

ORTHOPTIC
ASSOCIATION
of AUSTRALIA inc

Australian Orthoptic Journal

Australian Orthoptic Journal

Australian Orthoptic Journal

2009 Volume 41 (1)

Myopia, Near Work,
Atropine and Bifocals

Case Conferencing to
Enhance Students'
Learning

Triplopia and Conversion
Disorder?

Brown's Syndrome
Associated with
Goldenhar Syndrome

Double Elevator Palsy
and Congenital Esotropia

AUSTRALIAN ORTHOPTIC JOURNAL – 2009 VOLUME 41, NUMBER 1

- 06 Editorial - Searching AOJ Scientific Papers and Authors
- 07 **Myopia, near work, atropine and bifocals: Critical reflections of the key literature examining the influence of several factors upon the progression of myopia**
Inez Elderman, Meri Vukicevic
- 13 **The Use of Peer-Supported 'Case Conferencing' to Enhance Orthoptic Students' Learning in a Clinical School Environment**
Kulie Robinson, Zoran Georgievski, Konstandina Koklanis
- 17 **A Case of Triplopia: A Case of Conversion Disorder?**
Julie Fitzpatrick
- 20 **A Case of Brown's Syndrome in Association with Goldenhar Syndrome**
Kara Mueke, Linda Santamaria
- 23 **Double Elevator Palsy with Congenital Esotropia: A Case Study**
Marika Hensman
- 26 **Named Lectures, Prizes and Awards of the Orthoptic Association of Australia Inc.**
- 28 **Presidents of the Orthoptic Association of Australia Inc. and Editors of the Australian Orthoptic Journal**
- 29 **OAA Office Bearers, State Branches & University Training Programs**

2009 Volume 41 (1)



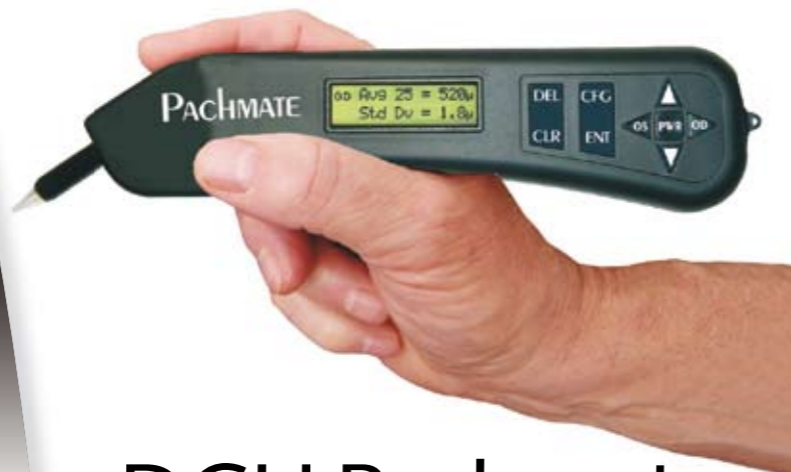
iCARE[®] Tonometer

THE NEW MARKET LEADER

Small, handheld, portable and accurate.
Easy to monitor non-compliant patients.

No anaesthetic or fluorescein required.

icare
FINLAND



DGH Pachmate Pachymeter

SMALL. LIGHT. ACCURATE.

A cordless handheld pachymeter
so small, it even fits in your pocket!

Instant IOP correction.

DGH TECHNOLOGY, INC.

Rayner
www.rayner.com

Talk to Designs For Vision now about the
exciting new developments from Rayner,
including the new Raytrace™ online Toric
IOL calculation system.

Raytrace™



Vision Testing Essentials

Designs For Vision can supply a full range of refractive,
orthoptic and vision testing tools;

- Sheridan Gardner test
- Reading booklets
- Prism bars
- LEA charts
- VA, colour & stereo tests
- Fresnel Prism

And much more - call for details.



DESIGNS FOR VISION www.dfv.com.au
Focused on the future, building on our history

ORDERS + ENQUIRIES
1800 225 307

Moistureyeser

Systane[®] lubricant eye drops restructures the tear
film for sustained comfort, promoting healing.¹⁻⁴



START WITH SYSTANE[®], STAY WITH SYSTANE[®]

References: 1. Lemp MA, Systane extends tear film stability for sustained protection and symptom relief, Advanstar Communications Inc., 2007, 80165B. 2. D'Arienzo P, Ousler III GW, Schindelar MR, A Comparison of Two Marketed Artificial Tears in Improvement of Tear Film Stability as Measured by Tear Film Break-Up Time (TFBUT) and Ocular Protection Index (OPI), Poster presented at the TFOS Meeting, September, 2007. 3. Paugh JR et al. The residence time of artificial tears in dry eye subjects, Paper presented at the American Academy of Optometry Annual Meeting, 2005, San Diego, CA, E-abstract #050062.

4. Meadows D, Is Relief in Sight for the Dry Eye Sufferer? Rev Ophthalmol, 2003, April (pt 2 of 2):10
® Registered Trademark. Alcon Laboratories (Australia) Pty Ltd. ABN 88 000 740 830. 10/25 Frenchs Forest Road East, Frenchs Forest, NSW 2086. POPH.1764 SYS013

Alcon



Systane
LUBRICANT EYE DROPS

Measure at the *speed* of light



*The all-in-one biometer,
providing real choice.*

*Biometry, Keratometry, Pachymetry,
Pupillometry, & IOL calculations*

The Lenstar LS-900® provides a total of 9 precise eye measurements in one easy shot:

- Corneal thickness
- Keratometry
- White to white
- Pupillometry
- Lens thickness
- Anterior chamber depth
- Axial length
- Eccentricity of visual optical line
- Retinal thickness

Optimed have the products, service and experience to make your practice perfect.

Proudly supporting
the Orthoptic profession

F.I.S.O.

DUO



NEW

Chair and Stand with electric rise and fall on table and chair. Table has swivelling action and locks in any position.

CHAIRS & STANDS

Canon

CF-1



NEW

Compact and powerful. Completely redesigned camera with Colour, FA and RF modes. Integrated software package displays stunning high resolution images.

FUNDUS CAMERAS

IRIDEX

NEW



A powerful 532nm laser from Iridex. The TX delivers 2.5W of power. Comes complete with a wireless footswitch and optional remote control.

RETINAL LASERS

NEW



Prism bars, colour vision tests, stereo tests, occluders, fixation aids and children's vision tests.

BITS & PIECES

OPTIMA

NEW



Expanding diagnostic possibilities with 3µm resolution, twice that available in current generation OCT. Normative database, Macula and disk analysis modules.

SPECTRAL DOMAIN OCT

Optimed is a leading supplier of cutting edge instrument technology and Orthoptic equipment. Our team of industry experts is committed to the finest installation, service and support. Call 1300 657 720 today and experience the Optimed difference.

Suppliers of ophthalmic instruments and diagnostic pharmaceuticals • Sydney Melbourne Brisbane Perth Auckland
Australia-wide Phone 1300 657 720 Fax (02) 9420 1144 Email sales@optimed.com.au Web www.optimed.com.au



**MAKES YOUR
PRACTICE PERFECT**

Australian Orthoptic Journal

2009 Volume 41 (1)

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

Editors in Chief

Zoran Georgievski BAppSc(Orth)Hons
Konstandina Koklanis BOrth(Hons) PhD

Editorial Board

Kyle Arnoldi CO COMT (Buffalo NY)
Carolyn Calcutt DBO(D) (London, England)
Nathan Clunas BAppSc(Orth)Hons
Elaine Cornell DOBA DipAppSc MA PhD
Catherine Devereux DipAppSc(Orth) MAppSc
Kerry Fitzmaurice HTDS DipAppSc(Orth) PhD
Mara Giribaldi BAppSc(Orth)
Neryla Jolly DOBA(T) MA

Linda Malesic BOrth(Hons) PhD
Karen McMains BA, OC(C) COMT (Halifax, Nova Scotia)
Jean Pollock DipAppSc(Orth) GradDip(Neuroscience) MAppSc
Gill Roper-Hall DBOT CO COMT
Kathryn Rose DOBA DipAppSc(Orth) GradDip(Neuroscience) PhD
Sarah Shea DBO(D) PhD (Bangor, Wales)
Sue Silveira DipAppSc(Orth) MHealthScEd
Kathryn Thompson DipAppSc(Orth) GradCertHealthScEd MAppSc(Orth)
Suzane Vassallo BOrth(Hons)PhD
Meri Vukicevic BOrth PGDipHlthResMeth PhD

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: Aug 2009).

Editors' details: Zoran Georgievski, z.georgievski@latrobe.edu.au; Konstandina Koklanis, k.koklanis@latrobe.edu.au; Department of Clinical Vision Sciences, La Trobe University. Fax: +61 3 9479 3692. Email: AOJ@orthooptics.org.au. Design & layout: Campus Graphics, La Trobe University. Printer: Printing Edge Melbourne Pty Ltd. Distributor: Orthoptic Association of Australia Inc (193 Surrey Hills VIC 3127 Australia).

All rights reserved. Except as permitted by the Copyright Act 1968, pursuant to a copying licence you may have with the reproduction rights organisation Copyright Agency Limited (www.copyright.com.au) or if the use is for personal use only, no part of this publication may be reproduced, stored in a retrieval system, communicated or transmitted in any form or by any means; electronic, mechanical, photocopying, recording or otherwise; without prior permission of the copyright owners. By publishing in the Australian Orthoptic Journal, authors have conferred copyright ownership to the Australian Orthoptic Journal. Copyright 2009 © Australian Orthoptic Journal 2009. All rights reserved.



Advertising in the Australian Orthoptic Journal

For information on advertising, please contact our Advertising & Sponsorship Manager, Karen Mill k.mill@orthooptics.org.au or AOJ@orthooptics.org.au

Advertisements can be full page (210 x 297 mm, plus bleed), half page (186 x 135.5 mm) or quarter page (90 x 135.5 mm).

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 SUBMISSION

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

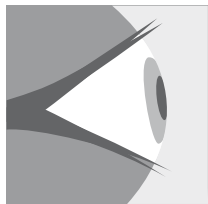
Tel: Assoc. Prof. Zoran Georgievski 03 9479 1919

Dr. Connie Koklanis 03 9479 1903

Fax: 03 9479 3692.

Australian Orthoptic Journal

2009 Volume 41 (1)



ORTHOPTIC
ASSOCIATION
of AUSTRALIA inc

CONTENTS

- 06 Editorial - Searching AOJ Scientific Papers and Authors**
- 07 Myopia, Near Work, Atropine and Bifocals: Critical Reflections of the Key Literature Examining the Influence of Several Factors Upon the Progression of Myopia**
Inez Elderman, Meri Vukicevic
- 13 The Use of Peer - Supported 'Case Conferencing' to Enhance Orthoptic Students' Learning in a Clinical School Environment**
Kylie Robinson, Zoran Georgievski, Konstandina Koklanis
- 17 A Case of Troplopia: A Case of Conversion Disorder?**
Julie Fitzpatrick
- 20 A Case of Brown's Syndrome in Association with Goldenhar Syndrome**
Kara Muecke, Linda Santamaria
- 23 Double Elevator Palsy with Congenital Esotropia: A Case Study**
Marika Hensman
- 26 Named Lectures, Prizes and Awards of the Orthoptic Association of Australia Inc.**
- 28 Presidents of the Orthoptic Association of Australia Inc. and Editors of the Australian Orthoptic Journal**
- 29 OAA Office Bearers, State Branches & University Training Programs**
-

Editorial

Searching AOJ Scientific Papers and Authors

One of the many challenges of running a small 'society-based' journal, such as our Australian Orthoptic Journal, is in trying to attract contributing authors from a research base who are willing to submit papers for publication. This is in part because our journal does not have a 'journal impact factor', which is sought by universities; but also because of problems with searching publications that are not included in various databases (like Medline). Our journal is not unique in this regard - there are 9 other orthoptic journals world-wide with exactly the same problem that we have.

Some time ago, the AOJ had discussion and signed up with Informit (<http://search.informit.com.au/>, who are part of RMIT Publishing) for the promise of 'searchability' for our contributors' works and so to disseminate Australian orthoptists' published work as best as possible. The results have been fantastic.

We have recently become aware that AOJ papers, even the editorials we write or invite to be written and the abstracts we include, come up on a Google Scholar search <http://scholar.google.com.au/> - try it by typing your name into Google Scholar in the format e.g. "Z Georgievski".

This is a great development and milestone for the Australian Orthoptic Journal, and should stimulate people to submit their manuscripts to further this peer-reviewed (albeit 'society-based') scientific orthoptic publication, which is indeed the only English-language orthoptic journal in the world that is issued semiannually.

Zoran Georgievski & Connie Koklanis

Department of Clinical Vision Sciences
La Trobe University

Myopia, Near Work, Atropine and Bifocals: Critical Reflections of the Key Literature Examining the Influence of Several Factors on the Progression of Myopia.

