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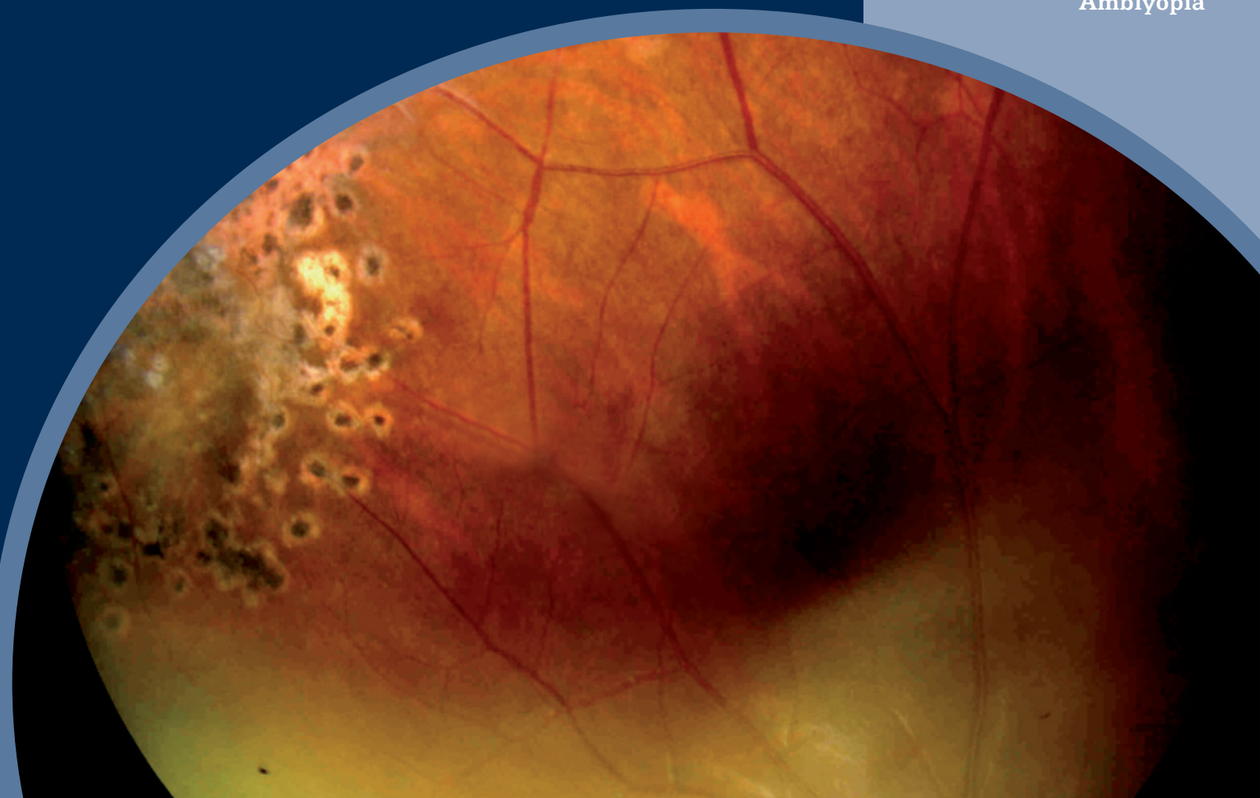

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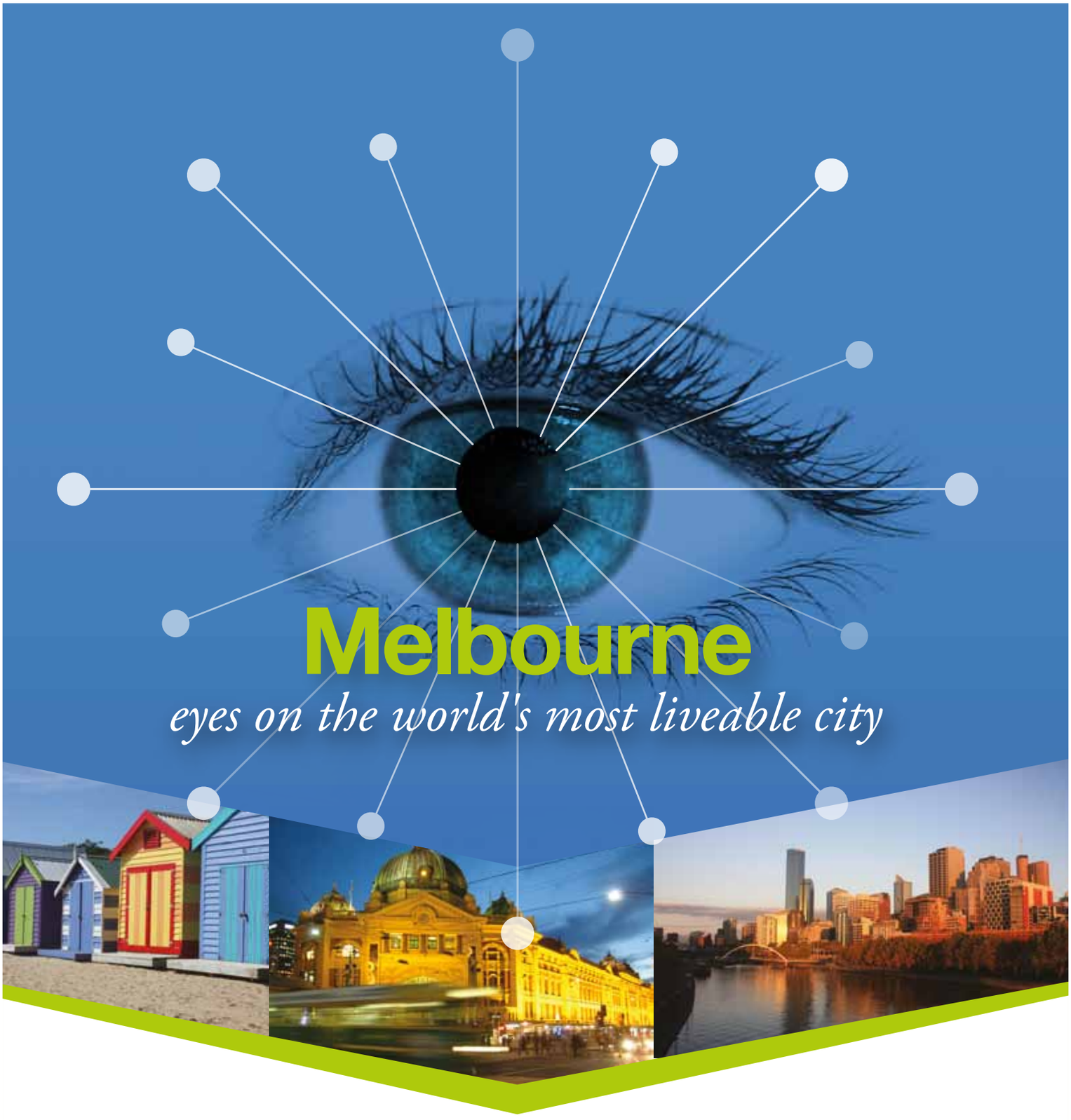

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TRIBUTE

Zoran Georgievski

Associate Professor
Department of Clinical Vision Sciences, La Trobe University
Department & Clinical School of Orthoptics, Royal Victorian Eye & Ear Hospital

3 September 1970 – 7 April 2011

There are no words to describe Zoran's contribution to orthoptics and the significant impact he has made on our profession. His influence has been at both a local and international level, and he was undoubtedly one of the most influential orthoptists of our time.

