therefore, due to the anatomy of these cells if one blue-yellow coded ganglion cell is damaged there is a more dramatic effect on colour discrimination than if one red-green ganglion cell is damaged as there are "fewer ganglion cells which code blue-yellow signals and little overlap between receptive fields".[3]

Verriest reclassified acquired colour abnormalities into three categories, which relate the type of acquired loss to the retinal distribution of ganglion cells in the area of pathology[3]. Table 1 describes the fundamental principles of Verriest's classification. This classification highlights that when visual acuity is preserved (early stages of pathology) blue-yellow defects occur. It is only when the disease progresses and visual acuity is affected that red-green defects do also occur. This classification also highlights that acquired colour defects are often mixed affecting both red-green and blue-yellow discrimination.

The Ishihara may detect the red-green defects of advanced colour vision anomalies, however, when the colour abnormality has progressed to this stage the patient may not have the level of visual acuity necessary, as best corrected vision of 6/18 is required to accurately resolve the test.[3] Furthermore, the Ishihara is likely to miss the majority of early abnormalities, which only involve blue-yellow defects, as the test does not contain designs for the

detection of tritan defects. While this test is able to "isolate certain factors of colour deficiency it does not measure general colour discrimination"[4] and is therefore also unable to quantify the severity of this loss.

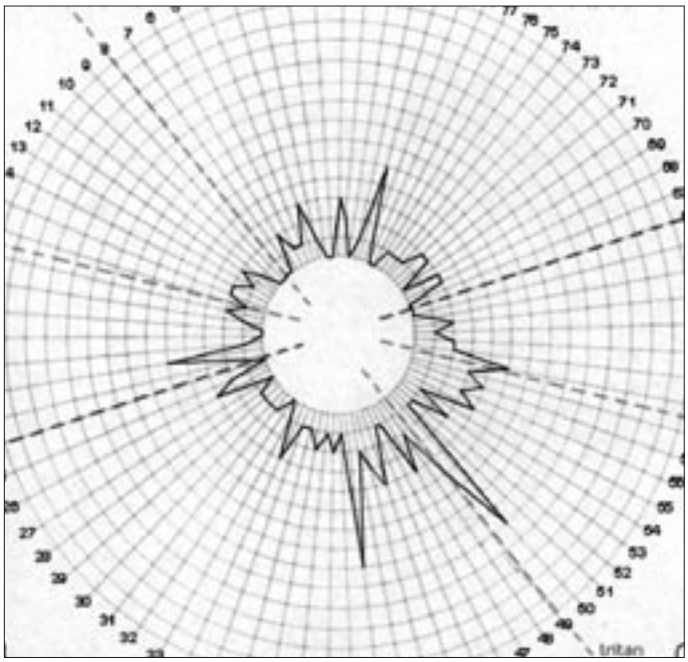
A more appropriate and sensitive test for detecting acquired colour vision abnormalities is the FM 100.1,[5] As stated by Farnsworth, this test was designed to separate persons with normal colour vision into classes of superior, normal and low colour discrimination and to measure the zones of confusion in patients with defective colour vision.[6] While this test is more time consuming than the Ishihara, it provides a more thorough evaluation. The FM 100 is able to both classify the colour abnormality by graph formation and provide a quantitative evaluation by means of the Total Error Score (TES), which allows changes in the abnormality to be monitored.[3,5] Although the FM 100 is particularly useful in monitoring progression in acquired deficiency, it is unable to distinguish subtle differences, such as between severe trichromatic anomalies and pure dichromacy. This can only been determined with the Nagel Anomaloscope.[3]

Another clinical test used for detecting acquired colour vision defects is the Roth 28. This is effective in detecting several severe acquired abnormalities however, it may not be sensitive enough to detect subtle defects, which could predict the onset, severity or progression of pathology. Furthermore, the Roth 28 does not give an exact score making it difficult to monitor subtle changes.[5]

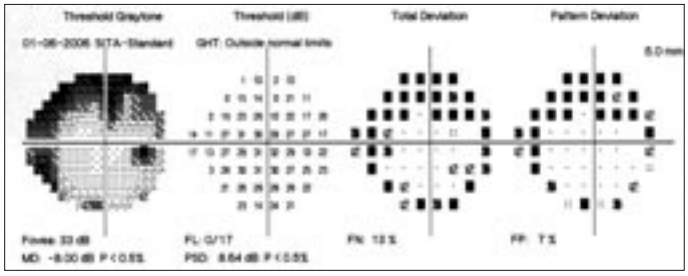
Having identified from the literature, possible shortfalls of the most commonly used colour vision tests and the physiology of acquired colour vision abnormalities, we conducted a quality improvement project to compare colour vision tests and evaluate the appropriateness of these tests in assessing acquired colour vision abnormalities. This paper presents these results of this study with particular emphasis on several cases to highlight the clinical differences between the tests.

METHOD

Ethics approval was gained to conduct a quality improvement project to ensure the Orthoptic Department at Sydney Eye Hospital utilises the most appropriate colour vision test when investigating acquired colour vision defects. The study consisted of all patients referred to the orthoptic clinic over a six month period for colour vision assessment due to suspected or diagnosed optic nerve or macular pathologies in the presence of no other documented ocular pathology. Patients with other ocular pathologies were excluded. There were a total of 30 patients, aged 23-76 with an mean age of 46 years. Best corrected visual acuity, fundus examination and Humphrey Visual Field tests were performed with additional tests such as contrast sensitivity and radiological imaging carried out when appropriate. An ophthalmologist assessed all patients.



a)



b)

Figure 2.
Pre Steroid Treatment right eye
a) RE FM 100 Hue TES = 314 b) RE Humphrey Visual Field Test

The purpose of colour vision assessment was to either identify the severity of the pathology or assist in confirming its presence. Colour vision was assessed using the Ishihara, Roth 28 and FM 100. Each eye was tested separately with near correction and the assistance of a blue daylight globe to give optimal and even illumination. The order of the tests was randomised and all tests were performed at the one visit. Of the 30 patients, 24 had both their eyes tested, whilst for 6 patients only one eye was tested due to existing pathology of the fellow eye. In total 54 eyes were assessed.

The 24-plate edition of the Ishihara test was used with the patient given three seconds to respond to each page as suggested in the instruction manual.[2] Plates one to fifteen were tested and, as stated in the manual, colour vision was considered to be normal if thirteen or more plates were read correctly.[2]

When performing the Roth 28 and FM 100 the patients were given an unlimited amount of time to complete the test and an opportunity to review placement and make any changes. The patients were instructed in the correct handling of the colour chips and supervised at all times to ensure the test

Table 2. Individual Patient Results

Patient ID	Age	Pathology	Corrected VA	Ishihara	Roth 28	FM 100
1	43	Optic Neuritis	R 6/7.5 L 6/6	15/15 R&L	No errors	Score = 286 Score = 318
2	76	Glaucoma	R 6/7.5 L 6/6	15/15 R&L	No errors	Score = 298 Score = 318
3	69	TED	R 6/12- L 6/6	15/15 R&L	R no errors, L 3 cap reversals	Score = 266 Score = 258
4	42	TED	R 6/6	15/15 RE	No errors	Score = 214
5	30	Optic Neuritis	R 6/9 L 6/12	15/15 R&L	No errors	Score = 234 Score = 218
6	66	Empty Sella Syndrome	R 6/7.5 L 6/6	15/15 R&L	R No errors, L 1 cap reversal	Score = 314 Score = 310
7	39	Optic Neuritis	R 6/6 L 6/9	15/15 R&L	R 2 reversals, L 2 reversal 1 error	Score = 294 Score = 338
8	38	Optic Neuritis	R 6/6 L 6/6	15/15 R&L	R 3 reversals, L No errors	Score = 298 Score = 202
9	51	Optic Neuritis	R 6/6	15/15 RE	R 3 errors	Score = 238
10	63	Ethambutol	R 6/6 L 6/9	15/15 R&L	No errors	Score = 306 Score = 342
11	53	Plaquenil	R 6/7.5 L 6/6	15/15 R&L	No errors	Score = 210 Score = 226
12	47	TED	R 6/6 L 6/7.5	15/15 R&L	No errors	Score = 218 Score = 194
13	62	RP	R 6/15 L 6/21	15/15 R&L	No errors	Score = 230 Score = 206
14	48	TED	R 6/6 L 6/6	15/15 R&L	R & L 1 reversal	Score = 234 Score = 262
15	24	Optic Neuritis	R 6/6 L 6/15	15/15 R&L	No errors	Score = 198 Score = 446
16	32	R Optic Neuritis (severe)	L 6/5	15/15 LE	1 reversal	Score = 314
17	31	Optic Neuritis	R 6/6 L 6/6	15/15 R&L	No errors	Score = 258 Score = 266
18	57	TED	R 6/6 L 6/12	15/15 R&L	R 2 reversal, L 2 error 1 reversal	Score = 238 Score = 286
19	31	Papilledema	R 6/12 L 6/9	15/15 R&L	R 1 reversal, L 3 reversals	Score = 322 Score = 370
20	35	TED	R 6/6 L 6/6	15/15 R&L	R No errors, L 2 errors, 2 reversal	Score = 342 Score = 322
21	33	Optic Neuritis	R 6/12 L 6/30	15/15 R&L	No errors	Score = 238 Score = 274
22	56	TED	R 6/6 L 6/9	15/15 R&L	No errors	Score = 238 Score = 210
23	62	TED	R 6/5 L 6/6	15/15 R&L	No errors	Score = 194 Score = 210
24	64	R CRVO, L Optic Atrophy	R 6/7.5 L HM	R strong deutan	R Strong deutan	Score = 306
25	23	Optic Neuritis	R 6/6 L 6/5	15/15 R&L	No errors	Score =226 Score = 226
26	59	TED & MG	R 6/9 L 6/6	15/15 R&L	1 reversal R & L	Score = 310 Score = 294
27	47	TED	R 6/6 L 6/12	15/15 R&L	R 2 reversals ,L 2 reversals	Score = 234 Score = 194
28	44	TED	L 6/4	15/15 R&L	No errors	Score = 250
29	40	TED	R 6/6 L 6/6	15/15 R&L	No errors	Score = 202 Score = 186
30	41	Optic Neuritis	R HM L 6/9	15/15 LE	L 2 reversals	Score = 314

TED = Thyroid Eye Disease, RP = Retinitis Pigmentosa, MG = Myastenia Gravis, CRVO = Central Retinal Vein Occlusion

was being performed accurately.

The results of the Roth 28 were recorded by the examiner and plotted on the hue circle recording sheet. Colour vision was considered normal when the hue circle was a complete circle, or only minor errors or reversals occurred that were not clustered in the one area. The results were considered abnormal when the cap errors were clustered or created lines that crossed the hue circle.[5]

The results of the FM 100 were recorded by the examiner and calculated to obtain the hue circle and TES. A perfect score was considered to be 170, superior discrimination 171-186, normal discrimination 190-270 and low discrimination scores greater than 270.[5,6]

The results of each of the three tests were recorded on a spreadsheet along with the patients' age, best corrected visual acuity and pathology or suspected pathology. The results of the three tests were compared to ascertain if the Ishihara is able to detect acquired colour vision loss, or should a more detailed colour vision assessment be carried out in patients with suspected or diagnosed pathology.

As the test duration and performance requirements have been identified as major drawbacks of the FM 100, 10 control subjects with no ocular pathology were randomly selected to undergo the test procedure.[1,3,5] The purpose of this was to evaluate if accurate results could be achieved on the first attempt, despite the length and difficulty of the test. The subjects in the control group were aged 20 - 55 with a mean age of 40. A hue circle and TES were formulated and recorded by the examiner on a spreadsheet.

RESULTS

Of the 30 patients with pathology or suspected pathology 1 patient had an abnormal Ishihara result in one eye. All other patients scored within the normal range with the Ishihara in each eye tested.