Inez Eveline Elderman, DipOrth&Optom¹
Meri Vukicevic, PhD²

¹Department of Ophthalmology & Neuroscience, Royal Melbourne Hospital, Melbourne, Australia

²Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia

ABSTRACT

In the last century there have been many studies into the factors that influence the progression of myopia. Genetics, exposure to light, intra ocular pressure, near work, stress, presence of esophoria, level of education and living environment are described as possible factors influencing myopia. Some studies¹⁻³ indicate that there is a possible connection between near work and myopia progression and other studies suggest that methods to delay myopia progression are negligible^{4,5}. The literature shows that it is impossible to measure the amount of influence each factor has on the progression of myopia as it is not possible to separate one individual factor from another. The exact mechanism that causes myopia progression is not known

and there are no evidence based studies that document what the causes may be. Whilst it is known that genetics have an influence, it is also possible that reading and near work have influence on myopia. Thus, could the progression of myopia be delayed with treatment such as atropine and bifocals?

The purpose of this paper is to investigate the factors that may contribute to myopia progression as outlined in the literature and to consider, by comparing two key papers, whether the use of atropine and bifocals is effective treatment. In addition, important considerations from an orthoptic perspective are also described.

Keywords: myopia, progression, atropine, bifocals

INTRODUCTION

Myopia is a common public health problem throughout the world and there are many adverse eye health care problems that can be associated with it⁶. Through the last few decades there have been many researchers who have investigated which factors have influence on myopia progression and whether it is possible to stop or delay this progression. Reading is documented as one of the most significant factors influencing the progression of myopia⁷⁻⁹.

A patient with myopia has an eye where the refractive index is unrelated to its axial length¹⁰. Young children are normally hypermetropic and if a child younger than 3 years is emmetropic there is a greater chance that he or she will develop myopia. The cause of myopia can be related to the lens or to the axial length of the eye. With lenticular myopia the lens is too thick and in turn the refractive index is too high, or the eye is of normal size but the corneal curvature

is too high. In pure axial myopia the axial length of the eye is too long but the optical components are normal. There are 3 different types of myopia: physiological or low myopia (up to -2.00 dioptres); intermediate or moderate myopia (from -2.00 to -4.00 dioptres) and pathological or high myopia (greater than -6.00 dioptres). Myopia can also be categorised by age according to Grosvenor's classification system¹¹ with congenital or early onset myopia occurring from ages 5 to 12 years or late onset myopia from adulthood¹².

FACTORS THAT HAVE AN INFLUENCE ON MYOPIA

Myopia is a common public health issue mainly in Asian countries where it has a larger impact compared with Australian or European countries and it has been reported that 75% to 80% of the Asian population has myopia^{7,13-17}. Many studies have investigated effective treatment or prevention of myopia but to compare these studies it is important to investigate the factors that have influence on myopia progression. All ocular activities have an influence upon refractive error and inevitably undertaking near work and reading at a further focal distance reduces myopia

Correspondence: Meri Vukicevic
Department of Clinical Vision Sciences, La Trobe University, Vic 3086, Australia
Email: m.vukicevic@latrobe.edu.au

progression⁸. The progression of axial myopia in monkeys as a result of form and light deprivation has been reported^{8,18} and other researchers suggest that accommodation, convergence, performance of daily living tasks, level of education, intra-ocular pressure, exposure to light and esophoria also have an influence^{1,19-22}. Genetics however, are probably one of the largest factors causing myopia and one paper suggests that the children of myopic parents have longer eyes even before they have myopia^{8,23}. Genetic factors cannot be denied in the refractive status of the patient and the specific genes for myopia have been identified^{24,25}. However, the genetic factor is not the only issue as there has been an increase in the incidence of myopia in the last decade that cannot be explained solely by genetic factors and researchers suggest that near work is the other reason for the increase in myopia². Wu and Edwards⁹ conducted a study on familial myopia over three generations and conclude that the chance of myopia in children is five times greater if the parents and grandparents are also myopic. The chance of developing myopia in children was greater in the last three generations which concludes that probably it is not only genetic factors which influence the progression and that environmental factors may also play a part. Wu and Edwards⁹ describe that the chance of a child from the third generation developing myopia is 22% when there is no parent with myopia and the chance is 30% if there is one parent with myopia and 46% if both parents are myopic. Mutti et al¹ suggest that the chance of a child with two myopic parents developing myopia is 30 to 40%, 20 to 25% with one myopic parent and smaller than 10% without myopic parents.

There are three possible hypotheses that explain the relationship between near work and myopia and are presented in Table 1.

Table 1. Hypothesis about the influence of near work on myopia

| Researchers | Hypotheses |
|---------------------------|--|
| Coleman ²⁶ | <ul style="list-style-type: none"> • Accommodation causes permanent change in the convexity of the lens. • The ciliary muscle holds the lens in the accommodative position contributing to new lens vessels growth. • If this persists, it can result in permanent change. |
| Smith et al ²⁷ | <ul style="list-style-type: none"> • Biochemical processes cause the eye to grow. • These biochemical processes exist when there is a blurred image on the retina |
| Young ²⁸ | <ul style="list-style-type: none"> • A relationship exists between accommodation and intra ocular pressure. • During accommodation the volume of the posterior chamber is compressed and the pressure increases • This causes pressure on the sclera and may lead to an increase in the axial length, mainly in patients (especially in children) where the sclera is more flexible |

Three significant studies, those by Mutti et al¹, Saw & Nieto²² and Zylbermann and Landau³ specifically address the influence of near work on myopia.

Mutti et al¹ suggest that children with myopia are more likely to have parents with myopia. Myopic children are also more likely to spend significantly more time reading and studying and less hours playing sport compared with emmetropic children. In addition, myopic children performed better on measures of reading and language compared to their emmetropic counterparts, although the interviews used to determine this were subjective and required parental response. One particular problem with the study by Mutti et al is that 'watching television' had been classified by the researchers as near work and all refractions performed used 1% tropicamide and autorefraction without the use of cycloplegia. However, a positive relationship was found between family history, increased near work and the development of myopia.

Unlike Mutti et al, Saw & Nieto did not specifically investigate family history and the number of myopic parents prior to commencement of the study. Instead they used a questionnaire to compare myopic children residing in Chinese cities and those in rural areas and retrospectively discovered that children from urban areas were more likely to have a family history of myopia. The researchers also found that parents of the city children had higher levels of education. The children from urban areas spent less time on school activities compared with those in rural areas and those with myopia spent 2.3 hours a day on near work compared with non-myopic children who performed near work for 1.9 hours a week. The conclusion of Saw & Nieto is consistent with Mutti et al in that there is a positive association between near work, genetic factors and myopia.

Zylbermann and Landau³ undertook a much larger study compared with Saw & Nieto and Mutti et al and investigated the prevalence and degree of myopia in 870 Jewish students and compared students attending single sex public schools and single sex religious orthodox schools. It is important to note that the authors describe a difference in the amount of near work undertaken by boys in the orthodox school, who are required to read for three hours per day from age 4 to age 13 after which they study for 16 hours a day. In addition, the sustained near vision is affected by changes in print size of the text and swaying of the upper torso which results in frequent changes in accommodation. The female students attending the orthodox schools and the students attending public schools have a similar education without the high volume of near work. Figure 1 shows the prevalence of myopia in students from the different schools, with the highest prevalence in boys attending religious schools.

Zylbermann and Landau suggest that the amount of near work is a contributing factor to the progression of myopia but does not completely rule out the influence of family history. The authors suggest that the student's ethnicities

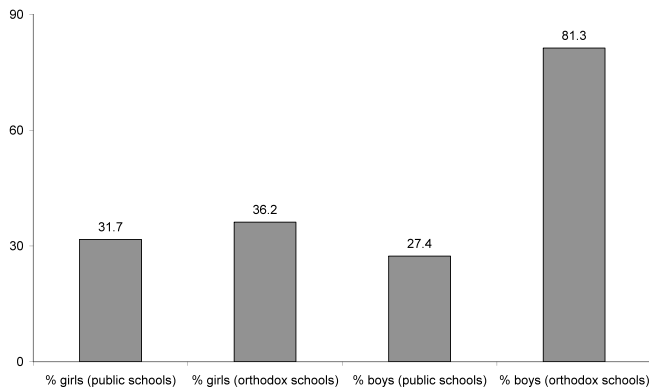


Figure 1. Prevalence of myopia in children, by sex and school. Adapted from Zylbermann and Landau³

are comparable, but did not research ethnicity or family history as part of the study. The researchers also compare the findings to animal studies, however, it has been suggested that it is not possible to relate human studies to animal ones as the eyes are not comparable. Also, the studies used for comparison were conducted on infantile animals, however main myopia progression in humans occurs in the juvenile period (the primate infant period being up to 2 years and the juvenile period after the age of 2 years until puberty)^{8,29}.

THE USE OF ATROPINE IN COMBINATION WITH BIFOCALS TO DELAY MYOPIA PROGRESSION

If the suggestion is true that accommodation has any influence on myopic progression, then this progression might be delayed or even halted with the use of atropine and bifocals. Chiang et al⁴ and Syniuta and Isenberg⁵ conducted a study to investigate the treatment of atropine and bifocals and whether this had an influence on the progression of myopia and these studies are compared. Whilst both studies investigated the combined use of atropine and bifocals, they were conducted in different parts of the world with participants of different ethnicities. A comparison of the characteristics of both studies is shown in Table 2a and 2b with critical reflections of the features of both studies emphasised by the grey highlighted areas.

The study by Syniuta and Isenberg⁵ was a small pilot study compared with Chiang et al's large study which included 706 participants. There were a greater proportion of females to males in both studies and a slight difference in their average age. Both studies did not include investigation of family history of myopia. There was also a difference in the average treatment time given to participants in both studies. The study by Chiang et al did not incorporate a control group, but compared results with a previous

Table 2a. A comparison of the studies by Chiang et al⁴ and Syniuta and Isenberg⁵

| Study Features | Chiang et al ⁴ | Syniuta and Isenberg ⁵ |
|---|--|---|
| Participants randomly chosen | No | No |
| Number of participants | 706 | 30 |
| Male | 296 (42%) | 12 (40%) |
| Female | 410 (58%) | 18 (60%) |
| Age | 6 - 16 years | 4 - 13 years |
| Average Age | Unkown | 8 years |
| Ethnicity | Caucasian race | Not investigated |
| Investigation of family history of myopia | No | No |
| Time of study | 12 years | Unknown |
| Average treatment time | 3.62 years (range: 21 days - 10.1 years) | 2.4 years (29.3 months) (range: 3 months - 96 months) |
| Control group | None Comparison made with a differrent longitudinal study | Yes |
| Compliance measured | Using questionnaire to parents | No |
| Vision chart used | Unknown | Snellen-chart |
| Review time | Once per year | Once per 6 months |
| Investigator | Unknown | Technical Nurse |
| Method of refraction | With cyclopentolate (Objective, subjective and auto-refraction included) | With cyclopentolate (Objective and subjective included) |
| Glasses prescription given to participants | Yes | Yes |
| Use of photochromatic glasses | Yes | Yes |
| Size of addition prescribed | 2.25 dioptres | 2.50 dioptres |
| Ocular pathology such as strabismus and amblyopia excluded? | Yes | Yes |
| Refraction transcribed into the spherical equivalent? | Yes | Unknown |
| Was atropine used, how often? | For 1st two years every other day then for 5 years once per week (1% atropine) | Daily (1% atropine) |
| Number of patients with low myopia (0.00 - 2.00 dioptres) | 472 (69%) | 11 (73%) |
| Number of patients with moderate myopia (2.00 - 6.00) | 215 (31%) | 4 (26%) |
| Total number of patients | 687 (100%) | 15 (100%) |

Table 2b. Average yearly myopia progression:

| Study Features | Chiang et al ⁴ | Syniuta and Isenberg ⁵ |
|---|-----------------------------------|---|
| Low myopes with use of atropine | 0.11 (+/-0.2) dioptres each year | 0.038 (+/-0.71) dioptres each year |
| Low myopes without use of atropine | No matching control group | 0.76 (+/-0.26) dioptres each year Control group from own study |
| Moderate myopes with use of atropine and bifocals | 0.16 (+/-0.05) dioptres each year | 0.19 (+/-0.38) dioptres each year |
| Moderate myopes without use of atropine | No matching control group | 1.05 (+/-0.11) dioptres each year Control group from own study |
| All myopes with use of atropine and bifocals | 0.05 (-0.14 dioptres each year)* | 0.05 (+/-0.26) dioptres each year |
| All myopes without use of atropine | 0.24 (-0.91 dioptres each year)** | 0.84 (+/-0.26) dioptres each year Control group from own study |

* Average variation of 4 longitudinal studies

** Average variation of 8 longitudinal studies

longitudinal study which was conducted in a different part of the world using participants with different ethnicities. As suggested by Fulk et al³⁰, different outcomes may arise with different ethnic groups and the degree of myopia can also differ between ethnic groups.

Chiang et al investigated participant compliance with treatment with the use of a questionnaire given to the parents and a comparison was made between participants that had complete compliance to those with moderate compliance. Syniuta and Isenberg on the other hand did not test for compliance and the exact amount of hours that the patients wore their glasses was not reported in either study. Whilst all participants in Chiang et al's study had an examination yearly, the researchers do not indicate what type of chart was used to measure vision and whether this was consistent for all participants. Annual review of patients receiving treatment with atropine and bifocals is considered too infrequent and as some authors suggest, the chance of bilateral amblyopia or hypo accommodation is present and would not be identified with such a long duration between visits. Also, an increased risk of adverse side effects including dryness of the mouth and skin, fever, delirium, tachycardia and a chance of allergic reaction or hyper toxicity can occur^{31,32}.