Zoran had an inquisitive, brilliant mind that never settled. He was pragmatic, could always see the shades of grey and think outside the square. This all too often translated into great, innovative ideas. He was also wildly ambitious but more so for his profession than for himself. He was keen to see the advancement of the discipline of orthoptics, so we as allied health professionals could better contribute to excellence in eye health care for our patients. He perceived the development of the Clinical School at the Royal Victorian Eye & Ear Hospital and his conjoint appointment between La Trobe University and the hospital as a vehicle to achieve this.

He was considered an explemplary scholar who had mastered the art of strabismus. He passionately imparted this immense knowledge to students and colleagues and his tips and pearls will be treasured by many. He had a gift of effortlessly explaining complex ideas and theories; a truly imaginative and engaging teacher. He served as an inspirational role model and mentored many towards self-determination through encouragement and challenge.

From an education and academic perspective, his contribution is paralleled by few. Early in his career he was recognised as a talented orthoptist joining the Faculty of Health Sciences at La Trobe University soon after completing his studies. Since then he has consistently worked towards furthering orthoptics through education and research. He also dedicated over a decade to the clinical education of students, developing an outstanding program providing students with broad experiences and the opportunity to travel and study overseas. To him we owe much of the shape of our world class orthoptic program at La Trobe University.

Beyond traditional orthoptics he was a pioneer in the development of models of service delivery aimed at seeing orthoptists emerge as allied health practitioners meeting 21st century challenges. He was one of the first, if not the first, to develop diabetic retinopathy screening clinics, orthoptic triage clinics and orthoptist-led spoke clinics.

Zoran was a visionary leader. Rarely has orthoptics seen someone so progressive and far-sighted. He was a gifted strategist and lobbied federal and state governments on various issues to provide opportunities for the profession. As only one example, he was the first in Australia to advocate for and create change in legislation to allow orthoptists to prescribe glasses independently. He recognised our pivotal role in eye health care and understood the changes required in the legislative, academic and clinical space. In his own words he believed 'if you're a professional, you're responsible to yourself to be part of a collective of people who can steer where the profession is taking you'. He was certainly that, in fact he led this collective of people.

In one capacity or another he was involved in our national and international association, leaving a mark of his ideas and work in all areas. Most recently he is responsible for our association's name change to Orthoptics Australia, a change he wanted to reflect the dynamic movement forward of orthoptics and a way to embrace change and improvement.

His standing resulted in an immense level of deference to his professional opinion. His finger was on the pulse of all things. So many colleagues sought Zoran's advice and perspective and despite all he was involved in, he was always accessible. I often admired his insight not only in all things orthoptics but his unique insight into human nature and his ability to see through things and cut to the basic reality. Indeed he will also be remembered for his direct and poignant honesty. On many occasions he did not mince his words. He truly lit up our association council meetings and any discussion he was involved in with fervent, witty debate.

Zoran was always inclusive, had a positive outlook and brought with him an element of fun. He had a generous and caring disposition. He demonstrated a great capacity for

love and friendship; a master in connecting with individuals and creating networks between people. His ability to touch hearts and minds was profound.

Zoran's intelligence, uniqueness of mind, warmth, humour, passion, and determination set him apart. He forged a formidable reputation and legacy; an infinite imprint in the orthoptic community that will live on.

Dr Connie Koklanis
Department of Clinical Vision Sciences
La Trobe University

Clinical Management of Coats Disease: A Case Study

Christopher R Drowley, BOrth&OphthSc
Justin O'Day, FRANZCO AM

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ABSTRACT

Coats disease, is a rare unilateral retinal vascular disease of unknown aetiology though there may be a genetic predisposition to the disorder. If left untreated, severe and permanent vision loss can occur due to total exudative retinal detachment. Early intervention and close monitoring remains the most effective way to prevent potential vision loss and the progression to a blind and painful eye. This report describes the case of a 15 year-old healthy male who presented with a one-month history of unilateral blurred central vision. Fundus examination revealed a peripheral retinal vascular lesion which resulted in lipid deposits in the macular region. The patient was treated with argon laser panretinal photocoagulation and monitored over an 18-month period. He demonstrated a slow though significant resolution of the maculopathy which correlated with an improvement in visual acuity. This case highlights that early presentation followed with an appropriate management regime can result in a successful visual outcome.

Keywords: Coats disease, retinal telangiectasia, retinal detachment

INTRODUCTION

Coats disease, also known as retinal telangiectasia, is a rare unilateral retinal vascular disease[1] first identified by George Coats in 1908.[2] Though generally of unknown aetiology, some evidence has suggested a mutation of the Norrie disease gene could be a possible cause of Coats disease.[3] The exact incidence and prevalence of Coats disease is unknown, however a prospective population-based study in the United Kingdom has estimated an incidence of 0.09 per 100,000 of the population.[4] Those typically affected are males under the age of 20 years,[5] however the peak incidence of the disease occurs between six and eight years of age.[6] Coats disease is considered to be a severe form of retinal telangiectasia, a congenital retinal vascular anomaly.[6] Other types of retinal telangiectasia include idiopathic juxtafoveal telangiectasia, parafoveal telangiectasia and Leber's miliary aneurysms.[7]

The presenting signs of Coats disease, the stages and age of presentation contribute to its diagnosis as well as considerations for differential diagnosis. Children, who often present in the advanced stages of Coats disease can present with leukocoria, strabismus and intraocular mass lesions.[2] Differential diagnosis includes retinoblastoma, retinopathy of prematurity, ocular toxocariasis, choroidal hemangioma, and familial exudative vitreoretinopathy.[3] In older children and adults Coats disease must be differentiated from diabetic retinopathy, hypertensive retinopathy, retinal vein occlusions, vasculitis, melanoma, choroidal hemangiomas and juxtafoveal telangiectasia.[6] In each of these eye diseases patients present with retinal vascular anomalies with associated visual loss or disturbances.

The first sign of Coats disease is retinal telangiectasia.[8] When quiet, the telangiectasia does not cause any reduction in visual acuity and thus the patient remains asymptomatic. However, symptoms such as reduction in visual acuity occur when the retinal appearance alters and the vessels become dilated and tortuous. Aneurysms may form, giving rise to subsequent haemorrhages and exudates. Initially, the patient is usually unaware of any visual disturbance due to the often peripheral retinal location of the telangiectasia.