The results of the Roth 28 showed 20 patients had no errors in one or both eyes. Twelve patients had errors in adjacent caps in one or both eyes. These errors were considered to be within normal given that "the simple transposition of chips represents minor errors which are only significant where there is accumulation or a particular distribution".[7] Five patients had errors in one or more eyes that showed clustered errors or errors that crossed the hue circle. The specific colour defect could be ascertained in only one patient who had a deutan-like abnormality. The other patients' errors did not lie on one specific axis, therefore, the type of colour abnormality could not be classified.

The results of the FM 100 showed 15 patients had superior to normal colour discrimination in one or both eyes. Three of these 15 patients had one eye assessed. The remaining 15 patients had low colour discrimination with abnormal hue circles in one eye or both eyes. Three of these patients had one eye only assessed.

The 10 control patients were all able to achieve total error scores which fell in the superior or normal colour discrimination ranges, with normal hue circle patterns. These results do not imply there is no learning effect when performing the FM100, rather it highlights that this may not impact greatly on the final result.

The results showed that colour vision abnormalities were detected in 50% of the patients when using FM 100, while the Roth 28 detected 17% percent and the Ishihara only 3%. Therefore, if the Ishihara or Roth 28 had been used solely, a significant proportion of patients with acquired colour vision abnormalities would have been presumed to have normal colour vision.

The following three cases have been selected to demonstrate how critical colour vision assessment is to patient management and highlight that Ishihara and Roth 28 are not sensitive enough in detecting what can be gross colour abnormalities.

CASE 1

Mr L was diagnosed with respiratory tuberculosis in May 2006. His treatment included Isoniazid 300mg mane and Ethambutol 1000mg mane daily. He was referred to the orthoptic department for routine screening for optic nerve toxicity from Ethambutol. Testing revealed best corrected visual acuity of 6/9 right and 6/6 left, normal visual fields, normal contrast sensitivity with the CSV1000 and fundus examination by the ophthalmologist was unremarkable.

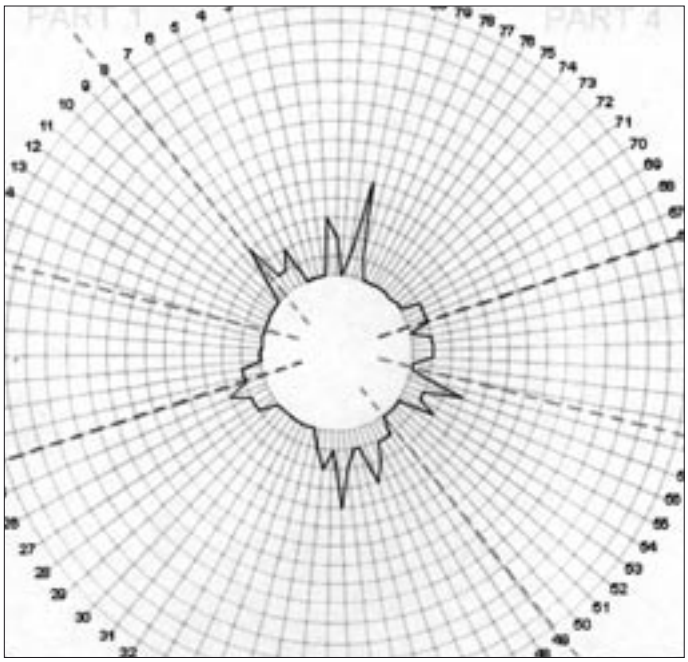
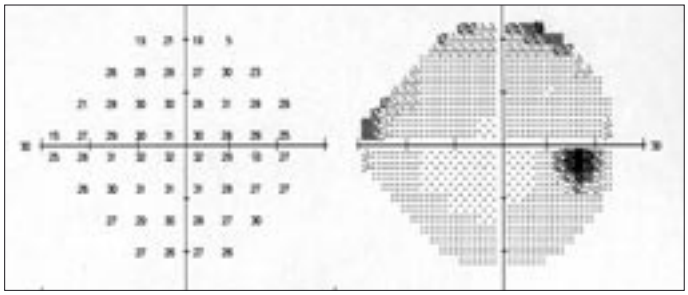
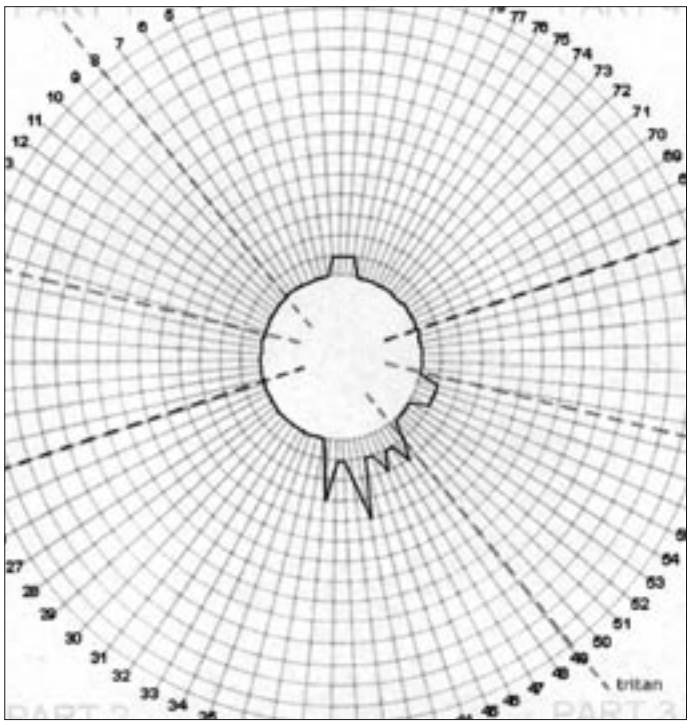


Figure 3. a)
Post Steroid Treatment
a) RE 100 Hue TES = 234 b) RE Humphrey Visual Field Test b)

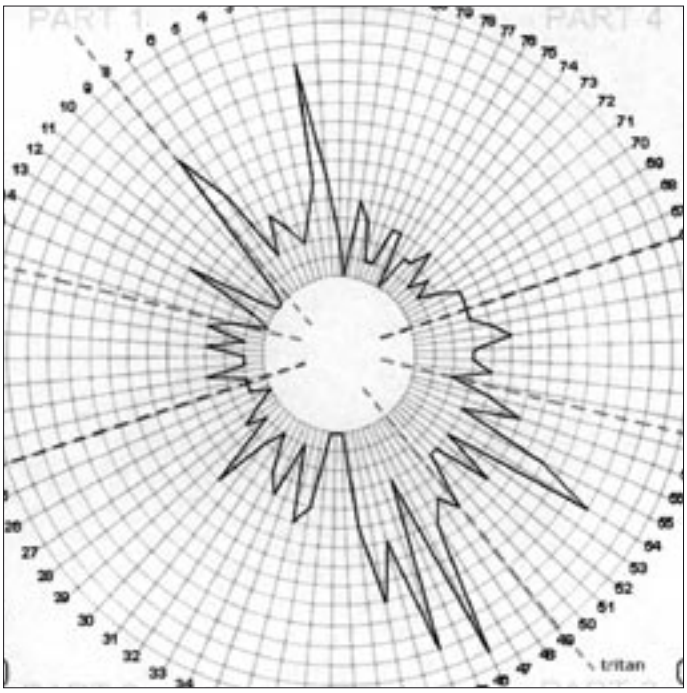


Colour vision testing was normal with the Ishihara and Roth 28, however the FM 100 showed gross tritan defects with TES of 362 in the right eye and 354 in the left. The FM 100 was repeated the following day and while there was a slight improvement, defects along the tritan axis were still present bilaterally. Ethambutol treatment was continued on the basis that all other tests were within normal limits and assessment was repeated four weeks later. At this visit fundus examination and Ishihara were normal and the Roth 28 was normal right and abnormal (tritan) left. The FM 100 showed gross tritan defects with TES of 306 in the right eye and 342 in the left eye.

Since commencing treatment, Mr L had also developed an itchy rash over his arms and lower body. Several treatments were tried throughout his admission but none could relieve the rash. It was then thought Mr L was having a reaction to the Ethambutol. A decision was made to discontinue Ethambutol based on this and his continued colour vision loss. The FM 100 hue was repeated four weeks later and improvement was seen. The right eye had improved to a TES of 286 and



a)



b)

Figure 4. Farnsworth Munsell 100 Hue
a) RE TES = 198 b) LE TES = 446

the left eye to within normal with a TES of 266 (Fig 1).

CASE 2

Mrs S was diagnosed with a left orbital pseudotumour in April 2004, causing fibrosis of the inferior oblique and a left superior scotoma. She was treated with oral prednisone until February 2005. When the visual fields were retested in May 2006, there had been a marked deterioration, involving bilateral superior scotomas. Colour vision was tested and was normal right and left with the Ishihara, normal right and left with the Roth 28 and outside normal discrimination right and left with the FM 100 (Fig 2). The right eye had a TES of 314 and the left eye a TES of 310. In both eyes the hue circle showed a tritan-like defect. It was thought Mrs S may have compressive thyroid optic neuropathy, however, all blood tests disputed this. A diagnosis of empty sella syndrome was made and Mrs S commenced intravenous steroids initially then oral steroids. The diagnosis is still under review, however, with the steroid treatment both the fields and colour vision have improved (Fig 3).

CASE 3

Mr M presented with a sudden onset of blurred left visual acuity with loss of peripheral visual field in the left eye. Visual acuity was 6/6 in the right eye and 6/15 in the left eye. He was referred to the orthoptic department for colour vision assessment due to suspected optic neuritis. The Ishihara and Roth 28 hue were normal in both eyes. The FM 100, however, showed a normal discrimination in the right eye with TES of 198 and grossly abnormal left eye discrimination with a score of 446 (Fig 4). Following treatment with oral prednisone Mr M's colour discrimination returned to within normal with a TES of 220.

DISCUSSION

The above cases outline the importance of accurately measuring colour vision, as it can be a critical factor in diagnosis and management. In the case of Mr L, the colour vision assessment with the FM 100 detected optic nerve toxicity at initial investigation. However, as the visual fields were normal it was considered that toxicity was not present. The fact that Mr L's colour discrimination improved after Ethambutol was ceased demonstrates that colour vision is an accurate method of assessing optic nerve function and possible toxicity. The case of Mrs S outlines that the changes occurring in visual fields mirrored changes in colour discrimination making accurate colour vision assessment as important as visual field assessment.

The case of Mr M also clearly highlights the inadequacy of the Ishihara and Roth 28, as a severe acquired colour abnormality registered normal on these tests. As shown in the literature review, acquired and congenital colour vision defects are two separate conditions that require different tools of investigation. If Ishihara is the sole test used in the investigation of acquired colour loss, severe colour defects may not be detected at all. Although the Roth 28 was designed

to detect both congenital and acquired colour defects, the findings of this study suggest that it is not sensitive enough to be a useful diagnostic tool when assessing acquired colour vision. In addition, as no error score is given, it is difficult to monitor progression and as is highlighted in the first case, monitoring progression of colour discrimination can be significant to patient management.

While the FM100 can be time consuming, in reality only a small amount of patients seen in an ophthalmic clinic will need this test. If more time and care is given to assessing these patients it may lead to earlier diagnosis and an increased ability to track changes in the disease process. Colour vision is a vital assessment for patients with optic nerve or macular pathology and should be adequately investigated.

CONCLUSION

The comparison of all three tests shows that the Ishihara is not sensitive enough to detect acquired colour loss and is a poor substitute. While the Roth 28 does detect some colour changes it does not show enough detail to be a useful diagnostic tool as compared to the FM 100. As a result of this quality improvement study, the orthoptic department at Sydney Eye Hospital now routinely perform the FM 100 on all patients referred for colour vision assessment due to macular or optic nerve pathology.