Whilst both research teams performed refraction using cyclopentolate, it is not clear whether objective refraction was performed with retinoscopy or by autorefraction. Some similarities in study design included the prescription of photochromatic glasses to patients to minimize light sensitivity and photophobia and the exclusion of ocular pathology including strabismus and amblyopia. Moreover, the near addition prescribed to patients in both studies was almost identical.

The use of atropine (1%) however, differed between the two studies. One study⁴ prescribed it for use every two days for the first two years and thereafter to be used weekly. The other study prescribed the use of atropine on a daily basis⁵.

In both studies, participants were divided into two groups for monitoring the yearly progression of myopia. It was found that low myopes using atropine in Chiang et al's study progressed more than those in Syniuta and Isenberg's study whilst the opposite was true for moderate myopes. Overall, myopes using atropine had similar progression patterns in both studies which were very small, whilst those not using atropine had greater progression of myopia. However the variation is very high as can be seen in Table 2, so it is still unclear exactly what level of effect atropine and bifocal treatment have. Whilst these authors conclude that myopia is delayed by giving atropine and bifocal treatment, the question that then arises is what happens to these patients after atropine and the use of bifocals is ceased?

A possible answer to this question can be found in a paper by Fulk et al³⁰ who conducted a similar study to Syniuta and Isenberg's and to Chiang et al's but only used bifocals as a treatment option. The conclusion was that the myopia will increase again soon after wearing bifocals has ceased. The level of myopia after the use of bifocals is stopped was found to be the same as that in participants who were not prescribed bifocals. Another important factor not considered in these papers is that of the influence of ethnicity upon myopic development and the differences in degree of myopia in various ethnic groups has been documented and underwrites the importance of family history and genetic factors. This is especially pertinent for Chiang et al's study as the researcher compares findings to the results of eight different studies using participants of various ethnicities. The opinion that pharmaceutical and lens therapies for myopia mostly have small treatment benefits, last for a short period of time and have significant side effects, is further supported by a more recent review conducted by Gwiazda³³.

IMPORTANT INVESTIGATIONS AND CONSIDERATIONS FROM AN ORTHOPTIC PERSPECTIVE

The influence of hereditary factors upon myopia development are well known^{1,2,8,14,15,23}. Therefore history taking and accurate documentation of family history is one of the most important tasks conducted by the orthoptist. In addition, a patient at onset of myopia often presents with asthenopic symptoms and this can lead to de-compensation of a latent deviation. Therefore, careful investigation of binocular function, including near and distance cover testing is imperative. If a latent deviation is present, prism cover testing to measure the size of the phoria also provides important information.

In the presence significant asthenopic symptoms, assessment of fusion to determine whether it is within normal limits can assist with excluding decompensation of the phoria as a contributing factor to the asthenopia. Accommodation and convergence tested on the RAF gauge or testing of accommodation using dynamic retinoscopy is especially useful in children with speech problems or handicap.

Assessment of ocular motility is also an important investigation. High myopes often present with mechanical motility problems due to the size of the eye in the orbit. Motility problems in this instance need to be carefully differentiated from VIth nerve palsies, divergence insufficiency, Graves Ophthalmopathy and accommodative-convergence spasm^{34,35}. One must also carefully investigate visual acuity prior to cycloplegic refraction and exclude pseudo myopia caused by accommodation. The ocular media and fundus also require examination as high myopes have increased prevalence of retinopathy.

If the presence of exophoria or exotropia is found on examination, this needs to be fully corrected in myopic patients as better vision leads to better control of the exo deviation.

In the presence of an esophoria or esotropia, a small under correction of the myopic prescription might control a latent or manifest deviation, especially when there is an accommodative factor involved. However, under correction is only advocated if it is certain that this will improve the eso deviation, binocular vision, provide adequate visual acuity and relieve asthenopic complaints³⁵. Young children, however, should always be fully corrected to ensure full development of the visual system.

CONCLUSION

Can we conclude that reading or near tasks have an influence on the progression of myopia? All the studies examined in this paper conclude that there is a possible relationship between near work in addition to genetic factors in the development and progression of myopia. However it is still not clear which factor has which effect and the studies illustrate the difficulty of answering this question. Some studies did not directly address family history, the number of myopic parents and their degree of myopia, whilst others did not differentiate between participants of different ethnicities and it has been suggested that the degree and prevalence of myopia will differ between ethnic groups⁷. In addition, several researchers used each others flawed findings to compare results and to suggest limited conclusions to the question.

According to the literature, it can be suggested that if near tasks have any influence on myopia, then the use of atropine and bifocals might stop or delay this myopia progression. Chiang et al⁴ and Syniuta and Isenberg's⁵ studies suggest

that myopia can almost be completely delayed using atropine and bifocals. As shown in table 2, the effect, if any, is minor and the variation is large. In addition, family history, number of myopic parents and their degree of myopia and ethnicity have not been investigated and these factors can greatly influence final results. There are also some issues related to the review time of participants and the increased chances of amblyopia and hypo accommodation. In addition, atropine is a very strong medication to give a child for such a long period of time, adverse reactions may occur and there may be psychological effects upon the child when they are given bifocals^{31,36}. Quality of life and psychological factors have not been addressed in any of these studies. Therefore, considering all of these issues, it is unlikely that the use of atropine and bifocals should be given consideration as a treatment option.

It is difficult to compare the direct relationship between near tasks and myopia progression as there are so many factors that can influence and skew the results. In addition, little work has been conducted that considers factors such as accommodation, fusion and latent deviations. For example, a patient with a large latent exophoria who uses accommodative effort to control the latent squint could decompensate with the use of atropine and as mentioned previously, young children can lose binocularity and there is an increased chance of amblyopia. In addition, the reality of what happens once atropine and bifocal therapy is ceased is impossible to gauge.

Whilst there has been a documented increase in the prevalence of myopia in Asian countries and this seems to be as a result of increased near tasks, the degree of influence of near vision upon the progression of myopia is still in contention. Objective prospective research over three generations would offer more answers about the influence of near vision upon myopia progression.

REFERENCES

1. Mutti D, Mitchell G, Moeschberger M, Jones L, Zadnik K. Parental myopia, near work, school achievement and children's refractive error. *Investigative Ophthalmology and Visual Science*. 2002;43:3633-40.
2. Saw S, Hong R, Zhang M, Fu Z, Ye M, Tan D, et al. Near work activity and myopia in rural and urban schoolchildren in China. *Journal of Paediatric and Ophthalmology Strabismus*. 2001 May-June;38:149-55.
3. Zylbermann R, Landau D. The influence of study habits on myopia in Jewish teenagers. *Journal of Paediatric Ophthalmology and Strabismus*. 1993;30:319-22.
4. Chiang M, Kouzis A, Pointer R, Repka M. Treatment of childhood myopia with atropine eyedrops and bifocal spectacles. *Binocular Vision and Strabismus Quarterly*. 2001;16:209-15.
5. Syniuta L, Isenberg S. Atropine and bifocals can slow the progression of myopia in children. *Binocular Vision and Strabismus Quarterly*. 2001;38:149-55.
6. Taylor D, Hoyt C. *Practical Pediatric ophthalmology*. 1st ed. Sydney: Blackwell Scientific; 1997.

7. Frederick D. Myopia: was mother right about reading in the dark? *British Journal of Ophthalmology*. 2001;85:509-12.
8. Wildsoet C, Norton T. Toward controlling myopia progression. *Optometry and Vision Science*. 1999;76:341-2.
9. Wu M, Edwards M. The effect of having myopic parents: an analysis of myopia in three generations. *Optometry and Vision Science*. 1999 June;76:387-92.
10. Lith G. *Inzicht in zien*. 1st ed. Rotterdam: Lioness Mediaforce BV; 1995.
11. Grosvenor T. A review and suggested classification of myopia on the basis of age-related prevalence and age of onset. *American Journal of Optometry and Physiological Optics*. 1987;64:545-54.
12. Pastor D. *Oogmeetkunde in de Praktijk*. Houten/Diemen: Bohn Stafleu van Loghum; 1995.
13. Edwards M. The development of myopia in Hong Kong children between the ages of 7 and 12 years: a five-year longitudinal study. *Ophthalmic and Physiological Optics*. 1999;19:286-94.
14. Lam C, Goh W. The incidence of refractive errors among school children in Hong Kong and its relationship with the optical components. *Clinical and Experimental Optometry*. 1991;74:97-103.
15. Lin L, Shih Y, Hsiao C, Chen C, Lee L, Hung P. Epidemiologic study of the prevalence and severity of myopia among school children in Taiwan in 2000. *J Formos Med Assoc*. 2001;100:684-91.
16. Saw S, Shankar A, Tan S, Taylor H, Tan D, Stone R, et al. A cohort study of incident myopia in Singaporean children. *Investigative Ophthalmology and Visual Science*. 2006;47:1839-44.
17. Yap M, Wu M, Wang S, Lee F, Liu Z. Environmental factors and refractive error in Chinese children. *Clinical and Experimental Optometry*. 1994;77:8-14.
18. Raviola E, Wiesel T. An animal model of myopia. *New England Journal of Medicine*. 1985 October;62(10):680-8.
19. Goss D, Rainey B. Relationship of accommodative response and nearpoint phoria in a sample of myopic children. *Optometry and Vision Science*. 1999;73:292-4.
20. Jensen H. Myopia in teenagers. *Acta Ophthalmologica Scandinavica*. 1995;73:389-62.
21. Rose K, Ip J, Robaei D, Huynh S, Kifley A, Smith W, et al. Near-work and outdoor activities and the prevalence of myopia in Australian school students aged 12-13 years: the Sydney Myopia Study. *Investigative Ophthalmology and Visual Science*. 2006;47:E-abstract 5453.
22. Saw S, Nieto F. Distance, lighting and parental beliefs: Understanding near work in epidemiologic studies of myopia. *Optometry and Vision Science*. 1999;76:355-62.
23. Zadnik K, Mutti D, Friedman N. Ocular predictors of the onset of juvenile myopia. *Investigative Ophthalmology and Visual Science*. 1999;40:1936-43.
24. Naiglin L, Gazagne C, Dallongeville F. A genome wide scan for familial high myopia suggests a novel locus on chromosome 7q36. *Journal of Medical Genetics*. 2002;39:118-24.
25. Young T, Ronan S, Alvear A. A second locus for familial high myopia maps to chromosome 12q. *American Journal of Human Genetics*. 1998;63:1419-24.
26. Coleman D. On the hydraulic suspension theory of accommodation. *Transactions of the American Ophthalmological Society*. 1986;84:846-68.
27. Smith E, Hung L, Kee C, Qiao Y. Effects of brief periods of unrestricted vision on the development of form-deprivation myopia in monkeys. *Investigative Ophthalmology and Visual Science*. 2002;43:291-9.
28. Young T. On the mechanism of the eye. *Philadelphia Trans*. 1801;1:23-88.
29. Zadnik K, Jones L, Irvin B. Myopia and night-time vision. *Nature*. 2000;404:43-4.
30. Fulk G, Cyert L, Parker D. A randomized clinical trial of bifocal glasses for myopic children with esophoria results after 54 months. *Optometry* 2002 August;73:470-6.
31. Luiten M, Trap N. *Geneesmiddelen en het oog*. Barendrecht: Derde druk; 1999.
32. Young F. The nature and control of myopia. *Journal of the American Optometric Association*. 1977;48:451-7.
33. Gwiazda J. Treatment options for myopia. *Optometry and Vision Science*. 2009;86:624-8.
34. Gutter M, Jellema H, Wijnen-Segeren I. *Orthoptische diagnostiek*. Ridderkerk: Luiten; 2001.
35. Von Noorden G. *Binocular vision and ocular motility*. 5th ed. Houston: Mosby; 1996.
36. Myers T, Wallace D, Johnson S. *Ophthalmic medications in pediatric patients*. *Comp Ophthalmological Update*. 2005;6:85-101.

The Use of Peer-Supported 'Case Conferencing' to Enhance Orthoptic Students' Learning in a Clinical School Environment.

Kylie Robinson, BAppSc(Orth)¹
Zoran Georgievski, BAppSc(Orth)^{1,2}
Konstandina Koklanis, PhD²

¹Department of Clinical School of Orthoptics, Royal Victorian Eye and Ear Hospital, Melbourne, Australia
²Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia

ABSTRACT

An orthoptic student 'case conferencing' program was developed and introduced at the Royal Victorian Eye and Ear Hospital with the aim of enhancing students' clinical experience. The aim of this study was to report on this initiative and on students' perceptions of the program. Students presently undertake their clinical placements in differing modes, according to the semester in which they

are enrolled. It was found that students undertaking the 'block' placement mode find case conferencing particularly beneficial, the key difference being the increased amount of contact time and engagement compared with students undertaking sessional placement.