Recommendations regarding the management of Coats disease are well described in the literature. The primary goal of treatment is to remove areas of active retinal telangiectasia and allow resolution of lipid deposits by arresting leakage of exudates from retinal vessels.[9] This can be achieved through argon laser pan retinal photocoagulation (PRP) or cryotherapy.[6] In the presence of exudation with no retinal detachment, argon laser PRP is appropriate.[9] In those severe cases which present with a shallow detachment,

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cryotherapy is more effective in sealing retinal vessels and repairing detachments.[6] In the presence of extensive retinal detachment, vitreo-retinal surgical techniques are utilised to reattach the retina.[9] At end-stage, enucleation is required for a sore, blind and painful eye. This might be indicated if the patient presents late in the progression of the disease.[6]

Visual outcomes vary considerably between patients with Coats disease and depend largely on the nature or the stage of the retinal change and thus the time at which the patient is seen. Table 1 summarises the various stages of Coats disease and management options at each of these stages.

Table 1. Stages and classifications of Coats disease (adapted from Shields et al[9])

Stage		Criteria	Management Options
Stage 1		- retinal telangiectasia	- observation
Stage 2	2A	- retinal telangiectasia - extrafoveal exudation	- observation (2A) - photocoagulation - cryotherapy
	2B	- retinal telangiectasia - foveal exudation	
Stage 3	3Ai	- extrafoveal execdative retinal detachment	- photocoagulation - cryotherapy - retinal detachment repair - enucleation (to relieve ocular pain associated with glaucoma)
	3Aii	- exudative foveal retinal detachment	
	3B	- total exudative retinal detachment	
Stage 4		- total retinal detachment - glaucoma	
Stage 5		- end stage	

CASE REPORT

A 15 year-old male presented complaining of blurred central vision in the right eye for one month. His general health was good and no past ocular history was reported. No family history of eye disease was reported. Snellen visual acuity was R 3/36 (no improvement with pinhole) and L 6/4. Intraocular pressures were on the higher side of normal, measuring R 21 mmHg and L 19 mmHg. A right relative afferent pupil defect was noted and fundus examination revealed a macular star exudate at the posterior pole of the right eye, with slight lipid deposition in the retinal periphery (Figure 1A). A retinal vascular anomaly was also found in the upper temporal quadrant of the right eye fundus which was identified with fundus flourescein angiography (Figure 1C). No retinal abnormality was detected in the left eye (Figure 1B).

At this visit, the patient was treated with argon laser PRP over the area of retinal telangiectasia. Over an 18-month period the patient was continually reviewed and required a total of four PRP treatments to his right eye. A slow and steady improvement in his visual acuity throughout this time was evident as the macular exudate resolved (Figure 2).

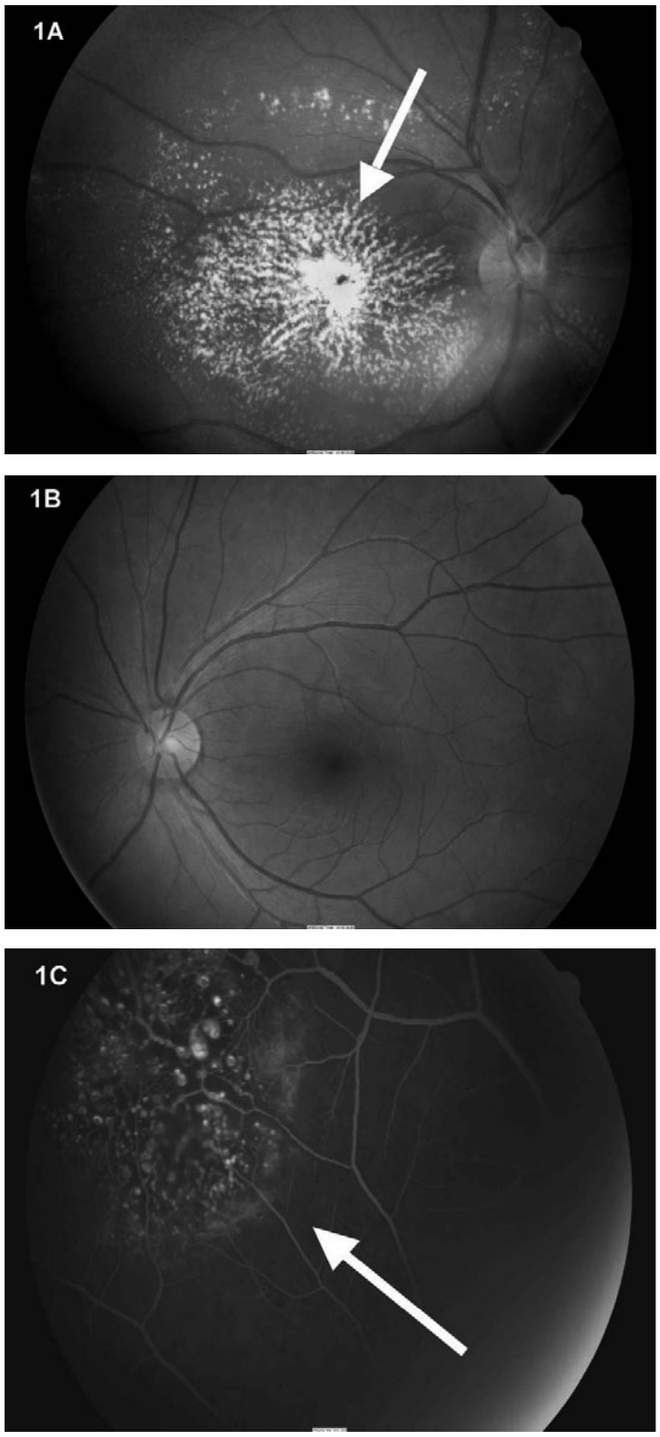

Figure 1. Fundus photographs show (A) a right eye macular star exudate; and (B) no abnormality in the left eye. Flourescein angiography (C) clearly shows an area of retinal vascular anomaly in the upper temporal quadrant of right eye.

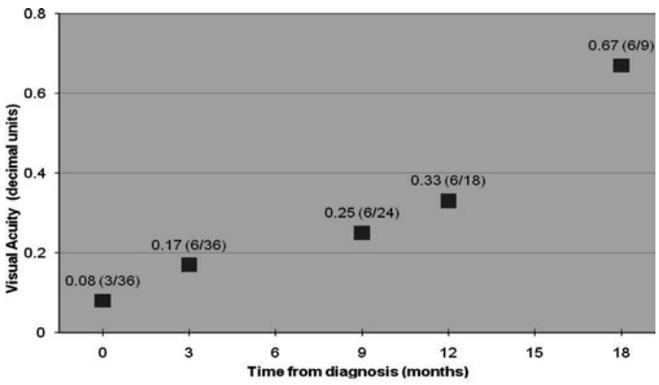

Figure 2. Improvement in visual acuity over time expressed in decimal units (Snellen equivalents shown).

At his last visit, visual acuity in the right eye had improved to 6/9 which was commensurate with an improvement in retinal appearance as most of the exudate at the posterior pole had resolved (Figure 3).