REFERENCES

1. Swanson W, Cohen J. Colour vision. Ophthalmol Clin North Am 2003;16:179-203.
2. Ishihara S. The Series of Plates Designed as Tests for Colour Blindness: Instruction Manual. Tokyo: Kanehara Shuppan Co., Ltd, 1939.
3. Pacheco M, Sahraie A, Edgar D. Acquired colour vision defects in glaucoma: their detection and clinical significance. Br J Ophthalmol 1999;83:1396-1402.
4. Bayer A, Thiel H, Zrenner E, et al. Colour vision tests for early detection of antiepileptic drug toxicity. Neurology 1997;48:1394-1397.
5. Fitzgerald A. A review of the Farnsworth Munsell type colour vision tests. Aust Orthopt J 1991;27:25-36.
6. Farnsworth D. The Farnsworth-Munsell 100 Hue Test for the Examination of Color Discrimination: Instruction Manual. Paris: Luneau Ophthalmologie, 1976.
7. Roth A. The Roth 28 Hue Test according to Farnsworth-Munsell: Instruction Manual. Paris: Luneau Ophthalmologie, (undated).

Intermittent Exotropia: A Review of the Natural History and Non-surgical Treatment Outcomes

Kailin Karen Zhang[1]
Konstandina Koklanis, BOrth(Hons) PhD[1,2]
Zoran Georgievski, BAppSc(Orth)Hons[1,3]

[1] Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia
[2] Department of Ophthalmology, Royal Childrens Hospital, Melbourne, Australia
[3] Orthoptics, Northern Health, Melbourne, Australia

ABSTRACT

Intermittent exotropia of the divergence excess type is the most common form of exotropia. Given the intermittent nature of this deviation, and that it can be controlled by fusional vergence but has the potential to develop sensory anomalies such as suppression, management options include non-surgical as well as surgical means. The aims of non-surgical or orthoptic treatment are to improve fusion, eradicate suppression and or teach control of the deviation, in order to decrease the frequency of the manifest phase and improve motor alignment for near and distance. This review focuses on the outcomes of various non-surgical treatments, including orthoptics, and discusses the natural history of intermittent exotropia which invariably has implications for management.

Keywords: intermittent exotropia, distance exotropia, divergence excess, orthoptics, natural history

INTRODUCTION

Intermittent exotropia X(T), the most common form of childhood exotropia, occurs in approximately 1% of the general population and 25% of strabismic children worldwide.[1] It is an ocular deviation demonstrating ortho- or exophoria when controlled by positive fusional vergence, or a manifest deviation with variable sensory adaptations when it is not.[2] Burian's classification system (Figure 1) divides X(T) into various types (shaded), based on the size of the near and distance deviations and state of fusional control.[3] Unless otherwise specified, the focus of this review will be on the most common X(T), divergence excess type or 'distance exotropia', where the angle of deviation at distance fixation is greater than that at near.

Non-surgical treatment modalities for X(T) have been described throughout the literature. All aim to decrease the frequency of the manifest phase of the strabismus, improving fusion and motor alignment for near and distance.[4] However, conflicting and limited knowledge of the natural history of X(T) and its non-surgical treatment outcomes hinders formulation of the best management plan for patients. The aim of this paper is to review the literature concerning the natural history of X(T) and its non-surgical management including outcomes.

Correspondence: **Zoran Georgievski**
Department of Clinical Vision Sciences, La Trobe University, VIC 3086, Australia.
Email: z.georgievski@latrobe.edu.au

NATURAL HISTORY

The natural history of X(T) remains uncertain. Whilst some have suggested it is a progressive disorder left untreated,[5,6] others have reported stabilisation or even improvement of the condition over time.[7-10] Table 1 presents a summary of the findings of various studies examining the natural history of X(T).

Progression of X(T) can be defined as an increase in the size or frequency of the exotropia, either at near or distance, with increasing suppression and loss of stereopsis.[2] This process relies upon the patient's fusional reserve and is expedited by the development of abnormal sensory patterns and more widely-spaced facial features.[11]

Hiles, Davies and Costenbader[7] conducted a study on 48 patients (primarily divergence-excess type), who underwent observation, but also non-surgical treatment, for a mean period of 11.7 years. They found that the larger the deviation, the greater the reduction in measurement at final follow-up. Most patients, however, remained within 10Δ of

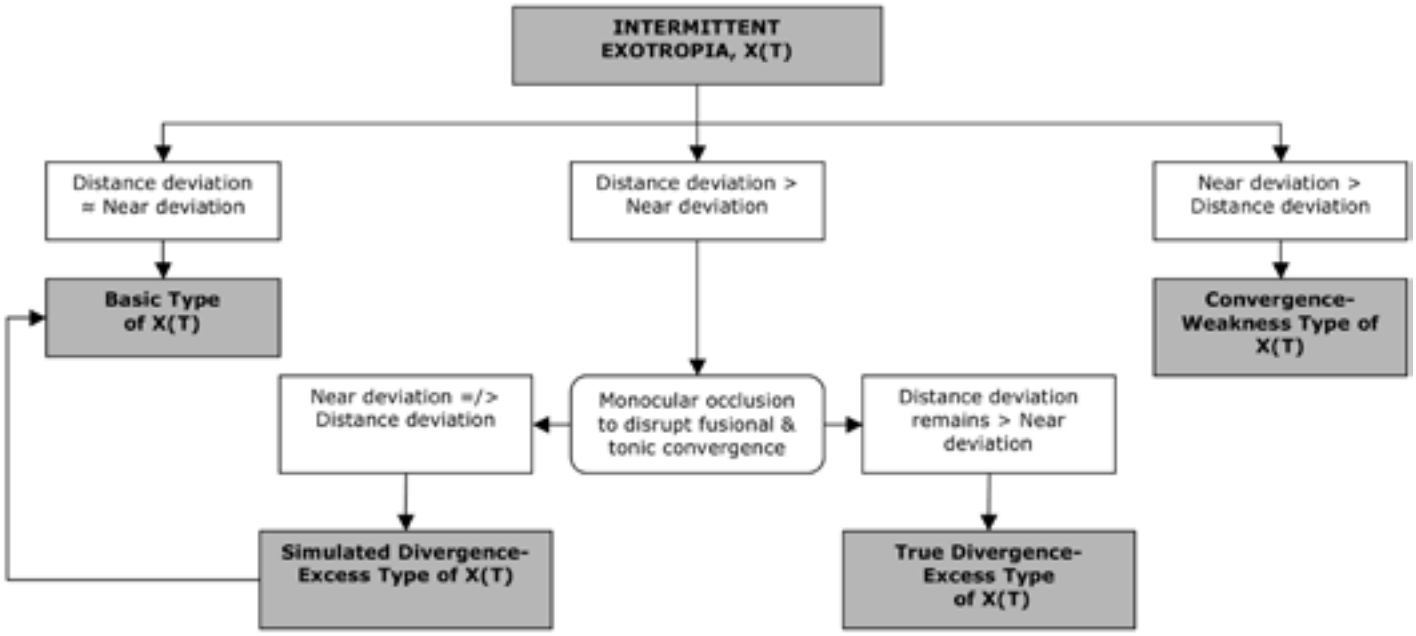


Figure 1. Burian's Classification of X(T).[3]

Table 1. Summary of studies on the natural course of X(T).

Study and Design	N	Mean age of onset and/or presentation	Mean follow-up	Outcomes
Hiles et al[7] Retrospective	48	Onset: 2.8 years Pres.: 4.8 years	11.7 years	83% stayed within 10Δ of distance deviation; 65% became exophoric in the distance
Chia et al[8] Retrospective	287	Onset: 3 years Pres.: 6.1 years	3 years	48% remained within 5Δ of distance deviation; 63% had stable control of distant exotropia
Clarke et al[10] Prospective	168	Pres.: 3.7 years	6-24 months	< 1/3 deteriorated in the control of deviation; 87% maintained or improved stere oacuities
Romanchuk et al[9] Retrospective	109	Onset: 33 months Pres.: 8 years	9 years	58% retained their distance deviation; 51% retained control of deviation
Rutstein & Corliss[2] Retrospective	73	Pres.: 20 years	10 years	36% became exo- or ortho-phoric; 67% of the deviations stabilized or decreased for distance and 81% for near
Von Noorden & Campos[6] Prospective	51	Pres.: 5-10 years	3.5 years	75% progressed; 25% improved or unchanged
Nusz et al[5] Retrospective	138	Pres.: 6.3 years	5.6 years	>50% will have distance deviation increase by at least 10Δ in 20 years; only 4% resolved
Pres. = Presentation				

their initial measurements. With a mean initial distance deviation of 23Δ, 65% of patients eventually became exophoric at distance and latency was maintained at near. However, it must be noted that many patients in this series were prescribed orthoptic treatment, which is likely to have contributed to improvement.

Similar findings were reported by Chia, Seenyen, and Quah[8] on 287 patients, though who did not have orthoptic treatment, over a mean period of 3 years. Like the Hiles et al[7] study, the deviation appeared to be relatively stable showing gradual improvements in its size and control at distance, especially in older patients or those who had larger deviations initially.

In another recent study, Clarke and coworkers[10] found that less than one-third of their 168 patients demonstrated deterioration in the control of the deviation, and fewer still (13%) a decrease in stereoacuity (near and distance) over a period of 6 to 24 months. Rutstein and Corliss[2] also reported showing no progression of X(T) and even an improvement of the deviation. However, many of the participants in this study were diagnosed with a basic exotropia or had undergone previous surgery. Furthermore, because improvements could not be attributed to treatment or length of follow-up, Rutstein and Corliss postulated that their findings were due to a regression toward the mean rather than physiological processes. This is a statistical phenomenon such that very

high or low baseline measurements reduce or increase respectively by chance on a subsequent follow-up visit.[12]

However, some studies have suggested that X(T) has an equal chance of becoming better or worse. For instance, Romanchuk, Dotchin and Zurevinsky[9] reported an equal possibility of improvement or deterioration of control of the deviation despite little change in the size of the distance deviation. Despite an improvement in stereoacuity found by Romanchuk et al,[9] it must be noted that without a control group, it is possible that improvement was due to a maturational or learning effect. However, this study is amongst the few to assess the change in stereoacuity between visits. Many authors evaluating various treatments have not considered changes in sensory fusion and have not measured distance and/or near stereoacuity, although its usefulness in assessing control and therefore progression of X(T) has been affirmed.[13]

Contrarily, few studies have reported progression of X(T). Von Noorden & Campos[6] cited a study wherein 75% of patients who were observed without treatment for a mean of 3.5 years displayed one or more signs of progression; the remaining 25% either improved or were unchanged. More recently, Nusz, Mohney and Dieh[15] conducted a study of 138 patients who were followed-up for an average of 5.6 years and found that only 4% resolved in deviation size, more than half having an increased deviation (of at least 10Δ) over a 20 year period.

Most studies published on the natural history of X(T) are retrospective.[2,5,7-9] Retrospective studies rely on the availability and accuracy of medical records and can often be confounded by selection bias may be problematic. The lack of concurrent controls and unreported clinical data also affect such studies' internal and external validities.[14] Previous studies have included various types of X(T)[2,7,8] and/or have included patients who have been undergoing active treatment.[2,5,7,9] The application of treatments in the investigation of the natural course of X(T) can confound conclusions and it is also possible that different types of X(T) have different progression rates.[6] To adjust for potential confounding factors and prevent measurement artifacts, prospective studies with matched case-controls are needed to further understand the natural history of X(T).

NON-SURGICAL TREATMENT

Non-surgical treatment of X(T) is indicated either pre-operatively to optimize sensory conditions or as primary management usually to delay surgery.[6] Such treatment includes the optical correction of refractive error and minus lens treatment, prisms and orthoptics. Table 2 provides a summary of the articles included in this review investigating non-surgical treatment for the management of X(T).