Keywords: case conferencing, peer-mentoring, clinical placements

INTRODUCTION

The Royal Victorian Eye and Ear Hospital (RVEEH) provides La Trobe University with its greatest number of undergraduate orthoptic student clinical placements (approximately 40-50% of all in proportion), and therefore accommodates up to several students on site at any one time. Whilst having numerous students on placement presents logistical challenges, it provides the unique opportunity for students, as peers, to support and learn from one another. Previous studies have, for instance, demonstrated positive peer mentoring experiences in orthoptics^{1,2}. Mentoring programs provide a rich learning experience and opportunities for collegial interaction and the development of various skills such as communication, the practice of leadership, and an understanding of the role of research and evidence based practice^{3,4}.

In the first semester of 2008, student 'case conferencing' was introduced at the RVEEH with the aim of enhancing students' clinical experience by ensuring optimal use of their clinical placement time. Within this program, an opportunity was created for students to benefit and learn from each other's clinical experiences and indeed encounters with patients and clinical educators.

In the broader context, case conferencing is promoted and encouraged to better manage and enhance patient care. In 1999 Australia introduced Medicare Benefits Schedule rebates for case conferencing (which includes orthoptists within multidisciplinary teams) with the aim of improving preventive healthcare and shifting from episodic care to providing longer-term care in a coordinated approach with collaboration of a wider healthcare team⁵. As such, case conferencing has increasingly become an integral part of the role of a health professional. In the medical setting, case conferencing provides useful information exchange between clinicians who may work within different specialties or disciplines. Case conferencing between health professionals has also been highlighted as being important in areas such as aged care, palliative care, diabetic care, mental illness and medical diagnosis⁶⁻¹⁰.

Although the purpose and design of orthoptic student case conferencing differs to case conferencing among health professionals, we believed that it would nevertheless allow for these skills to be developed for potential application later. This paper reports on the orthoptic student case conferencing program developed at the RVEEH and on students' perceptions of the program.

METHODS

La Trobe University orthoptic students were allocated to the RVEEH as part of their clinical placement program in

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

Table 1a. Forced Choice Survey Questions

| Question | Forced Choice | | | | | | |
|--|-------------------|---|---|---|---|---|----------------|
| 1. Case conferencing at the end of my clinic was a valuable part of my clinical placement- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 2. The time permitted for case conferencing was appropriate- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 3. Case conferencing encouraged me to clarify problems or answer questions that I had during my clinic- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 4. I took the opportunity during case conferencing to impart knowledge or information I gained during the clinic to my peers- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 5. Case conferencing improved my confidence in clinic- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 6. The necessary resources (texts, internet, and library access) were available to us to facilitate our case conferencing- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |
| 7. I felt supported by my clinical supervisor/s during case conferencing or in preparation for it, and assistance was readily available- | Strongly Disagree | 1 | 2 | 3 | 4 | 5 | Strongly Agree |

Table 1b. Open Ended Survey Questions

| Question |
|--|
| 8. How did you / your peers decide what to discuss during case conferencing? |
| 9. What was the best thing(s) about case conferencing |
| 10. How can case conferencing be improved? |

semesters 1 and 2 of 2008. During semester 1, students enrolled in either second, third or fourth year of the program attended clinics on a 'sessional' basis (one half day per week) for 12 weeks. During Semester 2 (and indeed the second half of the year), on the other hand, full-time 'block periods' were provided to third year students. Each block period consisted of 4 consecutive weeks of clinical placement. Almost all of the 24 third year students had at least one block period at the RVEEH.

During their placement at the RVEEH, students attended various general and special eye clinics in the hospital. Towards the end of each clinical session, students convened their case conferencing meeting in a designated room. Up to 5 students were present and the duration of the meeting was approximately 30 minutes. Students were encouraged to each contribute a topic, an issue or to report a patient case for discussion with the rest of the group.

A clinician was not present during these meetings as the purpose was for the students to have a forum to openly discuss with peers their ideas and what they learnt, their experiences and various clinical techniques they were exposed to. However, if students raised questions that could not be answered by their group peers, clinicians were available for assistance. The students were also provided with access to resources such as the internet and the department and hospital libraries.

At the end of the students' placement period, a survey was disseminated (by email or in person) to evaluate their case conferencing experiences. The survey (Table 1a and 1b)

consisted of seven forced-choice questions (with 5 options: 'strongly disagree', 'disagree', 'neutral', 'agree' or 'strongly agree') and 3 open-ended questions.

Two differing groups of students across two semesters were hence given the opportunity to experience case conferencing and to evaluate the program. The two groups differed not only in terms of their year and experience level, but in terms of their mode of clinical placement and therefore amount of weekly contact time at the RVEEH.

RESULTS

There were 33 students who responded to the survey of the 64 who attended the RVEEH in 2008. Figure 2 represents the relative proportions of students who responded favourably (with either 'agree' or 'strongly agree') to the first seven forced-choice questions or statements that were presented for quantitative analysis. For example, the first statement was "case conferencing was a valuable part of the clinical placement". In this instance, students in the semester 1 sessional placement responded favourably nearly 40% of the time, whilst students in the semester 2 block placement responded favourably 80% of the time. As can be seen in Figure 1, this trend was evident for all questions or statements. That is, students undertaking the block placements and attending the RVEEH daily for the 4 week period viewed their case conferencing experience more favourably overall.

The responses to the three qualitative open-ended questions are summarised in Table 2. Students generally discussed patient cases and clinical skills learnt and appreciated the discussion and resolution of issues and questions in a supported peer environment. Improvements related to the enhancement of resources and further involvement of clinicians. Ready access to the online resources during case conferencing was made available in semester 2 as a direct result from early feedback.

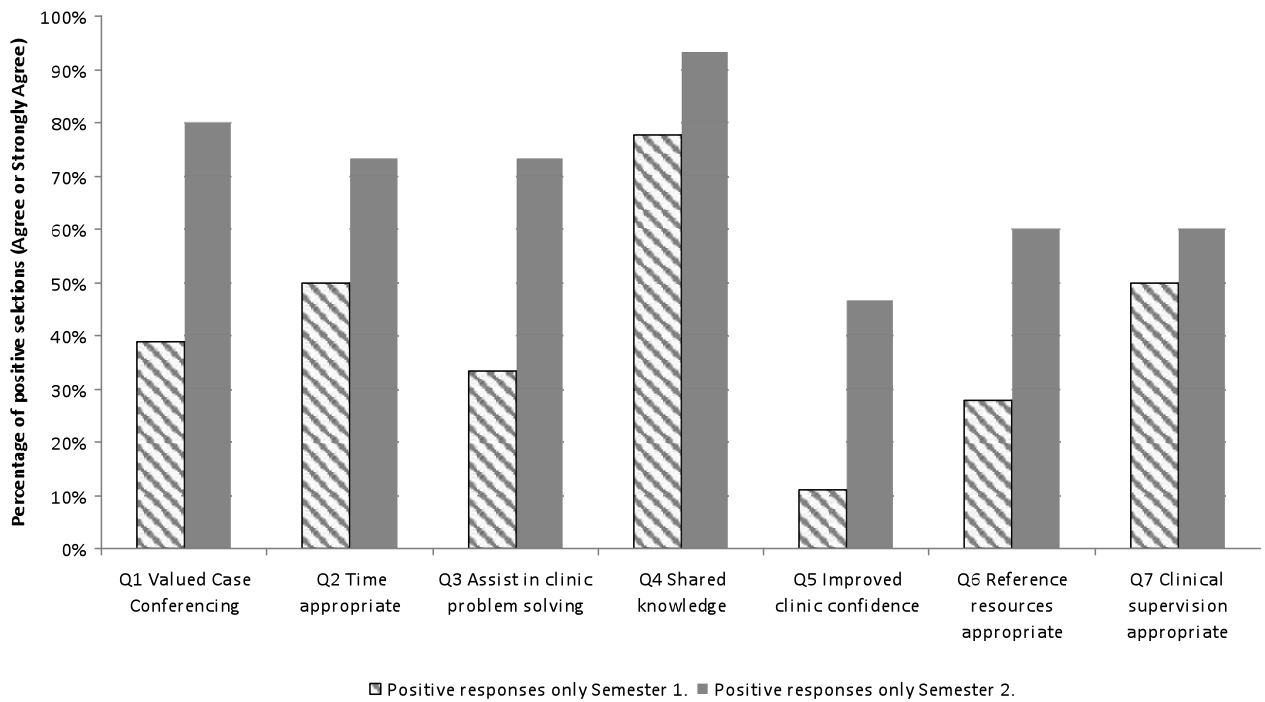


Figure 1. Relative proportions of favourable responses for semester 1 and 2

| Question | Responses |
|---|--|
| How did you / your peers decide what to discuss during case conferencing? | <ul style="list-style-type: none"> Overwhelmingly they responded that they discussed about what captured their interest during the clinic; They shared information regarding new techniques and skills they each learned during the clinic; and They used case conferencing to resolve problems they might have encountered with patients during the clinic. |
| What was the best thing(s) about case conferencing? | <ul style="list-style-type: none"> The students indicated that they liked the self-directed nature, allowing initiative to decide what to discuss; They were able to seek advice from their peers about protocols for each clinic and clinical scenarios; They had instruments available to them for practice and to demonstrate on each other; They were able to vent frustrations, discuss concepts that were not clear and have reassurance by their peers; They were able to share interesting patient cases with each other which otherwise some students would have missed out on; and They were able to observe the extent of the clinicians' roles in different clinical contexts. |
| How can case conferencing be improved? | <ul style="list-style-type: none"> Some students suggested that a clinical educator could observe the last 5-10 minutes of the meeting to assist with unanswered questions that may have arisen; It was suggested that a mini tutorial could be conducted by clinicians once a week to demonstrate and affirm key clinical skills such as Goldmann tonometry, OCT and pachymetry. It was suggested to allow students access to the internet during case conferencing so answers could be sourced during the meeting. (This was immediately made possible in semester 2 as a direct result from early feedback.) Finally, it was suggested that interesting topics could be researched, discussed and recorded for use as a resource for future students. = |

RESULTS

The purpose of this paper was to report on the orthoptic student case conferencing program that we developed at the RVEEH and on students' perceptions of the program.

The evaluation was made utilising a survey consisting of a variety of 'forced choice' and 'open-ended' questions.

It was evident that students in the semester 2 block placement responded favourably in higher proportions or more often than those in the semester 1 sessional placement.

However, the vast majority of both groups of students felt that case conferencing provided a good opportunity to share with each other knowledge or information gained throughout the clinical placement.

During block placement periods, students have greater contact time in the clinical setting and better continuity which therefore results in more commitment to the process. This mode of placement allowed for better enforcement of concepts and skills too. The students have a greater opportunity to apply what they had discussed during the case conferencing and practice new skills in the clinical setting with their clinicians. This certainly reflected in the questions relating to problem solving and improvement of confidence. The availability of resources was better rated by the students in the block placement and this could be attributed to increased familiarity with the department as they spend greater time on site and the improvements made subsequent to initial feedback.

There were some challenges that students faced with case conferencing. A few felt that the meetings needed structure rather than to meander through topics. Others felt that the discussion topics were limited on some days and therefore did not have an interesting case or situation to discuss. This was not such an issue with block placement as the groups were larger and were therefore more likely to find topics for further discussion. As stated earlier, some students commented that different opinions between students could be confusing and so they needed good access to resources. Based on this feedback, a student login was organised which allowed for easy access to the internet, in addition to their library access.

Another improvement that was introduced included nominating a scribe for the case conferencing meetings. This encouraged the students to focus on topics and produce a coherent summary of what was discussed which could be used as a resource for in the future.

CONCLUSION

To conclude, students perceive case conferencing during orthoptic clinical placement at the RVEEH to be

valuable. Students undertaking the block placement mode seem to find it particularly beneficial, the key difference being the increased amount of contact time and engagement compared with students undertaking sessional placement.

REFERENCES

1. Pollock J, Georgievski Z, Devereux C. Students teaching students refraction. Is there a role for peer mentoring in the education of orthoptic students? In: Verlohr D, Georgievski, Z, Rydberg, A (Eds.) *Global Perspectives Converge Downunder*. Transactions of the Xth International Orthoptic Congress, Melbourne, Australia, 14-17 November, 2004; 355-358.
2. Pollock J, Georgievski Z. Peer mentoring in undergraduate clinical education of orthoptic students. In *Cornerstones*. Proceedings of the HERDSA Annual International Conference, Melbourne, Australia, 12-15 July 1999; 1-12.
3. Gilmour JA, Kopeikin A, Douche J. Student nurses as peer-mentors: collegiality in practice. *Nurse Education in Practice* 2007;7:36-43
4. Grant MA, Smith LN. The qualities of effective mentor from the student nurse's perspective: findings from a longitudinal qualitative study. *Journal of Advanced Nursing* 2000;32:1542-1549
5. Department of Health and Aged Care. *Primary care initiatives: enhanced primary care package*. Canberra: Commonwealth Department of Health and Aged Care, 1999.
6. Riley J, Smith C, Thick M. Case conferencing: an answer to improving generalist end of life care? Experience from Royal Marsden Hospital. *British Medical Journal*. 2008;28:337:a2290.
7. Pfefferle SG, Gittell JH, Hodgkin D, Ritter G. Pediatrician coordination of care for children with mental illnesses. *Medical Care* 2006;44:1085-91.
8. Meherbano KM, Jaywant M, Girish J, Kiran B, Sanjay K, Anjali J, Nilima L, Dilip S, Vikas Y, Ratnamala K, Walde MS. Solitary intraductal papilloma of the breast: a diagnostic dilemma and the role of conferencing between surgeons and cytologist. *Indian Journal of Pathology and Microbiology* 2006;49:582-585.
9. Harris M, Blakeman T. Enhanced primary care items: Their use in diabetes management. *Australian Family Physician* 2001;30:1134-1140
10. King MA, Roberts M. Multidisciplinary case conferencing reviews: improving outcomes for nursing home residents, carers, and health professionals. *Pharmacy World and Science* 2001;23:41-45.