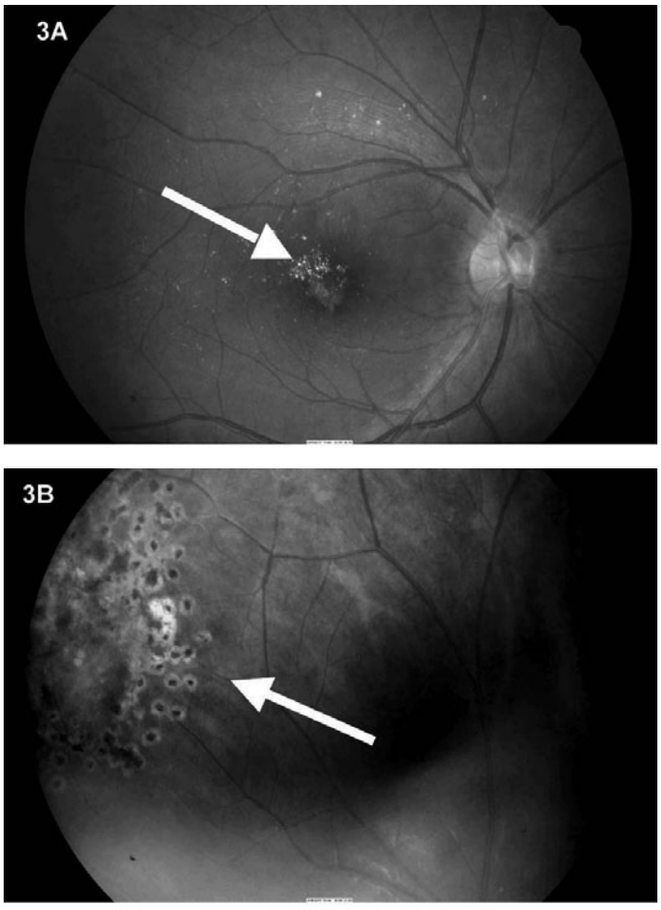

Figure 3. 18 months following initial presentation, (A) shows the remaining exudate at the posterior pole and (B) shows the area of retinal telangiectasia following multiple laser treatments.

DISCUSSION

This male patient presented before the age of 20 years with a complaint of unilateral vision loss. There was no systemic disease to indicate a cause for his visual disturbance, and fundus examination findings were typical of progressive Coats disease.

The vision loss described by the patient was due to the large area of exudate deposited at the posterior pole. The exudate had migrated from an area of retinal telangiectasia that had formed aneurysms with subsequent haemorrhaging and lipid leakage. It is most likely that the area of congenital retinal telangiectasia had remained quiet throughout early childhood, giving rise to a later presentation than the reported peak incidence of 6 to 8 years.[6]

The leakage of lipid in the posterior pole most often causes initial visual disturbances in Coats disease, particularly when the fovea is affected, as was noted in this case. The arrangement of nerve fibres in the fovea, which run parallel to the retinal surface, allow exudates to migrate there and remain in the outer plexiform layer.[2] This parallel radial distribution of exudate can be likened to a star – hence the term macular star, which can also be seen in other retinal disorders such as hypertensive and diabetic retinopathies.[2] This can occur when there is chronic retinal oedema and deposition of hard exudates around the fovea.[2] The macular region is slow to absorb the leaking exudates and the build-up of this lipid exudate can lead to an exudative retinal detachment causing severe and permanent vision loss.[5]

The patient in this case study presented at stage 2B (see Table 1). His peripheral retina showed signs of retinal telangiectasia and marked exudation at the fovea. This early presentation allowed treatment to commence immediately and arrest leakage from the area of retinal telangectasia via argon laser PRP. Table 1 shows how those presenting with stages 1 to 3 have the best visual prognosis due to viable treatments of the disease.[7] If left longer, the progression of the disease leads to a total exudative detachment with permanent vision loss and further complications of potential glaucoma.[10]

CONCLUSION

This case highlights an appropriate management regime applicable to an early-presenting case of Coats disease. In such instances, a successful visual outcome can result. This case serves as an important reminder that a dilated fundus examination in all patients, particularly children, is essential in order to adequately detect retinal abnormalities. This is of particular importance in patients with retinal telangiectasia, although initially asymptomatic, if left untreated can lead to severe visual disturbances.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the assistance of Dr Suzane Vassallo in providing commentary on this manuscript.

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'Does Size Matter?' - An Investigation of Anisometropia, Aniseikonia and Anisophoria

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ABSTRACT

Aim: To examine how acquired anisometropia commonly gives rise to symptoms of diplopia in patients and to differentiate the cause of these symptoms in terms of aniseikonia and anisophoria.

Method: Twenty-one patients with acquired anisometropia >1.00 D and astigmatism <1.00 D were examined. Symptoms of diplopia and cover test in primary position at 6 m and 1/3 m were recorded with the patient wearing spherical equivalent correction and then repeated through the reading position of the lenses, 12 mm below the optical centre. Stereopsis was assessed using the TNO test, and aniseikonia was measured using the Awaya New Aniseikonia Test.

Results: Twelve of 21 patients (57%) reported diplopia when viewing through the reading position, but only three (14%) when viewing through the optical centre. Cover test showed an induced vertical heterophoria in the reading position. Binocularity measured by TNO showed stereopsis to be markedly reduced when looking through the reading position of the spectacle lens. There was no obvious relationship between Awaya aniseikonia measurements and patients' symptoms.

Conclusion: Symptoms of diplopia in acquired anisometropia are more often due to optically induced anisophoria than to the aniseikonia. This finding is contrary to traditional teaching. Recognition of this and simple expedients in management resolve these symptoms for most patients.

Keywords: anisometropia, aniseikonia, anisophoria, prismatic effect

INTRODUCTION

Anisometropia is defined as a difference in refractive error between the two eyes. It is difficult to establish a symptomatic threshold due to significant tolerance variations between patients. It is commonly thought that the main cause of symptoms in patients with newly acquired anisometropia is the ocular image size difference or aniseikonia.[1] Early research in this area was greatly influenced by studies from the Dartmouth Eye Institute from 1920, where many researchers believed that the prism effect was not nearly as important as the differences in ocular image size.[2] Some studies suggest that aniseikonia begins to affect stereopsis at a subjective value of 3-5%.[3,4] This study investigates the degree of aniseikonia that has an impact on binocular function and whether there is any correlation between the amount of aniseikonia and the degree of anisometropia.

Anisophoria is the heterophoria induced by the prismatic effect of unequally ground lenses and it changes when looking from the primary position to other directions of gaze, for example, the reading position. Patients' symptoms can include ghosting, dizziness, feeling off-balance and diplopia. These symptoms increase as they look further away from the centre of the lens which is why many patients complain of double vision or other symptoms with reading. We believe that anisophoria is the main problem for newly acquired anisometropes. This agrees with the teaching of Hess (1903) who stressed the importance of considering the induced anisophoria. He felt that its treatment was just as important, if not more so, than aniseikonia in achieving patient satisfaction and ocular comfort.[2]

Patients who complain of symptoms of acquired anisometropia frequently have had cataract surgery or other types of refractive surgery. Their symptoms are characteristically vague and they have tried many different pairs of glasses and prismatic corrections. We aimed to establish the relative impact of aniseikonia and anisophoria on binocularity and explore which of the two is the major cause of diplopia.