REFRACTIVE CORRECTION & MINUS LENS TREATMENT

It is important to correct refractive errors before administering other forms of treatment as clearing blurred images provides a stimulus for fusion, facilitating control, and particularly for myopes.[4] However, the sole impact of refractive correction on treatment outcome of X(T) remains unknown. For hypermetropes, von Noorden and Campos[6] suggests correcting only hypermetropia >2 dioptres (D), the exact amount of correction being dependent on the patient's age and AC/A ratio. On the other hand, it has long been advocated that minus lenses of the strength required for fusion to be established at distance be added to the refractive correction to stimulate accommodative convergence, thereby improving the control of the X(T).[4] However, opponents of minus lenses suggest that treatment can cause temporary consecutive esotropia and accommodative asthenopia, particularly in older children.[6] In addition, myopic progression has been raised as an issue, but is refuted by studies that have found mean refractive changes similar to the general population.[15,16] Table 2 provides a summary of studies investigating minus lens treatment for the management of X(T).

In a retrospective study, Caltrider and Jampolsky[17] reported either qualitative or quantitative improvement in 72% of children who were over-minused by 2–4D for an average of 35 months. A qualitative improvement was regarded as one of increased control of the X(T) with a well-controlled exophoria; quantitative improvement was defined as a decrease in the exodeviation by at least 15Δ. Improvement was maintained in 70% of patients who were followed for at least a year after cessation of this treatment, demonstrating long-term stability in treatment outcome. Pre-treatment age and AC/A ratio did not seem to affect the outcome though.

Watts, Tippings and Al-Madfai[18] tested the success of minus lens treatment using a standardised scoring system – the Newcastle Control Score (NCS). Similar to Caltrider and Jampolsky[17], the NCS showed that 71% of the patients improved their control of the X(T) post minus lens treatment.

The strength of the minus lenses advocated for this treatment varies.[19-22] Merrick[19] supported the careful use of weak minus lenses in relieving symptoms of X(T). Goodacre[20], on the other hand, recommended stronger lenses of up to -3D, which improved the control of the deviation in 62% of their patients, especially in those with a high AC/A ratio and near deviation ≤24Δ. Further, 72% of patients in a study by Donaldson and Kemp[21] and 62% in a study by Reynolds et al[22] also had comparable success with minus lens treatment using a variation of -1 to - 3D lenses, dependent factors being patient compliance and size of pre-treatment deviation. Hence, to date although minus lens treatment has been shown to be effective, consensus for the strength of the prescription is yet to be established.

Table 2. Summary of studies investigating minus lens treatment for X(T)					
Authors and Study	**Treatment Details**	**N**	**Mean Age**	**Mean Duration of Treatment**	**Outcomes**
Caltrider & Jampolsky[17] Retrospective	Minus lens treatment (-2 to -4DS)	35	Onset: 1.5 years	35 months (2-156 months)	72% improved either fusion quality or both fusion quality and deviation size; 70% followed-up for 1 year maintained their improvement
Watts et al[18] Prospective	Minus lens treatment (-2 to -4DS)	24	6.8 years	4 months	70.8% improved control of deviation
Goodacre[20] Prospective	Group1: Minus lens treatment (Mean -2.50DS) Group2: Minus lens treatment + surgery	34	Group1: 3 years Group2: 4 years	Group1: 32 months Group2: 24 months	62% (of groups 1 &2) became exophoric at all distances;27% had at least 15Δ of reduction in deviation and exophoria
Donaldson & Kemp[21] Retrospective	Minus lens treatment (-2 to -3DS)	27	2-17 years	Approx 6 months or more (67%)	72% wearing lenses for at least 6 months became asymptomatic and recovered BSV
Reynolds et al[22] Retrospective	Minus lens treatment (-1 to -2.50DS)	74	4.8 years	3-6 months	Overall "success" rate: 61.7%; 92% with deviation <20Δ "successful"
Table 3. Summary of studies investigating prism treatment for X(T)					
Pratt-Johnson & Tillson[24] Prospective	Prism (neutralising) treatment	25	2-8 years	1- 2.5 years	66% wearing prisms for at least a year were "cured"
Moore & Stockbridge[26] Retrospective	Prism (overcompensating) treatment or prisms + surgery + orthoptics when needed	50 (Prisms only: 5; Prisms + surgery: 45)	Not stated	Prism therapy alone: 3-18 months; Prisms + surgery: 7 months	Prism therapy: No change in deviation size or control; 13% of patients with residual exodeviation "cured"
Veronneau-Troutman et al[29] Retrospective	Prism treatment or prisms + exercises or prisms + exercises + surgery	37	8-9 years	3.9 months	19% improved fusion quality without surgery; 92% had a decrease in deviation
Table 4. Summary of studies investigating occlusion treatment for X(T)					
Chutter[35] Prospective	Occlusion treatment (38 part-time patching; 8 full-time) + orthoptic exercises when needed	51	2-62 years	3-12 weeks	Fusion strengthened in 70% occluded part-time, 54% of them became exophoric
Spoor & Hiles[32] Prospective	Occlusion treatment (3-6 hrs/day)	38	29 months (7 months-7 years)	15 months (3-42 months)	90% achieved latency for near and 65% for distance; 58% no longer required surgery
Spoor & Hiles[33] 3-year follow-up	--	34	11 years	3 years without occlusion	78% maintained improvement in control and size of deviation
Freeman & Isenberg[34] Prospective	Occlusion treatment (4-6 hrs/day)	11	Onset: 18 Mths;Treatment: 23.5 months	22 months	100% became ortho- or exo-phoric initially; 27% eventually became orthophoric
Iacobucci & Henderson[36] Prospective	Occlusion treatment (constant)	28	--	Up to 3 months	73% (occluded) initially exotropic and 53% initially intermittent at distance became exophoric
Berg & Isenberg[37] Prospective	Occlusion treatment (4-6 hrs/day)	11	Onset: 28 months; Treatment: 4.7 years	9 months	100% achieved latency initially; 36% maintained control of deviation
Table 4. Summary of studies investigating occlusion treatment for X(T)					
Sanfilippo & Clahane[38] Prospective	Orthoptic treatments: occlusion, red-filter antisuppression treatment, convergence exercises etc	31	9 years and above (52%)	5-22 orthoptic sessions Follow-up: 4.5-6.5 years	97% had "excellent" or improved binocular status; 84% of them maintained their status on long-term follow-up
Altizer[39] Prospective	Group1:Orthoptic treatments: occlusion, convergence exercises, prisms Group2: Surgery	52 in total 1) 23 (13 X(T), 10 constant XT) 2) 29	--	1 year; Follow-up: 1-2 years	1) 69% X(T) pts became exophoric from orthoptics; 62% X(T) pts improved convergence 2) 44% exophoric from surgery
Chryssanthou[40] Prospective	Orthoptic treatments: occlusion, red-filter, convergence exercises	27	5-33 years	3-16 sessions; Follow-up: 6-30 months	89% improved binocular status; 67% had"excellent" or "good" status 6-30 months after
Newman & Mazow[42] Retrospective	Group1:Orthoptic treatments: occlusion, minus lenses, exercises, glasses Group2: Surgery	60 in total 1)30 orthoptic 2)30 surgical	Orthoptics: 8 years; Surgery: 6 years	Follow-up: 2 years	Groups 1 & 2: 67% with deviation <30Δ became exophoric and size of deviation reduced to <15Δ

Table 5. Summary of studies investigating combined orthoptic treatment for X(T)

Study and Design	Type/s of Treatment	N	Mean Age	Mean Duration of Treatment	Outcomes
Cooper & Leyman[41] Retrospective	Group1:Occlusion Group2: Surgery Group3: Orthoptic treatment + surgery Group4: Orthoptic treatment (anti-suppression & convergence exercises)	673 in total 1) 11 2) 264 3) 216 4) 182	--	12 weeks Follow-up: 1 year	59% of group 4, 52% of group 3, 42% of group 2 & 36% of group 1 had "good" results
Singh et al[11] Prospective	Orthoptic treatments: occlusion (6hrs/day), bar-reading, convergence and fusional exercises, glasses	30	Presentation: 19.8 years	8 weeks-1 year	64%-86% improved their binocular status and symptoms
Pejic et al[43] Retrospective	Group A: Orthoptic fusion exercises Group B: Control group with no orthoptic treatment	96	6-34 years	Group A: 12-36 weeks	Group A: 74% achieved better distance stereoacuity; 93% increased distance fusional amplitude by at least 50% Group B: No improvement in distance stereoacuity, 12% deteriorated
Moore[44] Retrospective	1)Orthoptic treatment + surgery 2) Surgery 3)Orthoptic treatment	180 in total 1) 106 2) 57 3) 17	3-18 years	Follow-up: 10 months-10 years	1) 73% improved or "cured" 2) 84% improved or "cured" 3) 18% improved or "cured"
Figueira & Hing[31] Retrospective	1) Orthoptic treatment/ occlusion + surgery 2) Surgery 3) Orthoptic treatment/ occlusion 4) Observation	150	Onset: 2.5 years Treatment: 5.2 years	Follow-up: 3.3 years	Highest rate of "success" of orthoptic treatment & surgery at approx 85%

PRISM TREATMENT

Prisms are used to shift target images closer to or on the fovea, aiding in sensory fusion.[23] According to Coffey and coworkers,[4] there are three approaches to prism treatment: prisms can be demand-reducing by compensating for part of the deviation and relieving the load on fusional vergence; neutralising and fully compensating the deviation; or over compensating to increase the convergence response so that fusion is maintained when the prism strength is reduced.

There are few studies on prisms as a primary treatment for X(T) (Table 3). One of the earliest is by Pratt-Johnson and Tillson,[24] who investigated the effects of neutralising prisms on patients with X(T) of less than 20Δ. They had patients wearing them for more than half of their waking hours for 12–30 months. Despite most patients having reduced vision due to the prisms, 66% who wore them for at least 12 months were deemed to be "cured". Hardesty[25] also reported that the younger the child having prism treatment, the higher the chance of improving their fusional amplitudes. However, this study had incorporated orthoptic exercises in conjunction with prisms.

Moore and Stockbridge[26] purported that prism treatment should not be administered alone, but as an adjunct to surgery. In their study, only patients who underwent surgery experienced an improvement in the deviation size and control. Immediate prism treatment was more successful for a number of patients with residual deviation postoperatively.

The success of prism treatment in exodeviations, when used in conjunction with other treatments, have also been reported by other studies.[27-29] In these studies, not only were the size and control of the deviation improved, but convergence amplitudes and retinal correspondence were also enhanced. In a study by Veronneau-Troutman, Shippman and Clahane,[29] 19% of patients receiving pre-operative prism treatment had their fusion improved to the point of no longer requiring surgery. There was also no difference in the results of those receiving prisms alone or both prisms and orthoptic exercises.

However, not all advocate prism treatment. Friendly[30] was skeptical regarding the usefulness of prisms due to the many disadvantages (optical distortion, weight, cosmesis and visual degrading properties), especially for patients unaccustomed to wearing glasses. Possible reliance on the prisms may also develop, causing exodeviations to increase over time.[23]

OCCLUSION TREATMENT

By reducing binocular stimulation, occlusion treatment abolishes the abnormal sensory adaptations developed in avoiding diplopia (suppression) (see Table 4), reduces the suppression scotoma size and reinforces fusional processes after cessation of patching.[31] Studies have demonstrated significant improvements in exodeviation with part-time patching of the non-deviating eye (or alternate patching for those with equal fixation preference) as a passive form of anti-suppression treatment.[32-34] Full-time patching is generally not prescribed because of possible disruption to fusional mechanisms and subsequent manifestation of the deviation.[34] Moreover, part-time patching has been proven to be just as effective as full-time patching in these patients, strengthening fusion in 70%.[35]

Spoor and Hiles[32] prescribed occlusion (3–6 hours daily) over an average of 15 months, aiming to reduce patients' deviation size and increase fusional amplitudes. Control of the deviation improved substantially - the number of patients with latent deviations increased from 26% to 65% for distance fixation. Improvement in the size and control of

the deviation was such that surgery was no longer required in 58% of patients. In a follow-up study, 80% of patients were re-evaluated three years after cessation of occlusion and 78% had maintained their improvements.