A Case of Triplopia: A Case of Conversion Disorder?

Julie Fitzpatrick, BOrth, BSc, PostGradDipHlthResMth

Vision Australia, Geelong, Australia

ABSTRACT

The low vision rehabilitation orthoptist is involved in assisting clients to maximize independence despite functional vision loss, which may come in the form of reduced vision, field loss, reduced contrast sensitivity, or loss of binocular functions. In this paper, a case study of an elderly female who presented with monocular triplopia is discussed. The relationship between conversion disorder

and the patient's symptoms, the importance of tailoring management to the patient's functional requirements and the role of the orthoptist within a multidisciplinary team is discussed.

Keywords: Binocular functions, conversion disorder, monocular triplopia, binocular diplopia

INTRODUCTION

Triplopia is an uncommon presenting symptom with limited information on its incidence. A recent retrospective study showed that less than 1% of neurology patients complain of triplopia and that in most of these cases the symptoms were related to abnormal eye movements.¹ Triplopia can be caused by a number of ocular conditions, including eye movement disorders², lens irregularities²⁻⁶, retinal disorders², cerebral polyopia², impaired lateral inhibition of the visual cortex⁷, corneal irregularities², abnormal corneal steepening⁸ and small pupils.⁹

However, transient monocular triplopia has also been associated with Conversion Disorder.¹ Conversion disorder, previously known as "hysteria", is a condition where patients present with symptoms of motor and sensory dysfunction that are not explained by known physical disorders or pathophysiological mechanisms.^{10,11} Penman describes these symptoms as subconscious and out of the control of the patient experiencing this.¹² However, more recent studies suggest it could in fact be an early disruption to the nervous system rather than a psychological disorder.¹³

It has been reported that the total incidence of conversion disorder is between 15 to 22 per 100,000.¹⁴ Visual symptoms

in conversion disorder are not confined to triplopia, but also include rapid onset of vision impairment, sudden blindness, spiral or star-shaped loss of visual field, purple shadows, bilateral ptosis, hallucinations, and convergence spasm.^{1,15-21} This case study describes a patient with suspected conversion disorder presenting with transient monocular triplopia in addition to intermittent binocular diplopia due to a decompensating exophoria.

CASE REPORT

CC, a 67 year old female was referred to Vision Australia with a history of experiencing triplopia which could not be relieved with new glasses. CC was diagnosed with Multiple Sclerosis (MS) 12 years prior to the referral and was in a wheelchair. CC's general health conditions also included osteoarthritis, inflammatory heart disease and urinary incontinence. There were no obvious cognitive issues associated with the MS according to her general practitioner.

CC enjoyed crosswords and cross-stitch prior to the onset of the triplopia. She was very keen to keep up the cross-stitch, which requires use of binocular vision and depth perception. For CC this was the main functional issue that needed to be addressed. Due to the combination of health problems and being in a wheel-chair, sight-related activities had become increasingly important to her.

Correspondence: Julie Fitzpatrick
Vision Australia, 79 High St, Belmont Victoria 3216 Australia
Email: julie.fitzpatrick@visionaustralia.org

On the first orthoptic investigation cover test revealed an intermittent alternating exotropia at near estimated to be 5 degrees by corneal reflections. For distance she appeared orthophoric. Her convergence near point was reduced to 25 centimeters. Visual acuity was 6/18 in the right eye and 6/12 in the left eye with correction of +0.75/-2.50 x 100° and +0.50/-2.50 x 80° respectively. Near acuity with a +2.00 add was N5 with both eyes open. A subjective refraction was not performed at this visit as the patient was awaiting new glasses.

During reading, CC complained of vertical ghosting around words in each eye, which was subjectively better with both eyes open and disappeared beyond 25cm. When monocular, she preferred fixing with her left eye. CC did not describe symptoms of diplopia or triplopia during this examination. A focal light enabled print of poor contrast to be read and improved reading comfort. CC was advised to hold reading material slightly further away from her eyes (beyond 25cm), in order to relieve symptoms, whilst waiting for the new glasses. Given the importance of binocularity for the tasks she enjoyed, occlusion to relieve the monocular ghosting was not prescribed. One week later CC reported to be finding benefit in the use of the task lamp and occasionally closing one eye, but had not tried holding reading material further away to relieve the symptoms of ghosting.

CC was subsequently reviewed by her local ophthalmologist. The ophthalmologist confirmed there was no retinal, lens or corneal pathology. Interestingly, on this visit CC reported monocular triplopia and noted that it disappeared when the orthoptist used a multiple pinhole.

On the follow-up visit at Vision Australia, best corrected visual acuity with CC's new glasses was recorded as 6/6 N5 and 6/5 N6 in the right and left eyes respectively. CC complained of monocular triplopia which was intermittently present for near, but more marked without correction. In clinic, the symptoms presented in the right eye mainly as three distinct images, but occasionally as vertical ghosting. Due to the intermittent nature of the symptoms, CC was unable to reliably demonstrate the distance at which the triplopia disappeared. However, CC also reported that on occasion she experienced binocular diplopia when looking into the distance at, for example, the moon or television.

Cover testing showed no significant change, extraocular movements appeared full and her saccades did not appear to be delayed. During convergence the right eye failed at 15cm with diplopia. On this visit CC was given convergence exercises to address the convergence weakness exotropia which was thought to be causing binocular diplopia. Given that CC reported monocular triplopia at this visit, she was also encouraged to compare limited total occlusion when symptomatic, versus the occasional use of the multi-pinhole glasses.

On review 2 weeks later, CC reported that the most effective

approach to alleviate symptoms was part time total occlusion in the form of covering the right eye whilst watching television. She was learning to adapt to the monocular triple images, which she now described as monocular ghosting. One year later CC was primarily complaining of monocular ghosting which she was able to ignore or manage by occasionally covering one eye.

DISCUSSION

This case presents a patient complaining of intermittent monocular triplopia. On testing these symptoms alternated between monocular triplopia, binocular diplopia and ghosting. Keane recently reported that a significant number of individuals complaining of triplopia offer this description as an interpretation of binocular diplopia or oscillopsia.¹ It is possible that CC was also misinterpreting binocular diplopia related to her exotropia as triplopia. However, it must be noted that she also complained of this symptom in the distance where she demonstrated orthotropia.

Given that there was no ocular pathology, other than a convergence weakness exotropia, another possible cause for the triplopia is conversion disorder.¹⁴ Conversion disorder is also frequently associated with organic disease whether coexistent or antecedent.¹⁷ Furthermore it is cited as common in those diagnosed with MS, and has been reported to possibly be a manifestation of the damage to the CNS.¹⁴ Russo²² and Fadil et al²³ also reported that conversion disorder can itself lead to an incorrect diagnosis of general disorders, including MS, further confusing the clinical picture.

The complex nature and manifestations of conversion disorder, requires a comprehensive multidisciplinary approach to the assessment of a patient suspected of this condition. Newman²⁴ recommends co-operation between the neuro-ophthalmologist and psychiatrist. Similarly Langmann et al²⁵ recommend neuro-ophthalmic and orthoptic investigation together with observation of patient behavioral habits that may warrant referral to a psychiatrist. Smith, et al²⁶ also recommended involvement with a social worker as well as the abovementioned medical specialists for early intervention and differential diagnosis. Health professionals in the area of low vision are also often encouraged to communicate widely with other professionals, such as medical practitioners and psychiatrists to explore aspects of the patient's general and mental health which may impact on their reported visual symptoms. In the case of CC, it is clear that a wider multidisciplinary approach may have assisted in clarifying the aetiology of the patient's monocular triplopia.

This case also demonstrates the importance of understanding the patient's functional needs and the impact of management. CC enjoyed tasks that required binocular functions and

as such alternatives to occlusion, a widely-implemented management regime for symptoms of monocular diplopia, triplopia or ghosting, needed to be considered. In CC's case the use of lighting and good contrast, the training of convergence and the trial of a multiple pinhole viewer were all provided as options to encourage binocularity.

CONCLUSION

In conclusion, this case study demonstrates that an interaction between a patient's general health and visual symptoms when unexplained by ocular pathology needs to be explored. Furthermore, whilst it is not definitively known whether our patient was having a conversion reaction, it is clear that an integrated multidisciplinary approach to the management of patients with complex health conditions is important.

REFERENCES

1. Keane JR. Triplopia. 13 Patients from a Neurology Inpatient Service. *Archives of Neurology*. 2006;63:388-389.
2. Morris RJ. Double vision as a presenting symptom in an ophthalmic casualty department. *Eye*. 1991; 5: 124.
3. Kim A et. al. Wavefront analysis of eyes with cataracts in patients with monocular triplopia. *Ophthalmic and Physiological Optics*. 2006;26: 65-70.
4. Fujikado T. Development of a new evaluation system for visual function. *Acta Societatis Ophthalmologicae Japonicae*. 2004;108: 809-834.
5. Fujikado T, et. al. Wavefront analysis of an eye with monocular triplopia and nuclear cataract. *American Journal of Ophthalmology*. 2004;137:361-363.
6. Goss DA, et. al. A case of monocular triplopia of lenticular origin. *Optometry and Vision Science*. 1992; 69:486-488.
7. Hirayama K, et. al. Cerebral diplopia and triplopia- a proposal for responsible lesion and mechanism. *Clinical Neurology*. 1995;35:744-750.
8. Takei K. Is abnormal focal steepening of the cornea related to persistent monocular diplopia? *Journal of Refractive Surgery*. 2002;18:253-262.
9. Vaughan D and Asbury T. *General Ophthalmology*, 8th Ed. 1977. Lange Medical Publications. California.
10. Ness D. Physical therapy for conversion disorder: case series. *Journal of Neurologic Physical Therapy*. 2007;31:30-39.
11. Ballmaier M and Schmidt R. Conversion disorder revisited. *Functional Neurology*. 2005;20:105-113.
12. Penman MF. When neurological symptoms are not what they appear; the challenge of caring for patients with conversion disorders. *AXON*. 1993;15:19-22.
13. Parobek VM. Distinguishing conversion disorder from neurologic impairment. *Journal of Neuroscience Nursing*. 1997;29:128-134.
14. Stefansson JG, Messina JA, Meyerowitz S. Hysterical neurosis, conversion type: clinical and epidemiological considerations. *Acta Psychiatrica Scandinavica* 1976;53:119-38.
15. Griffiths RF and Ellis PM. Visual conversion disorder in a harbor pilot leading to sudden loss of control of a large vessel. *Aviation Space and Environmental Medicine*. 2007;78: 59-62.
16. Kluxen G. Psychogenic blindness caused by a command to kill. *English Abstract. Psychotherapie, Psychosomatik, Medizinische Psychologie*. 1995;45:131-133.
17. Ziegler DK and Schlemmer RB. Familial psychogenic blindness and headache: a case study. *Journal of Clinical Psychiatry*. 1994;55:114-117.
18. Lai HC et.al. Functional visual disturbance due to hysteria. *Chang Gung Medical Journal*. 2007;30:87-91.
19. Al-Sharbaty MM et.al. A case of bilateral ptosis with unsteady gait: suggestibility and culture in conversion disorder. *International Journal of Psychiatry in Medicine*. 2001;31:225-232.
20. Zain AM. True hallucination as conversion symptom: a case report. *Medical Journal of Malaysia*. 1990;45:74-77.
21. Suzuki A et.al. A case of convergence spasm in hysteria improved with a brief psychiatric assessment. *No to Shinkei-Brain and Nerve*. 2001;53;1141-1144.
22. Russo MB, et. al. Conversion disorder presenting as multiple sclerosis. *Military Medicine*. 1998;163:709-710.
23. Fadil H, et. al. Differential diagnosis of multiple sclerosis. *International Review of Neurobiology*. 2007;79:393-422.
24. Newman NJ. Neuro-ophthalmology and psychiatry. *General Hospital Psychiatry*. 1993;15:102-114.
25. Langmann A, et. al. Functional reduction of vision symptomatic of a conversion reaction in paediatric population. *English Abstract. Klinische Monatsblätter für Augenheilkunde*. 2001;218:677-681.
26. Smith HE et. al. Evaluation of neurologic deficit without apparent cause: the importance of a multidisciplinary approach. *Journal of Spinal Cord Medicine*. 2007;30:509-517.

A Case of Brown's Syndrome in Association With Goldenhar Syndrome

Kara Muecke¹

Linda Santamaria, MAppSc, DipAppSc(Orth)^{2,3}

¹Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia

²Southern Health Ophthalmology Unit, Melbourne, Australia

³Department of Surgery, Monash University, Melbourne, Australia

ABSTRACT

A case study of a young girl diagnosed at birth with Goldenhar syndrome is presented. Ocular features are described, including the unusual finding of Brown's

syndrome, suggesting a possible teratogenic link between the two conditions.