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METHOD

A single examiner (KS) investigated 21 patients who had acquired anisometropia of >1.00 D and astigmatism <1.00 D. Patients were either pseudophakic in one eye or had LASIK surgery with monovision outcome. Best-corrected visual acuity was 6/12 or better in each eye for distance and at least N8 for near, with no significant ocular pathology and no history of strabismus or any current manifest ocular deviation or horizontal heterophoria greater than 15 prism dioptres for near or distance.

Spherical equivalent refractive correction was placed in custom-made frames (Ralph Clarke Optical, Castle Hill) using custom-made lenses to avoid the limitation of standard lens sizes and trial frames (Figure 1). Tests were first conducted in primary position through the optical centre of the lenses as marked by the optical dispenser. Alternate cover test and TNO (Clement Clarke International, Harlow, UK) were used to assess binocularity and stereopsis and the Awaya New Aniseikonia Test (NAT)[5] (Handaya Co, Tokyo, Japan) for image size difference. Patients were asked if they had any symptoms of diplopia. The tests were then repeated whilst the patient looked through the normal reading position. This was standardised with the use of the nose-piece lever, which moved the viewing zone to 12 mm below the optical centre.

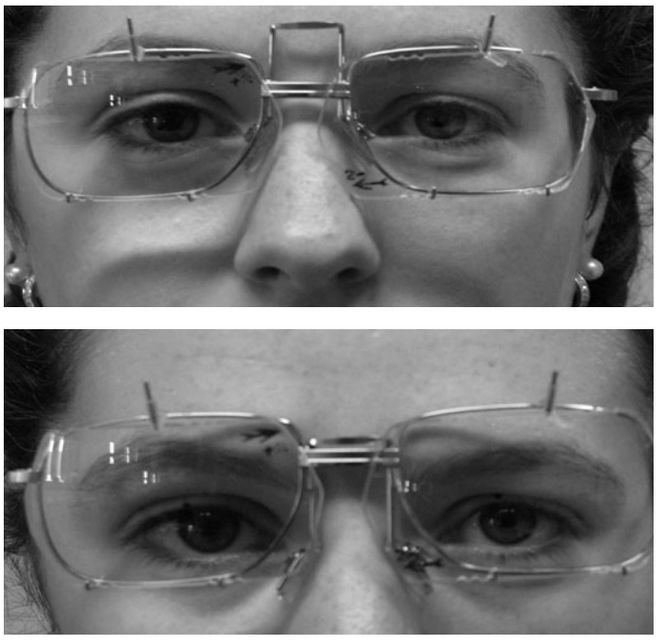

Figure 1. Custom frames made to allow custom lenses, including grooves to allow for horizontal adjustment of interpupillary distance and a centre lever on the nosepiece for standardised movement of the viewing zone 12 mm below the optical centre (Ralph Clarke Optical, Castle Hill, Sydney).

RESULTS

Three of the 21 (14%) patients complained of double vision through the optical centre and another nine had diplopia when looking through the reading position, giving a total of 12 patients (57%) who complained of double vision through the reading position (Figure 2). when looking through the reading position, giving a total of 12 patients (57%) who complained of double vision through the reading position (Figure 2).

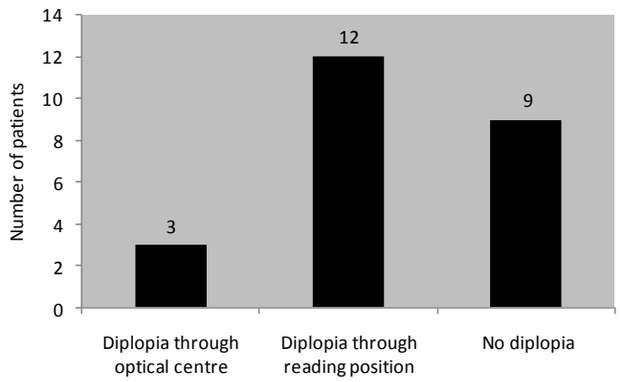

Figure 2. The number of patients with diplopia when viewing through the optical centre and through the reading position.

Results of the NAT showed no apparent relationship between the degree of anisometropia and subjective size difference (Figure 3), however no statistical analysis was performed. NAT results ranged from 0% to 18%. Some patients with large amounts of anisometropia reported no subjective image size difference and conversely a patient with only 1.50 D of anisometropia reported a 5% image size difference. Through the optical centre of the lens where there is no induced prism, only three patients were symptomatic (see circled points on graph in Figure 3).

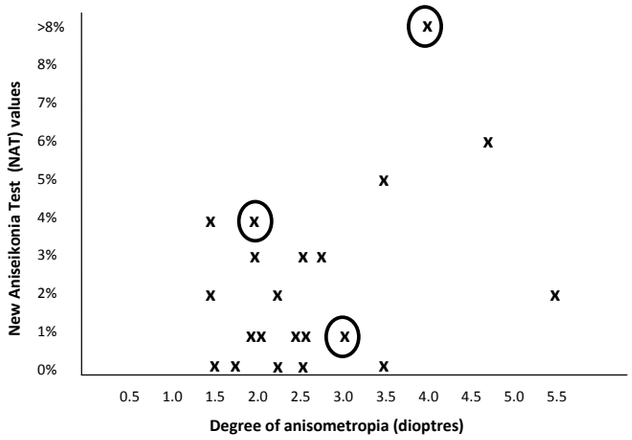

Figure 3. The relationship between the degree of anisometropia and amount of aniseikonia. The circled crosses indicate the three patients who were diplopic viewing through the optical centre of the lens.

The results shown in Table 1 compare the alternate cover test measurements at one-third of a metre through the optical centre of the lens with those through the reading position. In 18 cases (86%) a vertical heterophoria was detected through the reading position which was not present through the optical centre. All diplopic patients demonstrated an induced vertical heterophoria.