In addition to the above findings, occlusion has been reported to delay the need for surgery by at least 2 years, which can greatly reduce the risk of amblyopia following surgical overcorrection in young children.[34] In Freeman and Isenberg's study,[34] all patients who were patched for 4–6 hours daily became heterophoric at least temporarily, with 27% becoming "orthophoric" after a follow-up of 22 months. Their ability to control the deviation was improved more than the reduction in size. Similarly, Iacobucci and Henderson[36] stated that preoperative occlusion enhanced postoperative results by inhibiting the development of suppression, thus increasing postoperative fusional amplitudes. Indeed, 86% of patients demonstrated a decrease in the size of the distance deviation; and 73% of the patients having occlusion who were initially exotropic and 53% who were intermittent at distance eventually became exophoric.

In a similar study to Freeman and Isenberg,[34] Berg and Isenberg[37] also found that similar unilateral occlusion of older children yielded favourable results. All achieved latency or control in the distance for some amount of time with occlusion and 36% maintained their control of the deviation even after cessation of patching.

COMBINED ORTHOPTIC TREATMENTS

Orthoptic exercises (see Table 5) like anti-suppression and convergence exercises aim to make patients aware of when their deviation becomes manifest, reinforcing their control.[6]

Sanfilippo and Clahane[38] examined the immediate and long-term results of combined orthoptic treatments in exodeviations. Red-filter anti-suppression treatment and convergence exercises were prescribed. In most patients, the pre-treatment binocular status was deemed to be "poor" but improved (even to "excellent") in 97% post-treatment. This improvement was maintained in 84% after long-term follow-up.

Altizer[39] compared the effects of non-surgical treatment on X(T) with that of surgery (but including constant exotropia). Non-surgical management consisted of constant occlusion, convergence exercises (that trained relative fusional vergences and simple, jump and voluntary convergence), and base-in Fresnel prisms for approximately 1 year. More X(T) patients attained a "cure" than constant exotropes, especially those undergoing non-surgical treatment (69%) as compared to surgery (44%). "Cure" referred to achieving an exophoria of less than 20Δ at both near and distance that was controlled under stress. Most patients (62%) also improved their convergence ability after non-surgical treatment.

Later studies demonstrated equally favourable outcomes for orthoptic management on X(T), each according to its own definition of success.[11,40-43] Chryssanthou[40] and Newman and Mazow[42] found orthoptics useful even for the treatment of moderate-sized exodeviations (25–30Δ), more than half of patients gaining control both at distance and near fixation. However, Chryssanthou's[40] orthoptic treatment varied from that of Newman and Mazow,[42] the latter including minus lenses in addition to occlusion and convergence exercises. Good results were also obtained in 59% of patients receiving only orthoptic treatment in a study by Cooper and Leyman[41] compared with those receiving just surgery (42%) or both orthoptics and surgery (52%).

More recently, Singh, Roy and Sinha[11] and Pejic and coworkers[43] reported that orthoptic exercises (convergence and fusion exercises) can indeed enhance the binocularity of all types of X(T), particularly convergence-weakness type. More than half of the patients receiving fusion exercises obtained significant improvement in binocularity. In the Pejic et al 43 study, 74% achieved better distance stereoacuity and 93% also demonstrated an increase in their distance fusional amplitudes by at least 50%. In the Singh et al 11 study, orthoptic treatment was found to be effective functionally in 64% and symptomatically in 86% of the patients, most having deviations of ≤25Δ.

While some argue that orthoptic treatment is more effective for certain X(T), others stress that orthoptics as a supplement (and not a substitute) to surgery generates better results. Moore[44] reported the lowest success rate for orthoptic treatment alone on X(T) children when compared to those treated with surgery or a combination of both. Figueira and Hing[31] also demonstrated that combined surgery and orthoptics (including minus lens treatment and convergence exercises) achieved better outcomes than orthoptics alone.

Finally, orthoptic treatment has been criticised for being time-consuming as it requires regular follow-ups, and because patient cooperation and compliance is important for its success.[4] Despite patient compliance being principal in determining the success of various treatments (including minus lenses, prisms and orthoptics), compliance was not evaluated or controlled in any of the above studies. Future studies must actively monitor adherence to treatment. Without any record of compliance, it is difficult to attribute findings to the prescribed treatment.

CONCLUSION

Consensus regarding the efficacies of various non-surgical treatments remains unattainable despite their inherent ability to strengthen fusional control and diminish the size of exodeviation. The lack of uniform success criteria and outcomes as well as inconsistent definitions of treatments and lack of recording of compliance hinder the evaluation and comparison of treatment options to determine best management.

Furthermore, sub-categorisation of X(T) types are often not clearly defined even though they are likely to have a different response to treatment. Prospective randomised controlled trials with larger sample sizes and standardised definitions and scoring systems are required in order to better assess the effectiveness of various treatments. A randomised controlled trial is considered the best type of study for the assessment of healthcare interventions. By conducting such studies, clinical guidelines for the use of non-surgical treatment in X(T) could be developed more readily. Finally, further investigations on the unpredictable course of X(T) are also important as it has implications on the true value of non-surgical management, and the need for and timing of surgery.

REFERENCES

1. Jenkins R. Demographics geographic variations in the prevalence and management of exotropia. Am Orthopt J 1992;42:82-87.
2. Rutstein RP, Corliss DA. The clinical course of intermittent exotropia. Optom Vis Sci 2003;80:644-649.
3. Burian HM, Spivey BE. The surgical management of exodeviations. Am J Ophthalmol 1965;59:603-620.
4. Coffey B, Wick B, Cotter S, Scharre J, Horner D. Treatment options in intermittent exotropia: a critical appraisal. Optom Vis Sci 1992;69:386-404.
5. Nusz KJ, Mohney BG, Diehl NN. The course of intermittent exotropia in a population-based cohort. Ophthalmology 2006;113:1154-1158.
6. von Noorden GK, Campos EC. Binocular Vision and Ocular Motility: Theory and Management of Strabismus. St Louis, USA: Mosby-Year Book Inc, 2002.
7. Hiles DA, Davies GT, Costenbader FD. Long-Term observations on un-operated intermittent eotropia. Arch Ophthalmol 1968;80:436-442.
8. Chia A, Seenyen L, Quah BL. A retrospective review of 287 consecutive children in Singapore presenting with intermittent exotropia. J AAPOS 2005;9:257-263.
9. Romanchuk KG, Dotchin SA, Zurevinsky J. The natural history of surgically untreated intermittent exotropia: looking into the distant future. J AAPOS 2006;10:225-231.
10. Clarke M, Richardson S, Haggerty H, et al. Intermittent exotropia is not always a progressive condition. In: Verlohr D, Georgievski Z, Rydberg A, editors. Transactions of the Xth International Orthoptic Congress. Melbourne, Australia, 14-17 November, 2004; 408-411.
11. Singh V, Roy S, Sinha S. Role of orthoptic treatment in the management of intermittent exotropia. Indian J Ophthalmol 1992;40:83-85.
12. Streiner D. Regression toward the mean: its etiology, diagnosis, and treatment. Can J Psychiatry 2001;46:72–76.
13. Stathacopoulos RA, Rosenbaum AL, Zanoni D, et al. Distance stereoacuity: assessing control in intermittent exotropia. Ophthalmology 1993;100:495-500.
14. Hess DR. Retrospective studies and chart reviews. Respir Care 2004;49:1171-1174.
15. Rutstein RP, Marsh-Tootle W, London R. Changes in refractive error for exotropes treated with overminus lenses. Optom Vis Sci 1989;66:487-491.
16. Kushner BJ. Does overcorrecting minus lens therapy for intermittent exotropia cause myopia? Arch Ophthalmol 1999;117:638-642.
17. Caltrider N & Jampolsky A. Overcorrecting minus lens therapy for treatment of intermittent exotropia. Ophthalmology 1983;90:1160-1165.
18. Watts P, Tippings E, Al-Madfai H. Intermittent exotropia, overcorrecting minus lenses, and the Newcastle Scoring System. J AAPOS 2005;9:460-464.
19. Merrick F. Use of Concave Lenses in the management of intermittent divergent squint. Aust Orthopt J 1975;12:13-17.
20. Goodacre H. Minus overcorrection: conservative treatment of intermittent exotropia in the young child: a comparative study. Aust Orthopt J 1985;22:9-17.
21. Donaldson PJ, Kemp EG. An initial study of the treatment of intermittent exotropia by minus overcorrection. Br Orthopt J 1991;48:41-43.
22. Reynolds JD, Wackerhagen M, Olitsky SE. Overminus lens therapy for intermittent exotropia. Am Orthopt J 1994;44:86-91.
23. Rutstein RP, Cogen MS, Cotter SA, et al. Optometric Clinical Practice Guideline Care of the Patient with Strabismus: Esotropia and Exotropia, 1999.
24. Pratt-Johnson JA, Tillson G. Prismotherapy in intermittent exotropia: a preliminary report. Can J Ophthalmol 1979;14:243-245.
25. Hardesty HH. Prisms in the management of intermittent exotropia. Am Orthopt J 1972;22:22-30.
26. Moore S, Stockbridge L. An evaluation of the use of fresnel press-on prisms in childhood strabismus. Am Orthopt J 1975;25:62-66.
27. Berard PV. Prisms: Their Therapeutic Use in Concomitant Strabismus with Normal Correspondence. St Louis: C.V. Mosby Co, 1968.
28. Ravault AP, Bongrand M, Bonamour G. The Utilisation of Prisms in the Treatment of Divergent Strabismus. Amsterdam: Excerpta Medica, 1972.
29. Veronneau-Troutman S, Shippman S, Clahane AC. Prisms as an Orthoptic Tool in the Management of Primary Exotropia. New York, U.S.A: Stratton Intercontinental Medical Book Co, 1976.
30. Friendly DS. Surgical and non-surgical management of intermittent exotropia. Ophthalmol Clin North Am 1992;5:23-30.
31. Figueira EC, Hing S. Intermittent exotropia: comparison of treatments. Clin Exp Ophthalmol 2006;34:245-251.
32. Spoor DK, Hiles DA. Occlusion therapy for exodeviations occurring in infants and young children. Ophthalmology 1979;86:2152-2157.
33. Spoor DK, Hiles DA. Occlusion therapy for exodeviations in young children: a three year follow-up. Am Orthopt J 1983;33:116-120.
34. Freeman RS, Isenberg SJ. The use of part-time occlusion for early onset unilateral exotropia. J Paediatr Ophthalmol Strabismus 1989;26:94-96.
35. Chutter CP. Occlusion treatment of intermittent divergent strabismus. Am Orthopt J 1977;27:80-84.
36. Iacobucci I, Henderson JW. Occlusion in the pre-operative treatment of exodeviations. Am Orthopt J 1965;15:42-47.
37. Berg PH, Isenberg SJ. Treatment of unilateral exotropia by part-time occlusion. Am Orthopt J 1991;41:72-76.
38. Sanfilippo S, Clahane AC. The effectiveness of orthoptics alone in selected cases of exodeviation: the immediate results and several years later. Am Orthopt J 1970;20:104-117.
39. Altizer LB. The non-surgical treatment of exotropia. Am Orthopt J 1972;22:71-76.
40. Chryssanthou G. Orthoptic management of intermittent exotropia. Am Orthopt J 1974;24:69-72.
41. Cooper EL, Leyman IA. The management of intermittent exotropia: a comparison of the results of surgical and non-surgical treatment. New York, U.S.A: Stratton Intercontinental Medical Book Co, 1976.
42. Newman J, Mazow ML. Intermittent exotropia: Is surgery necessary? Ophthalmic Surgery 1981;12:199-202.
43. Pejic Z, Wong W, Husain R, Ling Y, Farzavandi S. Fusion exercises for treatment of intermittent exotropia and phoria. Am Orthopt J 2006;56:138-146.
44. Moore S. Orthoptic treatment for intermittent exotropia. Am Orthopt J 1963;13:14-20.