Keywords: Goldenhar syndrome, oculo-auriculo-vertebral dysplasia, Brown's syndrome

INTRODUCTION

Goldenhar syndrome was first described in 1952 by Swiss ophthalmologist Maurice Goldenhar.¹ It is a manifestation of the oculo-auriculo-vertebral spectrum (OAVS). Structural malformations found in Goldenhar syndrome are commonly unilateral and may involve the following; external and middle ears, eyes, face, skin, vertebrae and jaw.² Further associations may include congenital heart anomalies, cleft palate, dental anomalies, mental retardation and agenesis of corpus callosum.³⁻⁵

Gorlin has estimated the incidence of the syndrome at one in 5,600.⁶ It has a reported male to female ratio of between 2:1 and 3:2.^{1,7} The severity of the disease varies between individuals.

Ocular involvement differs from case to case. Findings can include microphthalmia, anophthalmia, upper eyelid coloboma, eyebrow coloboma, retinal coloboma, iris coloboma, ptosis, epibulbar dermoid, lipodermoid, nasolacrimal duct and canalicular obstruction, corneal anaesthesia, microcornea, peripapillary choroidal hyperpigmentation, macular hypoplasia, tortuous retinal vessels, optic nerve hypoplasia, tilted optic disc, cataract, dacryocystitis, cryptophthalmos, strabismus and Duane's syndrome.^{3, 5, 8}

CASE REPORT

Miss K was born prematurely at 35 weeks gestation, weighing 1,538gms, and was diagnosed with Goldenhar

syndrome. While the findings with Goldenhar syndrome can be numerous and varied, Miss K was born with the following manifestations; preauricular skin tags, mild left hemifacial microsomia and a right epibulbar dermoid. X-rays of spine and limbs, ultrasound of brain, chromosome testing and heart investigations were all shown to be normal, indicating a mild form of the syndrome. At two years of age, mild hearing loss in her right ear was also discovered.

Aged six and a half years old, Miss K had been regularly attending ophthalmology clinics since four months of age. Strabismus was recorded at four months of age, with a right essential infantile esotropia, which after some part-time occlusion was alternating. Surgery was undertaken at 19 months with bi-medial rectus recessions. The initial result post-operatively appeared straight, however, a year later a small consecutive left exotropia was apparent. At a later stage, a positive result to four diopter prism testing indicated a left microtropia with identity as no movement was seen on cover testing. It is uncommon for esotropia surgery to obtain perfect visual axis alignment and bifoveal fixation,⁹ and therefore it is likely that the microtropia was residual following previous esotropia surgery.

Miss K's ocular findings included the presentation of a right epibulbar dermoid. The epibulbar dermoid in Miss K's case was a dermolipoma located in the lateral canthus region of her right eye and was relatively inconspicuous. It was not impinging on the cornea or causing astigmatism and therefore no surgical intervention had been taken.

At three years of age a right Brown's syndrome was noted. No deviation was seen in primary position and surgical intervention was not required. Since then the Brown's syndrome has not altered or resolved.

Correspondence: **Kara Muecke**
Department of Clinical Vision Science, La Trobe University, VIC 3086, Australia
Email: kmuecke@students.latrobe.edu.au

At three and a half years of age, Miss K had an cycloplegic refraction of +2.50DS in each eye. At this point a difference in visual acuity was noticed with Right 3/6 and Left 3/9 (Kay pictures, single optotypes) and part-time patching was prescribed.

At six years of age her visual acuity was Right 3/3.8 and Left 3/4.8 (LogMAR). At this point patching was ceased due to poor compliance and given the presence of a microtropia, where it is generally accepted that levels of visual acuity greater than 6/12 or 6/9 are rarely achieved.¹⁰

Miss K showed classic ocular findings of Goldenhar syndrome of an epibulbar dermoid and esotropia. While Duane's syndrome has been reported in association with Goldenhar syndrome, Miss K presented with the unusual finding of a Brown's syndrome.

DISCUSSION

The aetiology of Goldenhar syndrome is poorly understood and mostly presents sporadically.^{7,11} Familial cases have also been reported, although the genetic basis for the disorder is not fully understood.¹¹ The aetiology in Miss K's case is unlikely to be of genetic origin as there was no family history of the condition. The cause in her case was attributed to an intra-uterine event during pregnancy. Goldenhar syndrome is thought to develop due to defects on the first and second branchial arch during foetal development.⁴

Epibulbar dermoids (dermolipomas and limbal dermoids) are commonly found with Goldenhar syndrome. Dermoids are histologically normal tissue (epidermal and connective tissue) in an abnormal location, usually present at birth and show little to no growth.⁵ The reported incidence of epibulbar dermoids with Goldenhar syndrome, or OAVS, varies between 32% and 78%.^{8,12-14} Limbal dermoids often contain hair and can involve deep corneal structures.⁵ Vision can be impaired if they encroach on the visual axis, cause astigmatism and/or amblyopia.¹¹

In Miss K's case, an epibulbar dermoid was found in the form of a dermolipoma. Dermolipomas are usually located in the conjunctiva near the lateral canthus and consist of epithelial, dermal and adipose tissues.⁵ Dermolipomas are yellowish or the colour of normal conjunctiva.⁸ They are usually well circumscribed and are rarely a functional or cosmetic problem.⁵ If removal is necessary a limited dissection should be performed to avoid symblepharon and scarring of the lateral rectus which can result in restrictive strabismus.^{5,6}

Strabismus is a common finding with Goldenhar syndrome,^{3,5,11,15} indicating a likely association in Miss K's case between the presentation of an essential infantile esotropia and Goldenhar syndrome.

Duane's retraction syndrome is also commonly reported in

the literature in relation to Goldenhar syndrome.^{8,15,16} Duane's retraction syndrome is thought to be due to branches of the oculomotor nerve innervating the lateral rectus muscle taking the place of absent or deficient abducent nerve fibres.¹⁷

The extraocular muscles innervated by the oculomotor nerve develop from the premandibular condensations, whereas the lateral rectus muscle and superior oblique muscles differentiate from the maxillomandibular mesoderm.¹⁸ The extraocular muscles become separate masses of mesoderm at four weeks.¹⁹ At around one month the extraocular muscles are innervated by the cranial motor nerves.⁵ Goldenhar syndrome manifests at a similar time in embryological development of approximately 30 to 45 days,^{6,14} which provides further basis for a link between Duane's syndrome and Goldenhar syndrome. Santamaria¹⁶ described an atypical vertical retraction syndrome in a child with Goldenhar syndrome, presenting a further ocular muscle innervation variant of Goldenhar syndrome.

To the best of the authors' knowledge, Brown's syndrome has not been reported previously in a case of Goldenhar syndrome. Brown's syndrome presents as an absence of elevation in adduction, with mechanical restriction on attempts to elevate the eye in adduction with forced duction testing.²⁰ While there are many possible aetiologies of Brown's syndrome, the exact aetiology in Miss K's case is unknown. Her Brown's syndrome was first noted at three years of age and is likely to have been congenital or may have developed in infancy. It is believed that the majority of cases of Brown's syndrome actually develop in infancy and that very few are congenital.¹⁷ There was no evidence of acquired trauma, juvenile rheumatoid arthritis, chronic sinusitis, systemic lupus erythematosus,⁵ or other aetiology supporting the diagnosis of an acquired Brown's syndrome.

One possible theory for the aetiology of a congenital Brown's syndrome in Miss K's case is the persistence of the embryonic trabecular connections between the superior oblique tendon and trochlea, thereby causing a restriction of movement.²¹⁻²³ The trochlea and superior oblique tendon are derived from mesenchymal tissue and are indistinguishable up to six weeks gestation (22mm embryo). At 26mm (seven to eight weeks) differential degeneration occurs between the trochlea and the tendon, being discernible as separate structures connected by thick trabeculae at 78mm (approximately 12 weeks). By 26 weeks, these septae generally degenerate, with only fine remnants remaining.^{21,22} This initial development of the tendon and trochlea occurs at the same time as the structures involved in Goldenhar syndrome.

A second possible aetiology is paradoxical innervation of the superior oblique similar to Duane's syndrome. Paradoxical innervation has been described with the co-contraction of the superior and inferior obliques on attempted elevation in adduction.²³⁻²⁶ Instead of the superior oblique muscle relaxing in elevation in adduction, there is

maximum innervation, restricting the globe from moving upwards.^{25, 26} On depression in adduction the innervation to the superior oblique is weaker, but still sufficient to move the eye in this direction.^{25,26} There have been three cases reported electromyographically showing this simultaneous paradoxical innervation,²⁴⁻²⁶ but this has not been confirmed by others.²³ However, in cases of paradoxical innervation one would expect a negative result on forced duction testing under anaesthesia, with von Noorden stating that this is never the case.²⁰ As forced duction and electromyography testing were not conducted in Miss K's case, this aetiological hypothesis can not be confirmed. The oculomotor nerve innervates the inferior oblique muscle at 31 days, and the superior oblique is innervated by the trochlear nerve at 33 days.¹⁹ The manifestation of Goldenhar syndrome is at a similar time period to the innervation of the extraocular muscles, providing a further possible basis for a relationship between the two syndromes.

CONCLUSION

While Miss K presented with a variety of ocular findings, only the epibulbar dermoid and infantile esotropia were common findings in Goldenhar syndrome. The presentation of Brown's syndrome has not been reported previously with Goldenhar syndrome. While we can not be certain Miss K's Brown's syndrome was congenital and therefore a true finding in Goldenhar syndrome, there are several possible causes of Brown's syndrome that could relate to the manifestation of Goldenhar syndrome, suggesting a common teratogenic effect.

ACKNOWLEDGEMENTS

We wish to thank Marianne Muecke for her help with German translations.

REFERENCES

- Kokavec R. Goldenhar syndrome with various clinical manifestations. *Cleft Palate Craniofac J*. 2006;43:628-634.
- Chaudhuri Z, Grover AK, Bageja S, et al. Morning glory anomaly with bilateral choroidal colobomas in a patient with Goldenhar's syndrome. *J Pediatr Ophthalmol Strabismus*. 2007;44:187-189.
- Ostler HB, Maibach HI, Hoke AW, Schwab IR. *Diseases of the Eye & Skin: A Color Atlas*. Philadelphia: Lippincott Williams & Wilkins, 2004.
- Setzer ES, Ruiz-Castaneda N, Severn C, et al. Etiologic heterogeneity in the oculoauriculovertebral syndrome. *J Pediatr*. 1981;98:88-90.
- Wright KW, Spiegel PH, editors. *Pediatric Ophthalmology and Strabismus*. 2nd Ed. New York: Springer, 2003.
- Gorlin RJ, Cohen MM, Hennekam RC. *Syndromes of the Head and Neck*. 4th Ed. New York: Oxford University Press, 2001.
- Bayraktar S, Bayraktar ST, Ataoglu E, et al. Goldenhar's syndrome associated with multiple congenital abnormalities. *J Trop Pediatr*. 2005;5:377-379.
- Baum JL, Feingold M. Ocular aspects of Goldenhar's syndrome. *Am J Ophthalmol*. 1973;75:250-257.
- Lang J. Management of microtropia. *Br J Ophthalmol*. 1974;58:281-292.
- Houston CA, Cleary M, Dutton GN, McFadzean RM. Clinical characteristics of microtropia - is microtropia a fixed phenomenon? *Br J Ophthalmol*. 1998;82:219-224.
- Taylor D, Hoyt CS, editors. *Pediatric Ophthalmology and Strabismus*. 3rd Ed. Edinburgh: Elsevier Saunders, 2005.
- Engiz O, Balci S, Unsal M, et al. 31 cases with oculoauriculovertebral dysplasia (Goldenhar syndrome): clinical, neuroradiologic, audiologic and cytogenetic findings. *Genet Couns*. 2007;18:277-288.
- Stromland K, Miller M, Sjogreen L, et al. Oculo-auriculo-vertebral spectrum: associated anomalies, functional deficits and possible developmental risk factors. *Am J Med Genet A*. 2007;143A:1317-1325.
- Tasse C, Bohringer S, Fischer S, et al. Oculo-auriculo-vertebral spectrum (OAVS): clinical evaluation and severity scoring of 53 patients and proposal for a new classification. *Eur J Med Genet*. 2005;48:397-411.
- Tillman O, Kaiser HJ, Killer HE. Pseudotumor cerebri in a patient with Goldenhar's and Duane's syndromes. *Ophthalmologica*. 2002;216:296-299.
- Santamaria L. An 'atypical' case of vertical retraction syndrome in association with Klippel-Feil syndrome. *Aust Orthopt J*. 1997/1998;33:77-80.
- Ansons AM, Davis H. *Diagnosis and Management of Ocular Motility Disorders*. 3rd Ed. Oxford: Blackwell Science Ltd, 2001.
- Bron AJ, Tripathi RC, Tripathi BJ, editors. *Wolff's Anatomy of the Eye and Orbit*. 8th Ed. London: Chapman & Hall Medical, 1997.
- Ozanic V, Jakobiec FA. Prenatal development of the eye and its adnexa. In: Jakobiec FA, editor. *Ocular Anatomy, Embryology and Teratology*. Philadelphia: Harper and Row, 1982.
- von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility: Theory and Management of Strabismus*. 6th Ed. St. Louis: Mosby, 2002.
- Sevel D. Brown's syndrome - a possible etiology explained embryologically. *J Pediatr Ophthalmol Strabismus*. 1981;18:26-31.
- Sevel D. The origins and insertions of the extraocular muscles: development, histologic features, and clinical significance. *Trans Am Ophthalmol Soc*. 1986;84:488-526.
- Wilson ME, Eustis HS, Parks MM. Brown's syndrome. *Surv Ophthalmol*. 1989;34:153-172.
- Ferig-Seiwerth F, Celic M. A contribution to the knowledge of the superior oblique tendon sheath syndrome (Brown's syndrome). In: Mein J, Bierlaagh JJ, Brummelkamp-Dons TE, editors. *Orthoptics - Proceedings of the Second International Orthoptic Congress, Amsterdam, 11-13 May, 1971*. Amsterdam: Excerpta Medica, 1972. p. 354-359.
- Papst W, Stein HJ. [Etiology of the superior oblique tendon sheath syndrome]. *Klin Monatsbl Augenheilkd*. 1969;154:506-518.
- Stein HJ, Papst W. [Electromyographic studies on the pathogenesis and therapy of the superior oblique tendon sheath syndrome (Brown's syndrome)]. *Ber Zusammenkunft Dtsch Ophthalmol Ges*. 1969;69:618-624.