Table 1. Comparison of cover test measurements taken through the optical centre and the reading position with the target at 1/3 m

Alternate cover test measurements through optical lens centre	Alternate cover test measurements through reading position
Ortho	2Δ Exo 2Δ R/L
Ortho	2Δ Exo
Ortho*	2Δ L/R*
Ortho*	4Δ R/L*
Ortho	2Δ Eso 2Δ R/L
Ortho	4Δ L/R*
Ortho	2Δ R/L
Ortho	2Δ Exo 2Δ R/L*
Ortho	2Δ Exo 2Δ R/L
Ortho	2Δ R/L
Ortho	2Δ L/R*
2Δ Exo	2Δ Exo 2Δ R/L
2Δ Exo	4Δ Exo
2Δ Exo	3Δ Exo 4Δ L/R*
4Δ Exo	6Δ Exo 2Δ L/R*
4Δ Exo	6Δ Exo 4Δ R/L*
4Δ Exo	6Δ Exo
4Δ Exo	4Δ Exo 2Δ R/L*
8Δ Exo*	6Δ Exo 4Δ R/L*
12Δ Exo	12Δ Exo 5Δ R/L*
14Δ Exo	14Δ Exo 4Δ R/L

Ortho = orthophoria, Exo = exophoria, R/L = right hyperphoria, L/R = left hyperphoria, * = diplopic patients

TNO stereopsis scores were used as a measure of binocular function both through the optical centre and reading position of the lens. Results show that through the optical centre of the lens, 18 patients (86%) scored better than 480 seconds of arc (Figure 4). Through the reading position only eight patients scored better than 480 seconds of arc (38%) as shown in Figure 5. The patients who developed diplopia looking through the reading position were predominantly the same patients who reported a reduction in TNO stereopsis (indicated by circles on the graph in Figure 5).

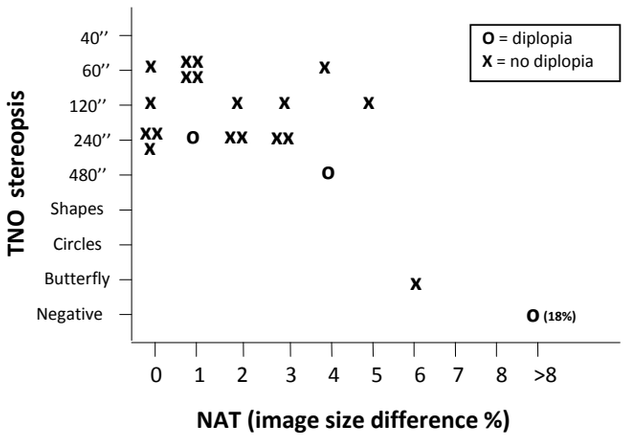

Figure 4. TNO results through the optical centre of the lens.

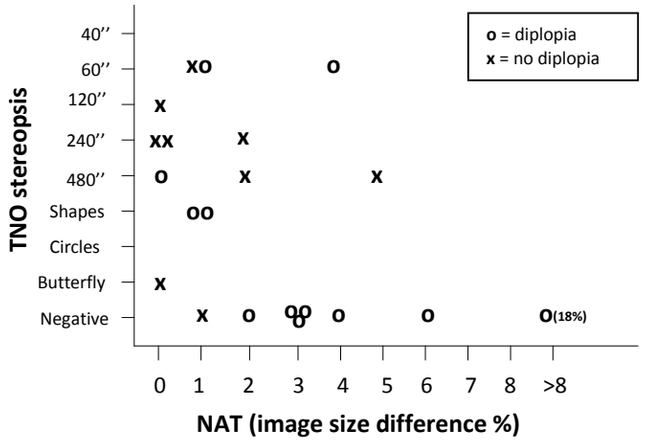

Figure 5. TNO results through the reading position.

DISCUSSION

The results in this group of patients indicate that diplopia, when it occurs, is more often caused by anisophoria or anisotropia secondary to induced prism than to aniseikonia. Prior to extrapolating this finding to other anisometropic patients it is important to consider some aspects of the study.

The range of anisometropic error in the patients studied extends only to 5.50 dioptres and all cases were newly acquired. Studying patients with larger errors, childhood onset, or those with longer periods of adaption may well give different results but the cohort selected represents a not uncommon clinical scenario and thus is of interest.

The use of the custom-made frames to physically move the lenses, thereby utilising the reading position without having the patients move their eyes, is clearly different to normal reading behaviour. The advantage is a standardisation of

optical change, difficult to achieve using normal glasses, and may not be directly comparable to the situation when the visual axes are rotated downwards in the normal course of reading. Utilising this device does however limit the change to a single variable which perhaps strengthens the conclusion.

Patients' descriptions of their symptoms are frequently vague including dizziness, ghosting, eyestrain and blurred vision as well as double vision. Reporting only double vision may underestimate the frequency of symptoms in this group but provides a clearer decision for the patients in their reporting. An accepted normal value for vertical fusion range is 2-4 prism dioptres[6] and it seems likely that as the induced vertical heterophoria reaches these levels it will manifest as a heterotropia resulting in double vision, albeit intermittently. This scenario fits with the intermittent and often vague nature of these patients' symptoms.

Stereopsis and the alternate cover test provided the measurement of binocular function in the study. Both of these measurements demonstrate deterioration in the reading position and closely follow the increased frequency of subjective diplopia. The only optical change is the prism-induced anisophoria and it seems reasonable to attribute the increase in symptoms to this change. Whilst not the primary aim of the study, it appears from the results that there is no clear link between the magnitude of anisometropia and the subjective appreciation of aniseikonia, however no statistical analysis was undertaken. It might be expected that the three patients who experienced diplopia when tested through the optical centre of the lenses were those with the larger NAT scores, however two of these three patients had only mild subjective image size differences of 1% and 3%. Literature reports vary on this issue with levels of up to 7% tolerated by some,[4,7,8] whilst levels as low as 1% were symptomatic in others.[4]

Recognition of the contribution of anisophoria in the patients' symptoms suggests treatment options other than 'size' or aniseikonic lenses. Simply prescribing separate reading glasses rather than bifocal or multifocal glasses will assist the patient to utilise the optical centre of the lenses and minimise diplopia. Contact lens correction or surgery to minimise anisometropia are also useful treatment options.

CONCLUSION

The results of this study suggest that in this patient group 'size does not matter much' and it is the anisophoria that more often disrupts binocular function leading to patient complaints of diplopia. They do not support the notion that the image size difference is the main cause of problems in patients with acquired anisometropia.

Even if patients do specify diplopia as their concern, simply performing a cover test in the primary position does not reveal the problem. Recognition of the likely cause, careful attention to the patient history, cover testing and tests of binocular function in gaze positions other than primary will assist in arriving at the correct diagnosis.

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Orthoptic Interventions in Stroke Patients

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ABSTRACT

Patients admitted to hospital following a stroke, as part of the recovery process may require active intervention to relieve visual symptoms. The interventions include therapy, correct use of or modification to spectacles (including use of prisms), appropriate occlusion or the adoption of compensatory strategies to support ocular comfort.

This paper falls into two sections. It initially provides an overview of the strategies currently used for vision problems found in patients who have had a stroke. It refers to the general indictors for intervention and the possible strategies that can be used. The second part of the paper looks at outcomes citing patient responses from a 2008 report to the Statewide Ophthalmology Service of the Greater Metropolitan Clinical Taskforce.

The strategies reported include therapy, correct optical use, occlusion and diplopia relief and strategies to maximise ocular comfort. Approaches used are often simple and very effective in terms of patient comfort or educating other team members about the need to support a compensatory strategy. Some strategies require active follow-up with variable outcomes. The outcomes support the benefits of orthoptic intervention in the care of patients recovering from stroke.