Selected Abstracts from the OAA 62nd Annual Scientific Conference, held in Hobart, 6 – 9 November 2005

THE ACCURACY OF CONVERGENCE WHEN THE NEAR TARGET IS NOT ON THE MIDLINE

Assoc Prof Elaine Cornell,
School of Applied Vision Sciences, University of Sydney

Introduction: Although orthoptists usually assess convergence by moving a target closer to the eyes along the midline (similar to 'pursuit' movement), in everyday life we normally make quick changes of fixation to a near target, sometimes on the midline but more often it is displaced to one side. This requires asymmetrical convergence. The dynamics of these movements are now well known, and they are usually brought about by the highly integrated action of conjugate and disjugate mechanisms, both of which are expressed preferentially in fast, saccadic movements. In an asymmetrical situation, where one eye needs to converge more than the other, the accuracy of this movement may affected if the eye that normally fails on convergence is the one that needs to make the larger movement. The purpose of this study was to evaluate: whether the accuracy of ocular alignment following a convergence movement is affected by whether the movement is symmetrical (near object on the midline) or asymmetrical (near object displaced to one side), and whether the accuracy of asymmetrical convergence is related to the eye that normally 'fails' when testing the convergence near point.

Method: The accuracy of binocular fixation was assessed in twenty two subjects after converging 10o symmetrically (5o each eye) and asymmetrically to the right and left (2.5o one eye, 7.5o the other eye, or 10o with one eye only). For each subject ocular dominance was assessed by a sighting test and also by noting the eye that failed at the convergence near point.

Results: All subjects, except one, maintained fixation with the right eye at the convergence near point, that is, the left eye 'failed' in almost every case. Eighteen subjects had right eye dominance and four were left eye dominant as assessed by the sighting test. In over half of the subjects (13/22) the accuracy of binocular alignment decreased as the position of the near target moved to one side. In eight subjects errors increased as the target was shifted to the left, in the other five the errors increased as the target was shifted to the right. In most of these cases, the errors were made by the eye that was making the larger adducting movement – this was more frequently the right eye.

Conclusions: Binocular fixations for near are more likely to be imprecise following asymmetrical vergence than those following symmetrical vergence. The findings from this study suggest that it is the adducting eye that tends to be less precise. This was most commonly the right eye, resulting is more errors on left gaze. Although there was a strong association between the right eye as the dominant eye and the eye that was less precise, it is not readily apparent how this would form a causal relationship. These findings are therefore not directly related to ocular dominance.

THE EFFECTS OF INCREASED VISUAL TASK DEMAND ON FOVEATION IN CONGENITAL NYSTAGMUS

Dr Linda Tkalcevic[1], **Dr Larry Abel**[2]
[1] Department of Clinical Vision Sciences, La Trobe University
[2] Department of Optometry and Vision Sciences, University of Melbourne

Purpose: Commonly, when an individual with congenital nystagmus (CN) performs a visually demanding task their nystagmus intensifies and their visual acuity decreases, probably due to poorer foveation. However, the relationship between fixation attempt and nystagmus waveform has never been quantified. This study attempted to determine the relationship between visual demand and mental state of CN subjects on their foveation time during reading of varying sized optotypes at distance fixation.

Methods: In this study, 14 CN subjects (7 classified as idiopathic & 7 as albino) viewed a Landolt C of varying orientation and size. They indicated its orientation via a push button array whilst eye movements were recorded via a binocular infrared oculographic system.

Results: Eye movement data were analysed for changes in duration of foveation periods (defined as those periods of the eye movement recording during which eye velocity was less than or equal to 4 deg/sec and eye position ± 2 deg from the point of fixation from cycle to cycle), and whether the CN waveform itself changed during times of increased visual demand. Foveation was uncorrelated with optotype size.

Conclusion: The results suggest that CN is not exacerbated by visual demand per se rather the need to do something visually demanding of importance to the individual. Explicit manipulation of anxiety, arousal and motivation during recordings of CN should aid in broadening our understanding of the behaviour of this disorder.

ALCOHOL EFFECTS ON THE SACCADIC EYE MOVEMENT SYSTEM

Dr Suzane Vassallo[1], **Dr Larry Abel**[2]
[1] Department of Clinical Vision Sciences, La Trobe University
[2] Department of Optometry and Vision Sciences, University of Melbourne

Alcohol, a central nervous system (CNS) depressant, was the first drug to be examined for its effect upon the saccadic eye movement system. Over time, the literature has shifted to the analysis of higher-order saccadic tasks under alcohol – e.g., self-paced and antisaccade tasks. This laboratory investigated a battery of saccadic eye movement paradigms under two doses of alcohol in healthy males. Low to moderate doses of alcohol (0.045 to 0.071% BAC) impaired reflexive not volitional saccade control. Error rates arising from the antisaccade and memory-guided saccade paradigms did not increase under alcohol. These latter findings are ill-explained in the context of the disinhibition theory used to describe alcohol's effect on behaviour. Also, it was discovered that the saccade latency distribution shifted under alcohol, causing a reduction of saccade frequency in the express saccade mode. The latency distribution has been hitherto unexplored under alcohol. These results suggest that low to moderate doses of alcohol appear to especially exert their effect upon posterior cortical pathways controlling reflexive saccades. Frontal areas mediating volitional control, including response suppression, appear unaffected by the drug at this concentration. It is interesting to note that there are some disorders (e.g., ADHD, Tourette's Syndrome, OCD) which are associated with failures of saccadic inhibition and disinhibited social behaviour. This does not seem to be the case under alcohol at low to moderate doses. This work has highlighted that different forms of behavioural inhibition exist and that, in some instances but not others, they may co vary.

EYE MOVEMENT CONTROL DURING THE VISUAL SCANNING OF OBJECTS

Lisa Jones, Dr Suzanne Vassallo
Department of Clinical Vision Sciences, La Trobe University

This study examined the scanpaths of young healthy individuals while they viewed half objects (e.g., half a car which had been dissected vertically). Eye

movements provide a remarkable guide to where attention is deployed in the visual environment. The visual scanpath is a sequence of eye movements (saccades) and fixations that not only depend upon visual features that capture attention, but also cognitive components such as planning, experience and memory. Empty regions of space are generally not fixated, yet new research into mental imagery has found that viewers sometimes do look towards empty, uninformative regions of a scene if it previously contained an interesting object or if viewers were imagining an object or scene. Recent research in the Department of Clinical Vision Sciences (Dyer, Vassallo, Abel, unpublished data) found that while healthy subjects viewed images of half faces, they made eye movements to the missing half of the face 64% of the time. This adds to evidence suggesting that there are specialised mechanisms within the brain for processing faces. Thus raising the question: what happens when viewers are presented with half objects? The current study has demonstrated that the types of eye movements used for face versus object viewing are different - people generally do not exhibit learned eye movement behaviour for viewing objects though seem to when they look at faces. Taken together, the hypothesis that there is a possible pre-wired way of looking at faces appears supported. Differences in the nature of the scanpaths will be discussed.

PATRICIA LANCE MEMORIAL LECTURE – THE SYDNEY MYOPIA STUDY: IMPLICATIONS FOR EVIDENCE-BASED PRACTICE AND PUBLIC HEALTH

Dr Kathryn Rose,
School of Applied Vision Sciences, University of Sydney

The Sydney Myopia Study is a study of refractive error and other ocular conditions in a large representative sample of Australian school-aged children in Sydney. Children attending Year 1 (aged 6) and Year 7 (aged 12) were recruited from 34 primary schools and 21 high schools that were selected using a stratified random cluster design based on socio-economic status. The study incorporates significant sub-groups of children of East Asian, Middle Eastern and South Asian origin. Children receive a full and comprehensive eye examination in their school including visual acuity, cover test, colour vision, slit lamp, ocular biometry, cycloplegic refraction, ocular computerized tomography and retinal photography. Parents of the Year 1 students completed a 193 item questionnaire on health, socio-demographic information and life-style, while the Year 7 students and their parents completed separate questionnaires addressing the same items. The examination of the 1740 children in Year 1 cohort is complete and preliminary data from the anticipated 2,300 Year 7 cohort is available. The overall participation rate thus far is 78%. A study of this design is able to address many issues relevant to the ocular health of Australian school children including the prevalence of refractive error and other ocular conditions, access to vision screening and the need for services, adequate and appropriate prescription of glasses, as well as factors that may associated with eye disease in children. This study is the first of its kind in Australia to addresses these issues in a population–based sample of school children.

THE IMPORTANCE OF MEASURING RADIUS OF CORNEAL CURVATURE WHEN USING THE HEIDELBERG RETINAL TOMOGRAPH

Anne Klawir

Introduction: In September 2004, we received correspondence from Heidelberg Engineering indicating that the 'absolute scaling of the HRT2 images is dependent on the correct measurement of the radius of corneal curvature'. If the default value of 7.7mm is not changed, measurement results will not be accurate.

Method: Over the last year, we have measured just over 200 eyes using the IOL master in the keratometry mode. Using the average between K1 and K2 results, we entered the correct radius of corneal curvature for each patient, replotted contour line segments and reprinted old results.

Results: Our average radius of corneal curvature results for these 100 patients was 7.66mm. However, there were many outliers with the distribution ranging from 7.06mm to 8.34mm. Different stereometric analysis of ONH values were revealed when radius of corneal curvature values were changed. The chart of 'possible amount of error' from Heidelberg Engineering shows that 'amount of error is dependant on the amount of deviation' from 7.7mm. For example, one patient aged 59 had radius of corneal curvature values of R 8.05mm and L 8.00mm. According to the chart, her error is -2.4% distance, -4.9% area and -9.9% volume. If only Moorfields regression analysis and change probability map analysis techniques will be used, the value of 7.7mm can be left.

Conclusion: To ensure accuracy and to take advantage of all analysis software of the HRT2, it is highly recommended that you measure and change all radius of corneal curvature measurements.

RETAANE VERSUS VISUDYNE PHOTODYNAMIC THERAPY IN WET AGE-RELATED MACULA DEGENERATION

Nathan Clunas,
Marsden Eye Research, Marsden Eye Specialists

Retaane (anecortave acetate) 15mg (n=255) was compared to Visudyne PDT (n=256) in a multi-centre, randomised, double-masked, active-controlled, parallel group 12 month study, with 12 months follow-up, in patients with predominantly classic wet AMD (Study C-01-99).

Patients received Visudyne PDT every 3 months if leakage was present, or Retaane every 6 months by posterior juxtascleral injection, plus the appropriate placebo. At 12 months, 45% of Retaane patients were within 15 letters of their baseline visual acuity, compared to 49% of PDT patients (p=ns). Efficacy in both groups was lower than that seen in other similar studies, most likely because the lesions were much smaller, younger and more aggressive.

Further analysis of prospectively collected data showed that 50% of Retaane patients did not receive the full dose of drug at 6 months due to reflux, and 30% did not receive the 6 month dose on time, adversely affecting the results. Several steps have been taken to improve the reliability of the injection procedure. The procedure is simple but requires attention to detail. There were no cases of globe penetration, endophthalmitis, serious side effects or significant systemic effects seen with Retaane.