Double Elevator Palsy with Congenital Esotropia: A Case Study

Marika Hensman, BOrth&OphthSc

Department of Clinical Vision Sciences, La Trobe University, Melbourne, Australia

ABSTRACT

This case study follows Master JT, a young boy who has congenital esotropia in addition to a right double elevator palsy. Ocular assessment of the patient is outlined as well as management and surgical treatment. The characteristics of congenital esotropia and double elevator palsy are

discussed in context with the child's presentation. The importance of performing a forced duction test to determine the classification of double elevator palsy and options or surgery is stressed. Rationale over surgical choices and likely prognosis are included.

Keywords: double elevator palsy, congenital esotropia, surgery, forced duction test, amblyopia

INTRODUCTION

Double elevator palsy (monocular elevation deficiency) has become a term used for any strabismus whereby there is reduced elevation in all horizontal orientations of the eye, not simply the paresis of the ipsilateral inferior oblique and superior rectus as it may imply^{1,2}. The incidence is unknown but it appears more prevalent in patients with congenital ptosis or pseudoptosis and Marcus Gunn Jaw-Winking syndrome². It is also not uncommon for a double elevator palsy to present in conjunction with a horizontal strabismus³. This case follows Master J.T. who initially presented with congenital esotropia and further testing revealed the patient also had congenital double elevator palsy.

CASE REPORT

Master J.T. presented to the New York Eye and Ear Infirmary at age 19 months for consideration of strabismus surgery following previous non-surgical management elsewhere. His mother first noticed a left esotropia when J.T. was aged 3 months and had been applying alternate patching. J.T. was carried to full term and weighed a healthy 3260 grams at birth. J.T. had no medical condition or allergies and was not on any medication. There is no family history of strabismus or any other ocular condition.

On examination, cycloplegic retinoscopy revealed a small amount of anisometropia R. +1.00 DS, L. -0.50 / -0.25 x 5°. On

cover testing there was an alternating fixation with a slight preference for the left eye, indicating no or insignificant amblyopia. At near J.T. measured 25-30Δ esotropia and 10Δ R hypotropia fixing left/and a L hypertropia 10-15Δ fixing right. J.T. adopted a chin up posture when fixating with his right eye, otherwise the head posture was a 15Δ right head tilt and right head turn. The chin-up posture put J.T. into down-gaze, whilst the head tilt and turn to the side of the vertically deviated eye displaced images down and to the side of the unaffected eye, which allowed him to maximise his field of binocular single vision when he was fixing with either eye⁴. On ocular movements there was a significant limitation of all forms of elevation of the right eye by -3 (past the midline) and his left inferior oblique showed +3 over-action. Horizontal movements were full and no signs of a ptosis were present. Alternate patching for 4 hours a day was prescribed to reduce the risk of developing post operative amblyopia and maintain equal vision owing to there being a constant strabismus present.

All of the results regarding the strabismus, ocular motility and head posture remained stable at 21 months so J.T. was scheduled for strabismus surgery to correct the horizontal and vertical deviations caused by the esotropia and double elevator palsy respectively. The result of a forced duction test of the right eye was negative indicating free passive movement of the globe and no mechanical restriction. Left inferior oblique anteriorization was performed to decrease the hyper element, as well as a bilateral medial recti recession (R. 4mm, L. 3.5mm) to correct the esotropia.

Post operatively JT achieved a 2Δ esotropia with 2Δ R hypotropia with a strong fixation preference for the left eye.

Correspondence: **Marika Hensman**
Department of Clinical Vision Sciences, La Trobe University, VIC 3086, Australia
E: marika_elizabeth@live.com

Elevation was not significantly improved as expected but the vertical deviation in primary position was decreased when fixing with his right eye. J.T. was then prescribed maintenance occlusion of one hour daily of the left eye.

DISCUSSION

Congenital esotropia presents within the first six months of life in the form of concomitant deviations of 30Δ or larger and there is generally a family history of strabismus^{1,3,4}. This case showed no family history of strabismus but measurements of J.T.'s angle and age of onset are consistent with this classification. J.T.'s fixation alternated freely the majority of time allowing equal vision and alternate suppression making significant amblyopia unlikely⁴. The alternate patching aids in maintaining this balance, and by avoiding amblyopia the likelihood of maintaining stable alignment of the eyes post-operatively was improved¹. J.T.'s left inferior oblique over-action is a common finding in congenital esotropia but may be influenced by his apparent double elevator palsy⁵. This could be due to the under-action of the left inferior oblique's contralateral synergist (right superior rectus) which requires increased innervations, leading to development of a consequent muscle sequelae according to Hering's law of equal innervations.

J.T.'s ocular movements were consistent with congenital double elevator palsy. There was very little movement past the midline during all attempts of elevation of his right eye which is the major characteristic. Additional features of double elevator palsy include a hypotropia in primary position increasing on up-gaze, ptosis or pseudoptosis and a chin up head posture with fusion in down-gaze or an extra deep lower lid fold^{1,3,6}. Commonly when fixing with the unaffected eye the affected eye appears hypotropic and then conversely when fixing with the affected eye there is a large hypertropia of the unaffected eye². J.T. is an excellent example of these findings and showed all signs with exception to ptosis.

Metz believed true paralysis of the elevator muscles was only involved in a quarter of cases⁷. Double elevator palsy is divided into three types. Type 1 is elevator paresis of both the superior rectus and inferior oblique of the deviated eye or the superior rectus alone. Type 2 is mechanical restriction of the inferior rectus and Type 3 is a combination of 1 and 2 due to a long standing paresis and consequent contracture of the inferior rectus^{1,2,5}. These categories can be distinguished by saccadic velocity, a forced duction test, presence of Bell's phenomenon and forced generation testing (FGT), which then allow for appropriate intervention to follow^{4,8}.

The need for treatment is dependent on the result of the "forced duction test", head posture and size of

the vertical deviation is in primary position¹. In cases of mechanical restriction (positive forced duction test) inferior rectus recession is recommended, and those with paresis (negative forced duction test) commonly undergo a Knapp surgical procedure whereby the horizontal recti are transposed towards the superior rectus of the affected eye³. Knapp's procedure can produce successful outcomes even when performed in conjunction with horizontal squint surgery⁹. J.T.'s forced duction test showed no mechanical restriction indicating pure parietic double elevator palsy most plausibly due to lesions in the oculomotor fascicle affecting the superior rectus and inferior oblique⁶. Therefore inferior rectus recession was not required and the surgeons chose not to perform a Knapp procedure.

The procedure of bimedial rectus recession used in J.T.'s case is most commonly practiced for congenital esotropia $25-45\Delta$ and was undertaken between age 6 months and 2 years which can be considered optimal⁴. Simultaneous surgery to weaken the overacting inferior oblique is favourable and is expected to improve the hypertropic element of J.T.'s deviation so both eyes appear more balanced (especially during up-gaze) and later possible presentation of dissociated vertical divergence would be minimal^{1,4,10}. Benefits of undergoing surgery now include greater potential for binocular single vision, improved interaction with his parents and a reduced mechanical component^{4,5}.

J.T.'s post operative outcome was favourable, but the strong fixation preference for the left eye puts him at high risk of developing strabismic amblyopia⁴. Therefore regular follow up will be required to prevent any post operative amblyopia development, monitor the ocular position and test for binocular functions. Cycloplegic refractions should be regularly performed as a recurrence of esotropia may result from an accommodative component⁴. Further surgeries may then become necessary¹.

CONCLUSION

It is not uncommon for congenital esotropia to co-exist with double elevator palsy. Clinical investigation should include a thorough patient history, observation of head posture, cover test and ocular motility exam to confirm the expected diagnoses. A forced duction test is then essential for the further classification of the type of double elevator palsy as it detects whether mechanical restrictions are involved or if there is purely muscle weakness. This is necessary for choosing the appropriate surgical intervention to treat the vertical component of the strabismus when it is problematic. Surgery was performed in the optimal timeframe and provided good outcomes in J.T.'s case.

REFERENCES

1. American Academy of Ophthalmology. Pediatric Ophthalmology and Strabismus. USA: American Academy of Ophthalmology, 2006.
2. Gallin, P. Pediatric Ophthalmology: A Clinical Guide. New York: Thieme, 2000.
3. Dornic, D. Ophthalmic Pocket Companion. 6th Ed. USA: Butterworth-Heinemann, 2002.
4. Ansons AM, Davis H. Diagnosis and Management of Ocular Motility Disorders. 3rd Ed. UK: Blackwell Science, 2006.
5. Pratt-Johnson J, Tillson G. Management of Strabismus and Amblyopia: A Practical Guide. 2nd Ed. New York: Thieme, 2001.
6. Wong A. Eye Movement Disorders. New York: Oxford University Press, 2008.
7. Metz HS. Double Elevator Palsy. Arch Ophthalmol 1979;97:901-903
8. Bagheri A, Sahebghalam R, Abrishami M. Double Elevator Palsy, Subtypes and Outcomes of Surgery. J Ophthalmic Vis Res [serial online]. 2008; 3: 108-113.
9. Lee JP, Collin JR, Timms C. Elevating the Hypotropic Globe. Br. J. Ophthalmol. 1986; 70: 26-32.
10. Burke JP, Ruben JB, Scott WE. Vertical Transposition of the Horizontal Recti (Knapp Procedure) for the Treatment of Double Elevator Palsy: Effectiveness and Long-Term Stability. Br. J. Ophthalmol [serial online]. 1992; 76: 734-737.

BINOCULAR VISION & Strabismus Quarterly
the First and Original International Scientific Periodical devoted to
Strabismus and Amblyopia, on-line in MEDLINE

YES!

Please enter my **PERSONAL** subscription
 (all subscriptions, USA & International) for

3 years 2006 - 2008 @ \$ 68/y = \$ 204 SAVE! \$48 = 30%/yr interest
 2 years 2006 - 2007 @ \$ 78/yr = \$ 156 SAVE! \$12 = 15%/yr interest
 1 years 2006 @ \$ 84/yr = \$ 84 No Increase in 10 years. 2007 + ?