Keywords: orthoptic intervention, therapy, stroke

INTRODUCTION

Stroke is the third greatest cause of death and the leading cause of disability in adults in Australia.[1] As stroke is a neurological condition, the likelihood of causing visual deficits is great because of the high representation of sensory and motor ocular areas within the brain.[2] Its impact includes visual field loss,[3,4] visual neglect, and ocular motility problems associated with diplopia (cranial nerve palsies, loss of convergence), nystagmus and gaze palsies.[4] The population who are most likely to suffer from a stroke are in the age group where other ocular conditions can also affect their visual status. These conditions include glaucoma, cataracts, age-related macular degeneration and vascular conditions such as diabetic retinopathy and hypertensive retinopathy. Both acquired and pre-existing vision defects can decrease the patient's ability to see clearly and so decrease their ability to participate in and maximise the rehabilitation process, and conversely their newly acquired impairments from stroke may hamper their ability to benefit from orthoptic intervention.

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Stroke rehabilitation commences once the patient's condition is stable. Several health care practitioners can be involved in rehabilitation through ongoing therapy. This may include speech therapists who assist in swallowing and communication skills; physiotherapists who assist motor skill enhancement and mobility training; and occupational therapists who support activities of daily living. Therapeutic strategies relating to hemianopia[5,6,7,8] and neglect training[8,9] are well documented in the literature, however there is no mention of convergence training or adaptations to assist patients to manage their ocular conditions.

A report of the Statewide Ophthalmology Service of the Greater Metropolitan Clinical Taskforce titled "The orthoptist and the management of visual problems in inpatients with stroke"[10] has revealed that ocular conditions both pre-existing and as a consequence of the stroke, have a significantly higher detection rate when tested by an orthoptist ($p<0.001$) compared to the detection rate by other health professionals in the inpatient setting of a stroke unit. The study was conducted across three sites where there were three different models of eye/orthoptic care (Model 1 – eye outpatient clinic and inpatient orthoptic assessment; Model 2 – eye outpatient clinic only; Model 3 – no eye clinic available). One unplanned outcome from the study was the identification and where possible, management of patients with vision problems that could benefit from intervention.

Patients requiring interventions could be identified across the whole study, but in only one hospital within the study was an orthoptist employed and available to provide follow-up for therapeutic interventions.

The first aim of this paper was to report on those cases within a study population of 150 people admitted to hospital following a stroke who required intervention strategies to assist them with their ocular problems and to enhance their response to the rehabilitation process. The second aim of this paper was to provide case studies as examples to illustrate outcomes.

PROCEDURE

A total of 150 randomly selected patients who had been admitted to one of three stroke units (50 from each unit) because of a vascular incident were included in the study. This study had ethics approval from the Sydney South West Area Health Service Ethics Committee and the University of Sydney Ethics Committee. At the time of invitation into the study the visual status of the participants was unknown. All participants were made fully aware of the purpose of their involvement and signed a consent form. The relatives of two patients refused participation and they were not included in the study.

After initial assessment by the orthoptist, those patients with ocular conditions requiring intervention were identified. The basis for their selection was that due to their ocular condition they would benefit from intervention such as convergence training, scanning for visual field loss, neglect training, optical intervention, occlusion, or training for compensatory strategies.

As a precursor to specific treatment strategies a clear explanation was given using a variety of approaches to ensure that the patient understood their deficit and its cause. This was achieved by written information, diagrams and demonstrations through miming and role play, which were used as appropriate for the patients' communication ability. An ocular care plan was developed and implemented with the assistance and consent of the patient.

ORTHOPTIC INTERVENTION STRATEGIES

Prior to presenting specific outcomes and patient responses the following provides an overview summary of the strategies generally employed in a stroke unit to assist in attaining the best possible ocular function.

1. Therapy is defined as a clinical strategy in which there is a program of structured active ocular procedures designed to bring about change in ocular function and to alleviate symptoms. This could include:

Convergence training: In patients with stroke, defective convergence affects the patient's ability to manage daily ward activities such as reading instructions, menus and for leisure. In addition, good convergence is needed in order to undertake fine motor tasks required by other therapists, for example games and activities involving cognition, object detection, location, placement and manipulation of objects. Table 1 identifies the triggers that prompted a need to treat the convergence and the action undertaken. Therapy was provided to enhance the extent and comfortable use of binocular single vision (BSV) so that tasks were easier to perform and the patient could be more comfortable. The orthoptist identified the exercises to be practised and these were carried out by the patient or supervised by another health care practitioner, for example the physiotherapist whilst undertaking their physiotherapy program. The orthoptist regularly checked progress and modified the exercises.

Table 1. Convergence deficiency features

Indicator - Patient symptoms	Clinical treatment (*when applicable)
Diplopia, words overlapping Asthenopia Problems reading	Involuntary convergence - target to nose. spot card Voluntary convergence* Stereogram cards*

Scanning to compensate for visual field loss: Visual field loss has a profound effect on the patient's ability to orientate in their environment,[6,7] either at the hospital or at home (Table 2). This is a serious issue that requires risk minimisation to prevent trauma occurring, such as falls or burns. Social skills such as initiating and maintaining eye contact whilst conversing are another problem.

Table 2. Visual field loss features

Indicator	Clinical treatment
Bumps into objects - doorways and furniture Knocks over objects Fails to make eye contact on affected side Reading does not make sense, eg left hemianopia - starting point half way across the page, right hemianopia - reads only first part of large words	Educate patient to be aware of loss and explain reason Position bed so stimulation occurs on hemianopic side, ie avoid a blank wall on hemianopic side Teach full field awareness by using a synchronous body and ocular adjustment, eg with left hemianopia when walking, look left as moving left foot Teach to scan – using light stimuli, then clock, pictures on wall, and objects on tray Left hemianopia – use red strip to locate beginning of line; right hemianopia – use an "occluder" to expose letter-by-letter the entire word to be read Coordinate with the physiotherapist to make an obstacle course to teach mobility with scanning

Essential tasks like reading become problematic with loss of place, missing parts of long words, difficulty locating the end of a line and beginning a new line. The impact of the field loss increases frustration levels with reading, results in a loss of contextual meaning and can lead to a reluctance and avoidance to attempt the task.

Therapy includes making the patient aware of their field loss and teaching strategies, especially scanning training, to minimise the effects. One such early scanning method uses a series of paired coloured stimuli equally and symmetrically distributed along a horizontal plane. The patient is asked to locate lights or dots of the same colour on each side of the central fixation stimuli as a means of raising the sensory visual awareness in both the seeing and unseeing peripheral areas.

Visual neglect training: Visual neglect is a failure to transmit information perceived by the visual cortex so that it is not appreciated by the visual association areas. It presents as inattention/awareness of one side of the body and or visual environment. Visual neglect is caused most commonly by damage to the parietal lobe. It may or may not be associated with a field defect.[8] Indicators of the presence of visual neglect are outlined in Table 3.