Retaane is effective for the treatment of predominantly classic wet AMD, utilising a safe procedure with significant convenience benefits due to the 6-monthly dose regimen. The role of the clinical trial coordinator in this study will also be explained in this presentation.

VISUAL HALLUCINATIONS – CHARLES BONNET SYNDROME

Lynn Dalmazzo,
Vision Australia

Patients with a severe and sudden vision loss can experience visual disturbances known as Charles Bonnet syndrome. Recognition of its possible existence is important. Primary consideration should be the management of the client's anxiety and their lack of understanding of what they are experiencing. Visual hallucinations related to vision loss go largely unreported. Routine enquiry about them is advised. It is for this reason that the question is included in a questionnaire completed by all clients before attending a low vision clinic. The significance of diagnosis and explanation of the hallucinations is important for the client's well being. Although there is no treatment, there are strategies that may lessen the frequency of the occurrence of visual hallucinations.

SOCIAL ISSUES EXPERIENCED BY 16-25 YEAR OLDS WITH VISION IMPAIRMENT

Cathryn Galtry, Assoc Prof Kerry Fitzmaurice,
Department of Clinical Vision Sciences, La Trobe University

Aims: To identify some of the social issues experienced by young adults with vision impairment; to compare the social issues reported by this group of young Australian adults with those reported in the literature.

Method: Four young adults between the ages of 16 – 25 who have vision impairment were recruited. Participants were engaged in semi structured interviews. The interviews were recorded, transcribed and analysed for themes.

Results: Data is not yet fully analysed but to date a number of themes have emerged. (1) Mobility: participants reported a preference for sighted guide rather than the use of a cane or guide dog, however they acknowledged this increased their dependence. (2) Relationships: participants reported enjoying passive activities such as going out for dinner or going to movies. This was an interesting report as both of these participants were severely vision impaired however both indicated that the interaction with company was the important feature not the activity. (3) Personal care and household tasks: both participants reported a large degree of dependence in these areas and reported being frightened to undertake activities such as cooking. (4) Education: neither participant felt schooling had been a problem but both indicated they had relied heavily on siblings when adjusting to a new school environment.

LIGHTING FOR LOW VISION: BEYOND TASK LAMPS

Luisa Ferronato, Lynn Dalmazzo,
Vision Australia

In the field of low vision and vision rehabilitation, the importance of lighting or illumination is frequently highlighted as a strategy to enhance not only vision for near tasks such as reading and writing, but also to enhance independence and safety when moving around an environment. Both quantity and quality of lighting need to be considered when formulating recommendations for people with vision impairment.

This paper will present a review of the literature and draw on the experiences of the authors to discuss the general principles surrounding people with vision impairment and their ambient lighting needs. The advantages and disadvantages of different types of light sources will be outlined as well as strategies to improve lighting quality through facilitating uniformity, glare control and use of contrast.

A MULTIDISCIPLINARY APPROACH TO ECCENTRIC VIEWING TRAINING: A CASE STUDY

Assoc Prof Kerry Fitzmaurice, Lee Clark,
Department of Clinical Vision Sciences, La Trobe University

Eccentric viewing is a strategy that has been shown to be effective in ameliorating the impact of vision impairment as a result of macular disease in adults. However clinically it is assumed children automatically learn to use this technique without assistance. In addition orthoptists are often unable to provide eccentric viewing training as it is seen as time consuming and therefore not cost efficient.

This case study reports our experience of two brothers aged 10 and 12 years with severe reduced vision and bi-lateral centre field loss. Best eccentric viewing position was established and a basic training program designed by the orthoptist. The visiting teacher provided eccentric viewing training using EccVue software as part of the normal support service. Both boys showed marked improvement in visual function and the program was considered a very positive experience by the boys, their mother and the visiting teacher. The impact of the program on the boys schooling, leisure and home activities was documented in a follow up interview. An outline of the program and the outcomes will be presented.

"WHAT'S WITH THE DOTS?" WHEN IS IT APPROPRIATE TO LEARN BRAILLE?

Marion Rivers,
Vision Australia

A diagnosis of severe vision impairment or blindness in early childhood is devastating news to most parents. Initial fear and dread for their child's future soon becomes focused on the practicalities of life without sufficient vision for normal learning and reading. Learning Braille is so foreign to most parents in this position that they find the concept difficult to understand. Professionals working with the families outside of blindness agencies are often in the same position as parents, with little understanding of the practicalities of Braille versus the newer alternative or supplementary technologies. This paper looks at the issues surrounding the introduction of Braille and the orthoptist's role in the decision making process.

ECCENTRIC FIXATION REVISITED – A CASE STUDY

Chen Jie[1], **Neryla Jolly**[2]
[1] Eye Hospital of Wenzhou Medical College (China)
[2] School of Applied Vision Sciences, University of Sydney

A review of the literature about the treatment of eccentric fixation was unable to disclose any recent information about strategies to change the pattern. This case study reports a patient in China, aged 11 years with a left esotropia who was unable to take up left fixation, had gross left amblyopia and retinal fixation near the disc. He had had no previous treatment and the parents were very concerned about the vision and wished to undertake treatment.

This case presents an interesting combination of a condition that is now rarely seen at such a marked level, which presented at an age that is less likely to respond to treatment. The treatment is described and the surprising out come discussed.

VISION SCREENING IN PRESCHOOL CHILDREN USING THE MELBOURNE ACUITY SCREENING TEST (MAST)

Sarita Ibbotson, Sue Silveira,
School of Applied Vision Sciences, University of Sydney

Routine vision screening of children within their first year of school has recently been abolished in NSW. Our focus for vision screening must now reside on establishing a vision screening program for the younger preschool population. Visual acuity tests currently used for vision screening of 5-6 year old children are not appropriate for use with preliterate preschool children. A simpler means of assessing visual acuity in preschool aged children is needed.

The Melbourne Acuity Screening Test (MAST) is a modified visual acuity test designed to provide a quick and simple method of vision screening in children. It incorporates a linear presentation with a simple pass/fail test method and does not allow for measurement of threshold visual acuity. The MAST is based on the principles of the Sheridan Gardiner seven letter chart, and consists of two pages for testing at 6 meters, along with a practice page containing larger optotypes. The size of the letters of the MAST is equivalent to the 6/9 line on the standard Sheridan Gardiner vision chart. The simplicity of the MAST makes it an ideal test for use by non-vision professionals such as nurses.

The ongoing research involving 140 subjects aged between 5 and 7 years comparing the MAST and the LogMAR charts, and 137 preschool children aged between 3-5 years old comparing the MAST and the Sheridan-Gardiner linear chart will be presented.

AMBLYOPIA AND READING DIFFICULTIES

Kate Brassington[1], **Connie Koklanis**[1,2], **Lesley Bretherton**[3]
[1] Department of Clinical Vision Sciences, La Trobe University
[2] Department of Ophthalmology, Royal Children's Hospital
[3] Department of Psychology, Royal Children's Hospital

Reading is an important part of learning and education. For this reason there has been extensive research into investigating the causes of reading difficulties. Most of the vision research completed in the last 20 years has focused on several areas such as visual acuity, refractive error, accommodation and binocular functions. However, the literature has overlooked specific vision disorders as a source of reading problems. In particular the impact of amblyopia on reading ability has been neglected. In order to address this issue, we have conducted a pilot study investigating the relationship reading disability in children with amblyopia. The findings of this study will be discussed.

PAEDIATRIC CATARACT MANAGEMENT: THE SCIENCE UNDERLYING CLINICAL PRACTICE

Prof Frank Billson AO,
Sydney Eye Hospital, and Sydney Children's Hospital,
and Save Sight Institute Australia, University of Sydney

Paediatric cataract is the commonest cause of surgically treatable blindness in early infancy. Its outcomes in paediatric cataract surgery owe much to the increased understanding of neurophysiology and neurobiology of lens and ocular development and their place in the maturing visual system. There is general agreement on the principles of cataract surgery in early childhood and the place of intraocular lens implantation in the paediatric age group particularly after the age of two. Where opinions are divided is about intraocular lens implantation in the first two years of life, with increasing divergence of opinion and controversy about lens implantation in early infancy and particularly at birth.

Management of cataract in children is different from the adult, because of increased intraoperative difficulties, propensity of postoperative inflammation, changing refractive state of the eye, difficulty in documenting anatomic and refractive changes due to poor compliance, and a tendency to develop amblyopia. Adoption of different techniques for cataract surgery is mostly due to a low scleral rigidity, increased elasticity of the anterior capsule, and high vitreous pressure.

This paper discusses controversies surrounding these issues and the science that underpins them, including case selection, timing of surgery, surgical technique and optical correction. It presents the contribution of basic science to improving outcomes of surgery. It refers particularly to the management of the post-operative complications including posterior capsular opacification, aphakic and pseudophakic glaucoma, post-operative inflammation and quality of paediatric anaesthesia.

Reference is made to the contrast between developed and developing countries in terms of available human and material resources and late presentation where expectations of communities are less. Such a divergence of resources and community awareness may occur in the one country, particularly where there are remote and rural areas.

ULTRASOUND BIOMICROSCOPY IN STRABISMUS MANAGEMENT

Dr Shuan Dai, Dr David R. Smith, Dr Raymond Buncic,
Prof Stephen P. Kraft,
Department of Ophthalmology and Vision Sciences, University of Toronto,
and The Hospital for Sick Children, and Toronto Western Hospital

Ultrasound biomicroscopy (UBM) utilizes high frequency (50 MHz) ultrasound waves that depict the anterior segment structures of the eye in high resolution, a goal not possible with conventional (10 MHz) ultrasound used for orbit and intraocular lens work. The UBM has proven to be very helpful in assessing various forms of glaucoma as well as in following lesions in the cornea and iris.

We previously reported the results of UBM in evaluating the insertions of 79 horizontal rectus muscles in patients undergoing primary strabismus repairs (Watts et al, J of AAPOS, 2002). At the time of surgery the UBM was used to localize the muscle positions. During surgery the actual muscle positions were measured with calipers by the surgeon who was masked to the results found with the UBM. We found a good correlation between the surgical and UBM measurements.

In a subsequent study we assessed the efficacy of UBM in determining the positions of horizontal rectus muscles that had previously undergone surgery. We performed UBM determinations of eye muscle positions in 43 muscles either at the preoperative visit or at surgery prior to the conjunctival incision. These were compared to the measurements by the surgeon at the time of surgery. We found a "very good" correlation between the measures found by the two methods. The UBM could detect the medial rectus muscle if it was within 12 mm. from the limbus, and the lateral rectus if it was no further than 15 mm. from the limbus.

The results suggest that UBM can be a reliable indicator of the position of the horizontal rectus muscles in patients undergoing reoperations, as long as the muscles are located within the ranges that are physically reachable with this technology. It is a short, easily performed study that can obviate the need for more expensive or time-consuming tests such

PROGRESS IN VISION SURVEILLANCE AND SCREENING IN NSW: THE FIGHT GOES ON

Sue Silveira,
School of Applied Vision Sciences, University of Sydney

In 2000 the NSW Department of Health introduced the Families First Initiative which changed the direction of child health surveillance and screening. This change saw a cessation of the traditional vision screening of children in their first year of school. Although the value of continued vision screening was recognised, there was no formal framework suggested. Concern was raised by orthoptists and ophthalmologists as to how the high numbers of children would continue to be vision screened at this early age.

During 2004 and 2005 a group of orthoptists, ophthalmologists and community nurses have met to develop the Early Childhood Health Plan which addresses both vision surveillance of babies and vision screening of 4-5 year old children, prior to school entry. The Plan addresses four key result areas, these being a service delivery model, professional development for nurses involved in vision surveillance and screening, tests and equipment and health promotion.

A brief overview of the Early Childhood Health Plan will be presented, with particular attention paid to the planned vision surveillance and vision screening protocols. Plans for research and implementation will also be discussed.