Please ADD for AIRMAIL: USA: \$15 /yr =\$ INT'L: \$30 /yr =\$

| PERSONAL | NOT BV&SQ | YES BV&SQ | S&H |
|---|------------------------------|------------------------------|----------------------------|
| BOOKS: Please send me 1 copy of | subscriber | subscriber | contUS |
| von Noorden: History Strabismology | <input type="radio"/> \$ 179 | <input type="radio"/> \$ 159 | <input type="radio"/> \$ 5 |
| Helveston: Surg Mgmt Strsbismus 5 | <input type="radio"/> \$ 179 | <input type="radio"/> \$ 139 | <input type="radio"/> \$ 5 |
| Kushner: Collection BV Gr Rounds | <input type="radio"/> \$ 149 | <input type="radio"/> \$ 89 | <input type="radio"/> \$ 5 |
| SUBSCRIBER SAVES (TOTAL FOR ALL 3) | | (\$ +120) | |

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

THE PATRICIA LANCE LECTURE

| | | |
|------|-------------------|---|
| 1988 | Elaine Cornell | (Inaugural) |
| 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 | |
| 2008 | Stephen Vale | A Vision For Orthoptics: An outsider's perspective |

THE EMMIE RUSSELL PRIZE

| | | |
|--------|--------------------|---|
| 1957 | Margaret Kirkland | Aspects of vertical deviation |
| 1959 | Marion Carroll | Monocular stimulation in the treatment of amblyopia exanosis |
| 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 | Quantitative 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 |
| 2007 | Thong Le | What is the difference between the different types of divergence excess intermittent exotropia |
| 2008 | Amanda French | Does the wearing of glasses affect the pattern of activities of children with hyperopic refractive errors? |

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 |
| 2007 | Jodie Leone | Prevelance of heterophoria in Australian school children |
| 2008 | Jodie Leone | Can visual activity screen for clinically significant refractive errors in teenagers? |

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 |
| 2008 | Julie Barbour |

Presidents of The Orthoptic Association of Australia Inc. and Editors of The Australian Orthoptic Journal

PRESIDENTS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA INC

| | | | | | |
|---------|------------------|---------|------------------|---------|---------------------|
| 1945-7 | Emmie Russell | 1964-5 | Lucy Retalic | 1981-82 | Marion River |
| 1947-8 | Lucy Willoughby | 1965-6 | Beverly Balfour | 1982-3 | J Stewart |
| 1948-9 | Diana Mann | 1966-7 | Helen Hawkeswood | 1983-5 | Neryla Jolly |
| 1949-50 | E D'Ombrian | 1967-8 | Patricia Dunlop | 1985-6 | Geraldine McConaghy |
| 1950-1 | Emmie Russell | 1968-9 | Diana Craig | 1986-7 | Alison Terrell |
| 1951-2 | R Gluckman | 1969-70 | Jess Kirby | 1987-9 | Margaret Doyle |
| 1952-4 | Patricia Lance | 1970-1 | Neryla Heard | 1989-91 | Leonie Collins |
| 1954-5 | Diana Mann | 1971-2 | Jill Taylor | 1991-3 | Anne Fitzgerald |
| 1955-6 | Jess Kirby | 1972-3 | Patricia Lance | 1993-5 | Barbara Walsh |
| 1956-7 | Mary Carter | 1973-4 | Jill Taylor | 1995-7 | Jan Wulff |
| 1957-8 | Lucille Retalic | 1974-5 | Patricia Lance | 1997-00 | Kerry Fitzmaurice |
| 1958-9 | Mary Peoples | 1975-6 | Megan Lewis | 2000-2 | Kerry Martin |
| 1959-60 | Patricia Lance | 1976-7 | Vivienne Gordon | 2002-4 | Val Tosswill |
| 1960-1 | Helen Hawkeswood | 1977-8 | Helen Hawkeswood | 2004-6 | Julie Barbour |
| 1961-2 | Jess Kirby | 1978-9 | Patricia Dunlop | 2006-8 | Heather Pettigrew |
| 1962-3 | Patricia Lance | 1979-80 | Mary Carter | 2008- | Zoran Georgievski |
| 1963-4 | Leonie Collins | 1980-1 | Karen Edwards | | |

EDITORS OF THE AUSTRALIAN ORTHOPTIC JOURNAL

| | | | | | |
|----------------|---------------------------------|----------------|----------------|----------------|-------------------------------------|
| Vol 8 1966 | Barbara Lewin & Ann Metcalfe | Vol 20 1983 | Margaret Doyle | Vol 34 1999 | Julie Green |
| Vol 9 1969 | Barbara Dennison & Neryla Heard | Vol 21 1984 | Margaret Doyle | Vol 35 2000 | Neryla Jolly & Nathan Moss |
| Vol 10 1970 | Neryla Heard | Vol 22 1985 | Margaret Doyle | Vol 36 2001-02 | Neryla Jolly & Kathryn Thompson |
| Vol 11 1971 | Neryla Heard & Helen Hawkeswood | Vol 23 1986 | Elaine Cornell | Vol 37 2003 | Neryla Jolly & Kathryn Thompson |
| Vol 12 1972 | Helen Hawkeswood | Vol 24 1987 | Elaine Cornell | Vol 38 2004-05 | Neryla Jolly & Kathryn Thompson |
| Vol 13 1973-74 | Diana Craig | Vol 25 1989 | Elaine Cornell | Vol 39 2007 | Zoran Georgievski & Connie Koklanis |
| Vol 14 1975 | Diana Craig | Vol 26 1990 | Elanie Cornell | Vol 40 2008 | Connie Koklanis & Zoran Georgievski |
| Vol 15 1977 | Diana Craig | Vol 27 1991 | Julia Kelly | | |
| Vol 16 1978 | Diana Craig | Vol 28 1992 | Julia Kelly | | |
| Vol 17 1979-80 | Diana Craig | Vol 29 1993 | Julia Kelly | | |
| Vol 18 1980-81 | Diana Craig | Vol 30 1994 | Alison Pitt | | |
| Vol 19 1982 | Diana Craig | Vol 31 1995 | Julie Green | | |
| | | Vol 32 1996 | Julie Green | | |
| | | Vol 33 1997-98 | Julie Green | | |

OAA Office Bearers, State Branches & University Training Programs

ORTHOPTIC ASSOCIATION OF AUSTRALIA

OAA OFFICE BEARERS

President: Zoran Georgievski
President Elect: Zoran Georgievski
Vice President: Heather Pettigrew
Treasurer: Julie Barbour
Secretary: Mara Giribaldi
Public Officer: Susan Sutton

STATE REPRESENTATIVES

Australian Capital Territory: Wendy Holland, Corinne Brignall
New South Wales: Mara Giribaldi, Lindley Leonard, Ann Macfarlane
Queensland: Colleen Wilkinson, Terri Leverty
South Australia: Hayley Neate
Tasmania: Julie Barbour
Victoria: Connie Koklanis, Karen Mill, Christopher Drowley
Western Australia: Lisa Biggs

STATE BRANCHES

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

Queensland:
 Contact: Colleen Wilkinson

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

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

Western Australia:
 Contact: Lisa Biggs

UNIVERSITY TRAINING PROGRAMS

MELBOURNE

Department of Clinical Vision Sciences
 Faculty of Health Sciences
 La Trobe University
 Bundoora, VIC 3086
 T: 03 9479 2570
 F: 03 9479 3692
www.latrobe.edu.au/orthoptics

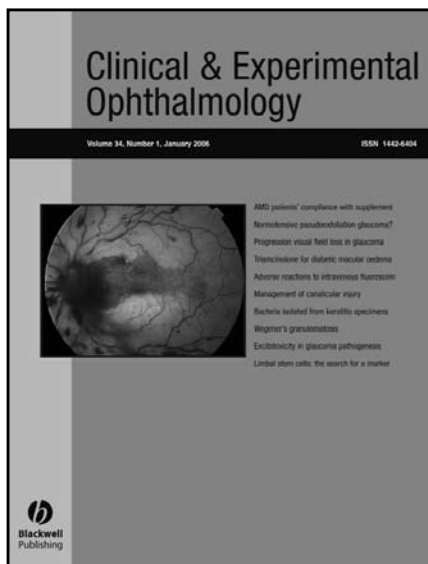
SYDNEY

Discipline of Orthoptics
 Faculty of Health Sciences
 The University of Sydney
 East St, Lidcombe NSW 2141
 T: 02 9351 9250
 F: 02 9351 9359
www.fhs.usyd.edu.au/orthoptics

Access online with Blackwell Synergy

Clinical & Experimental Ophthalmology

Published on behalf of the Royal Australian and New Zealand College of Ophthalmologists



Edited by: Charles N.J. McGhee
Print ISSN: 1442-6404
Online ISSN: 1442-9071
Frequency: 9 issues per year
ISI Journal Citation Reports® Ranking:
2005: 24/44 (Ophthalmology)
Impact Factor: 1.193

Blackwell Synergy is the online journals service from Blackwell Publishing that will improve the quality of your research time. It enables readers to search for relevant articles, read abstracts for free, print the full text of subscribed to articles, download citations, and make connections to other relevant research through reference linking.

Registering with *Blackwell Synergy* is free.

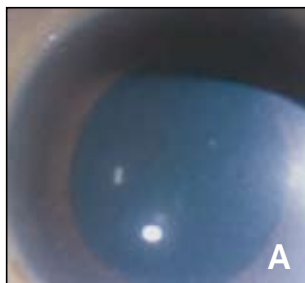
Go to: **www.blackwell-synergy.com** and register today!

While you're there, sign up for free emailed table-of-contents alert to over 800 journals.

**Blackwell
Synergy** 

For further information, and to subscribe to the Journal, please visit **www.blackwellpublishing.com/ceo**

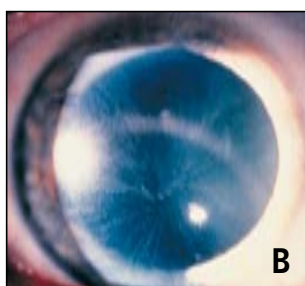
If you see a patient with...



Corneal Clouding¹

it could be Mucopolysaccharidosis I (MPS I)

Corneal clouding develops in virtually all patients with MPS I and can lead to significant visual disability.



Corneal Opacities²

it could be Fabry disease

Distinctive corneal opacity (corneal verticillata or vortex keratopathy) in Fabry disease. Note the whorl-like corneal rays emanating from a single vertex like the spokes of a wheel.

MPS I and Fabry disease are progressive, potentially life threatening disorders.^{1,2}

Eyecare professionals have the opportunity to identify these patients early.

If you suspect one of your patients may have MPS I or Fabry Disease contact Genzyme Australasia for further information.

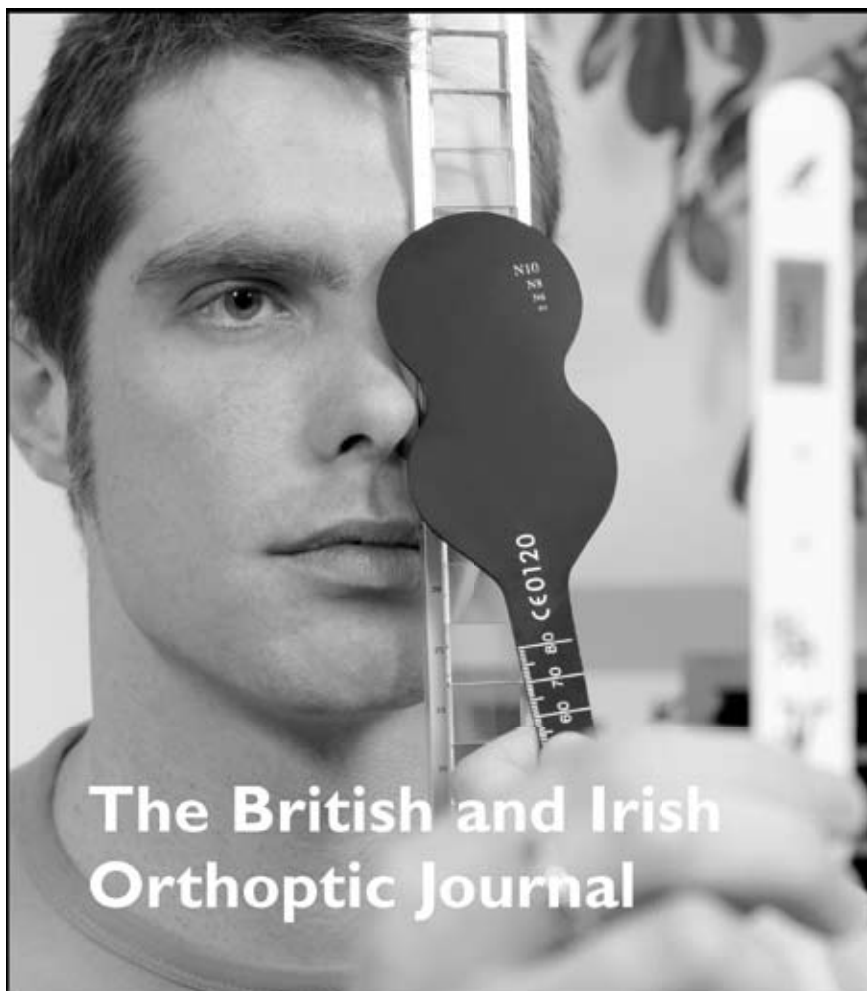
genzyme

Genzyme Australasia Pty. Ltd. ABN 24083420526
Level 1, Building C, 12-24 Talavera Rd, North Ryde, NSW 2113
Tel: +61 2 9978 3900 Fax: +61 2 9889 3900.

References: 1. Ashworth JL. *et al.* Survey of ophthalmology. Vol 51; No 1: 2006. 2. Desnick RJ. *et al.* Annals of Int Med. Vol 138; No 4: 2003.

Photo A. courtesy of S. Pitz, M.D.
Photo B. courtesy of RJ Desnick PhD, MD.

FZM 09-017



**The British and Irish
Orthoptic Journal**

The official annual publication of the British and Irish Orthoptic Society, the Journal contains papers covering orthoptics, ocular motility, amblyopia, binocular vision, strabismus, related paediatric ophthalmology and neuro-ophthalmology.

The editorial board comprises leading British and Irish orthoptists and ophthalmologists.

Original articles for publication may be submitted to the Editor:

Dr Sarah Shea PhD DBO(D), Orthoptic Clinic, North West Wales NHS Trust, Ysbyty Gwynedd, Bangor, North Wales LL57 2PW United Kingdom


British and Irish Orthoptic Society

ORTHOPTIC ASSOCIATION OF AUSTRALIA

66TH ANNUAL
SCIENTIFIC CONFERENCE

15-18 NOVEMBER 2009



BRISBANE
BRISBANE

www.orthoptics.org.au