YOKOYAMA PROCEDURE FOR ESOTROPIA WITH HIGH MYOPIA

Robyn Wallace,
Orthoptic department, Royal Victorian Eye and Ear Hospital

Increasing interest in anatomical abnormalities as the aetiology for strabismus has led to increases numbers of patients with squint undergoing imaging of muscles as an investigation prior to the decision of the surgical procedure.

One well documented group of patients where this applies and surgical techniques have been designed to address the underlying problem is that group with axial myopia.

Yokoyama in 1999 reported this his procedure where the Medial Rectus of

the esotropic eye is recessed while the Lateral Rectus and Superior Rectus of the same eye is split and the edges joined together.

This procedure reduces strabismus which if often very marked and increases the lateral visual field. RVEEH Ocular motility clinic has undertaken several of these procedures with some very pleasing results.

COMPARISON OF THE GOLDMANN PERIMETER AND ESTERMAN WHEN VISUAL FIELDS ARE ASSESSED FOR DRIVING

Sue Silveira, Neryla Jolly, Dr Robert Heard, Karen Pepper,
School of Applied Vision Sciences, University of Sydney

Visual standards for driving in Australia are currently determined by two bodies – Austroads and the National Transport Commission. A document titled "Assessing Fitness to Drive for Commercial and Private Vehicle Drivers" is released to practitioners, which outlines medical standards for licensing and clinical management guidelines.

Visual fields are considered in assessment of a person's fitness to drive. Visual fields can initially be assessed by confrontation. If the practitioner suspects visual field loss, then automated static perimetry is performed, using a Humphrey Field Analyser, Medmont M700, Octopus etc. If automated perimetry suggested that the criteria for an unconditional licence won't be met then Goldmann Perimetry or Esterman Perimetry should be performed.

Traditionally the accepted target on the Goldmann Perimeter has been the IV4e. The target on the Esterman however represents a smaller sized target, and is the equivalent to the III4e on the Goldmann Perimeter. Research was conducted to compare the visual fields plotted using the IV4e on the Goldmann Perimeter and the smaller Esterman target, over a population of 150 people, ranging from 18-85 years of age.

Orthoptists are routinely involved in assessing the visual standards and onroad performances of people attempting to gain or regain their licence. Understandably clinical work in this area is often complex for the Orthoptist, who may be responsible for presenting the most accurate clinical findings on a person's visual status. Variation in results on visual field assessment presents a dilemma. The findings of this ongoing research which examines the variation in visual field results according to the Perimeter and target used will be presented.

AMBLYOPIA MANAGEMENT – TRENDS FOR THE 21ST CENTURY?

Dr Connie Koklanis, Zoran Georgievski,
Department of Clinical Vision Sciences, La Trobe University

Occlusion therapy and atropine penalization are the most commonly prescribed treatment modalities for the management of amblyopia. However, recently amblyopia treatment has been criticised for not being sufficiently 'evidence-based'. In particular, there has been criticism of the lack of rigorous research investigating the effectiveness of these treatment modalities. To address this issue the North American Pediatric Eye Investigator Group (PEDIG) and several other researchers have conducted various randomized controlled trials (RCTs) to evaluate the effectiveness of amblyopia treatment. This 'interactive forum' will outline these developments and involve discussion of cases in the context of this current literature and the Australian eye health care system. A panel of experts will be asked to present their views on each case and active participation from the audience will be encouraged.

Named Lectures, Prizes and Awards of the Orthoptic Association of Australia Inc.

THE PATRICIA LANCE LECTURE

1988	Elaine Cornell	(Inaugral)
1989	Alison Pitt	Accommodation deficits in a group of young offenders
1990	Anne Fitzgerald	Five years of tinted lenses for reading disability
1992	Carolyn Calcutt	Untreated early onset esotropia in the visual adult
1993	Judy Seaber	The next fifty years in orthoptics and ocular motility
1995	David Mackey	
1997	Robin Wilkinson	Heredity and Strabismus
1998	Kerry Fitzmaurice	Research: A journey of innovation or rediscovery
1999	Pierre Elmurr	
2005	Kathryn Rose	The Sydney Myopia Study: implications for evidence based practice and public health
2006	Frank Martin	

THE EMMIE RUSSELL PRIZE

1957	Margaret Kirkland	Aspects of vertical deviation
1959	Marion Carroll	Monocular stimulation in the treatment of amblyopia exanosia
1960	Ann Macfarlane	A study of patients at the Children's Hospital
1961	Ann Macfarlane	A Case history "V' Syndrome
1962	Adrienne Rona	A survey of patients at the Far West Children's Health Scheme, Manly
1963	Madeleine McNess	Case history: right convergence strabismus
1965	Margaret Doyle	Diagnostic pleoptic methods and problems encountered
1966	Gwen Wood	Miotics in practice
1967	Sandra Hudson Shaw	Orthoptics in Genoa
1968	Leslie Stock	Divergent squints with abnormal retinal correspondence
1969	Sandra Kelly	The prognosis in the treatment of eccentric fixation
1970	Barbara Denison	A summary of pleoptic treatment and results
1971	Elaine Cornell	Paradoxical innervation
1972	Neryla Jolly	Reading difficulties
1973	Shayne Brown	Uses of fresnel prisms
1974	Francis Merrick	The use of concave lenses in the management of intermittent divergent squint
1975	Vicki Elliott	Orthoptics and cerebral palsy
1976	Shayne Brown	The challenge of the present
1977	Melinda Binovec	Orthoptic management of the cerebral palsied child
1978	Anne Pettigrew	
1979	Susan Coil	Nystagmus blocking syndrome
1980	Sandra Tait	Foveal abnormalities in ametropic amblyopia
1981	Anne Fitzgerald	Assessment of visual field anomalies using the visually evoked response.
1982	Anne Fitzgerald	Evidence of abnormal optic nerve fibre projection in patients with Dissociated Vertical Deviation: A preliminary report
1983	Cathie Searle	Acquired Brown's syndrome: A case report
	Susan Horne	Acquired Brown's syndrome: A case report
1984	Helen Goodacre	Minus overcorrection: Conservative treatment of intermittent exotropia in the young child
1985	Cathie Searle	The newborn follow up clinic: A preliminary report of ocular anomalies
1988	Katrina Bourne	Current concepts in restrictive eye movements: Duane's retraction syndrome and Brown's syndrome
1989	Lee Adams	An update in genetics for the orthoptist: a brief review of gene mapping
1990	Michelle Galaher	Dynamic Visual Acuity versus Static Visual Acuity: compensatory effect of the VOR
1991	Robert Sparkes	Retinal photographic grading: the orthoptic picture
1992	Rosa Cingiloglu	Visual agnosia: An update on disorders of visual recognition
1993	Zoran Georgievski	The effects of central and peripheral binocular visual field masking on fusional disparity vergence
1994	Rebecca Duyshart	Visual acuity: Area of retinal stimulation
1995–7	Not awarded	
1998	Nathan Clunas	Quantitive analysis of the inner nuclear layer in the retina of the common marmoset callithrix

1999	Anthony Sullivan	The effects of age on saccadis mode to visual, auditory and tactile stimuli
2001	Monica Wright	The complicated diagnosis of cortical vision impairment in children with multiple disabilities
2005	Lisa Jones	Eye Movement Control During the Visual Scanning of Objects
2006	Josie Leone	The prognostic value of the cyclo-swap test in the treatment of amblyopia using atropine

PAEDIATRIC ORTHOPTIC AWARD

1999	Valerie Tosswill	Vision impairment in children
2000	Melinda Symniak	
2001	Monica Wright	
2005	Kate Brassington	Amblyopia and reading difficulties
2006	Lindley Leonard	Intermittent exotropia in children and the role of non-surgical therapies

THE MARY WESSON AWARD

1983	Diana Craig (Inaugral)
1986	Neryla Jolly
1989	Not awarded
1991	Kerry Fitzmaurice
1994	Margaret Doyle
1997	Not Awarded
2000	Heather Pettigrew
2004	Ann Macfarlane

PAST PRESIDENTS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA INC

1945-7	Emmie Russell	1963-4	Leonie Collins	1979-80	Mary Carter
1947-8	Lucy Willoughby	1964-5	Lucy Retalic	1980-1	Karen Edwards
1948-9	Diana Mann	1965-6	Beverly Balfour	1982-3	J Stewart
1949-50	E D'Ombrain	1966-7	Helen Hawkeswood	1983-5	Neryla Jolly
1950-1	Emmie Russell	1967-8	Patricia Dunlop	1985-6	Geraldine McConaghy
1951-2	R Gluckman	1968-9	Diana Craig	1986-7	Alison Terrell
1952-4	Patricia Lance	1969-70	Jess Kirby	1987-9	Margaret Doyle
1954-5	Diana Mann	1970-1	Neryla Heard	1989-91	Leonie Collins
1955-6	Jess Kirby	1971-2	Jill Taylor	1991-3	Anne Fitzgerald
1956-7	Mary Carter	1972-3	Patricia Lance	1993-5	Barbara Walsh
1957-8	Lucille Retalic	1973-4	Jill Taylor	1995-7	Jan Wulff
1958-9	Mary Peoples	1974-5	Patricia Lance	1997-00	Kerry Fitzmaurice
1959-60	Patricia Lance	1975-6	Megan Lewis	2000-2	Kerry Martin
1960-1	Helen Hawkeswood	1976-7	Vivienne Gordon	2002-4	Val Tosswill
1961-2	Jess Kirby	1977-8	Helen Hawkeswood	2004-6	Julie Barbour
1962-3	Patricia Lance	1978-9	Patricia Dunlop		

OAA Office Bearers, State Branches & University Training Programs

ORTHOPTIC ASSOCIATION OF AUSTRALIA

OAA OFFICE BEARERS

President: Heather Pettigrew
President Elect: Heather Pettigrew
Vice President: Heather Pettigrew
Treasurer: Liane Wilcox
Secretary: Mara Giribaldi
Public Officer: Susan Sutton

STATE REPRESENTATIVES

Australian Capital Territory: Wendy Holland
New South Wales: Mara Girbaldi, Lindley Leonard, Val Tosswill
Queensland: Heather Pettigrew
South Australia: Hayley Neate
Tasmania: Julie Barbour
Victoria: Zoran Georgievski, Connie Koklanis, Karen Mill
Western Australia: Francine Hyde

STATE BRANCHES

New South Wales:
President: Ann Macfarlane
Secretary: Nhung Nguyen
Treasurer: Lindley Leonard

Queensland:
President: Heather Pettigrew

South Australia:
President: Hayley Neate
Secretary: Tania Straga
Treasurer: Barb Walch

Victoria:
President: Connie Koklanis
Secretary: Julie Ewing
Treasurer: Suzane Vassallo

Western Australia:
President: Francine Hyde

UNIVERSITY TRAINING PROGRAMS

MELBOURNE

Department of Clinical Vision Sciences
Faculty of Health Sciences
La Trobe University
Bundoora, VIC 3086
T: 03 9479 1920
F: 03 9479 3692
http://www.latrobe.edu.au/orthoptics/

SYDNEY

School of Applied Vision Sciences
Faculty of Health Sciences
The University of Sydney
East St, Lidcombe NSW 2141
T: 02 9351 9250
F: 02 9351 9359
http://www.fhs.usyd.edu.au/avs/

INTERNATIONAL ORTHOPTIC ASSOCIATION

International Orthoptic Congress 2008

Reflections on the diversity of current orthoptics

Scientific Programme Committee
- Dr Agneta Rydberg, Chairman, Sweden
- Gail Stephenson, UK
- Mirjam van Lammeren, Belgium

Keynote Speaker
Prof. Eugene HELVESTON
Indianapolis, USA

Abstract Submission Deadline
October 1, 2007

www.ioa2008.org

May 28-31, 2008 - Antwerp, Belgium

ISA, AAPOS and CLADE symposia

02/2007