Table 3. Visual neglect features

Indicator	Clinical treatment
Head and eyes turned away from the side with the neglect, to the unaffected side	Bed position (as for visual field loss)
Failure to realise someone is present	Exercises/tests – line bisection, letter crossing
Lack of ownership of self (face and limbs) or environment (meals)	Exercises – for left loss use a red strip to read; for right loss expose a few letters at a time
Bumps into objects as in Table 2	Use a picture scene to locate separate entities including wall mural
	Use a clock face, house features, paragraphs with random indentations, mazes, word games, adapted Diller-Weinberg apparatus
	Encourage tasks which require matching from one side to opposite side, ie food on tray

Therapy for visual neglect differs from visual field training in that the neglect has the capacity to resolve as the area of the brain involved is more diffuse than the tract of the visual pathway which has limited boundaries.[9.] Therefore, constant stimulation of the side with the neglect gives rise to brain adaptation to receive the visual information.

2. Optical Use and Intervention refers to ensuring that patients have their glasses available and use them whilst in the hospital environment. This includes strategies that lead to the prescription of glasses, the modification in the use of existing glasses, changing existing prescriptions or use of an optical appliance. Modification to glasses becomes necessary when vertical gaze defects are present and the patient cannot look down, preventing the use of previously prescribed bifocals. The patient thus requires single focus spectacles (Table 4). In addition, post stroke, many patients report not being comfortable with the small size of the reading segment of their bifocals and again, more effective reading segments may be required.

Table 4. Optical condition features

Indicator	Clinical treatment
Problems reading	If unable to look in depression, suggest separate readers and a reading stand to keep print level
	If reading glasses are poorly fitted and/or bifocal segment too small, suggest single vision glasses
	Use of magnifying device
	Increase light level
Diplopia	Prisms may be applied to glasses or loaned plano glasses, prism may be tilted to correct combined vertical and horizontal defect
Dilated pupil	Use of sunglasses for glare reduction

In the presence of diplopia, prism correction is highly beneficial.[11] In the presence of one or both pupils being dilated, the use of sunglasses to reduce the impact of glare is invaluable.

3. Occlusion. In the rehabilitation environment, diplopia as a result of a decompensating deviation or cranial nerve palsy results in uncertainty about object location and loss of balance.[11] It can result in many problems including misjudging position, leading to self-injury and falls. Occlusion of the most appropriate eye, according to the acuity, pathology and eye with the paretic muscle, is important to support the best response from the patient, particularly in active physical therapy sessions (Table 5).

Table 5. Occlusion features

Indicator	Clinical treatment
Diplopia	Evaluate which eye to cover - ie poor vision in one eye, lid closure with IIIN palsy
	Effect on proprioception if forced to use palsied eye
	Partial occlusion allows the use of BSV where possible with half-lens occlusion

4. Supporting Compensatory Strategies. Patients with visual problems linked to cranial nerve palsies who experience diplopia, or who have nystagmus with associated diplopia and images that are blurred and moving, often use a head posture to decrease the impact of the visual problem. Clinical experience has shown that in rehabilitation these patients may have physiotherapy involving posture control which is generally based on keeping the body vertically aligned to respect gravity, ie head and body held straight. If a compensatory head posture has been adopted by the patient to overcome diplopia, or place the eyes in the position of least nystagmus, straightening the head may cause the diplopia and or the vision to worsen. The orthoptist needs to advocate on behalf of the patient to retain the compensatory head posture (Table 6).

Closing an eye could be indicative of a problem such as diplopia. If the need to shut the eye is constant then occlusion or prisms may be required. Some patients may only shut an eye intermittently, for instance when looking into some positions of gaze. Segment occlusion may stop this need, or if the problem is minor, closing the eye may be supported to continue.

Table 6. Compensatory strategies

Indicator	Action
Presence of head posture – head tilt, chin position or face turn	Ascertain if ocular or due to stroke deficit
Closing an eye	Allow use if to join diplopia or to use null point of nystagmus
	Explain to other therapists the advantage gained by head posture
	Investigate reason

OUTCOMES

There were 150 participants with an age range of 24 to 95 years (mean age 75 years), 78 (52%) females and 72 (48%) males. All participants had been admitted because of a vascular incident which included stroke (70%), TIA (20%) or unspecified cause (10%).

Seventy-five participants (50%) were identified by the orthoptist as requiring intervention. As the investigation of the interventions was not planned as part of the original report, the outcomes were not followed in detail and are therefore provided as a broad description. The outcomes fall into two broad categories, the first where the participants were actively involved in therapy and the second, where the procedure was delivered to provide comfort but active participation was not required. Within the stroke population the acceptance of interventions was dependent on factors which included the cognitive and physical status of the participants. Such factors as refusal to cooperate, or discharge occurring prior to or during the treatment phase, also affect the ability to appropriately evaluate intervention outcomes.

Actual therapeutic strategies were only carried out at the hospital where the orthoptist was a permanent member of the inpatient team. Of the 50 participants seen at that hospital, eleven were given therapy, three for treatment of convergence insufficiency, five for visual field scanning and three for neglect training.

The identified non-therapeutic strategies were provided for the 150 participants across all three hospitals: four required occlusion; two eye-padding for medical conditions; five for compensatory strategies; forty-nine for optical strategies (30 referred for prescription, 13 left their glasses at home, three were not wearing their glasses, and three were asked to change their glasses from bifocals or multifocals to single focus lenses); and four were provided with Fresnel prisms. End-results are not known because of an inability to follow up the participants to determine the outcome.

1. Therapy Outcomes

Convergence training: Of the 150 participants, there were 61 (40.7%) whose convergence near point (CNP) was less than 6 cms, with 14 (9.3%) of those having a CNP of less than 10 cms. Each of these participants required questioning regarding near problems, particularly those with a near point further than 10 cms. The following case studies illustrate the clinical presentation of patients with convergence problems in a stroke unit and highlight the challenges faced when initiating a treatment program (Table 7).

Scanning for field loss: Of the 150 participants, 20 had either a hemianopia or quadrantanopia detected by confrontation or Bjerrum field test. These patients may benefit from orthoptic intervention to enable full and safe mobility and daily living skills. The following cases illustrate the clinical features and response of three patients, two of whom had reported that the field loss had an impact, and one where intervention was not actually sought by the patient (Table 8).

Visual neglect training: Eight patients were identified with visual neglect, confirmed by the patient's negative response to the "simultaneous binocular presentation test". In this test the patient is asked to fixate straight ahead and identify the total number of fingers presented separately and simultaneously on each side of the midline. The following cases illustrate the clinical features and responses of three of these patients (Table 9).

Table 7. Case studies for convergence deficiency				
Age	**CNP**	**Condition impact**	**Strategy**	**Outcome**
91 years	10 - 15 cms	Intermittent diplopia when reading	Treatment undertaken for two days, a total of 10 minutes involuntary convergence training	Non-compliant, could not see the point of the exercises
49 yrs	10 cms, fatigues to 15 cms	Fuzzy vision	Treatment recommended	No orthoptist to follow up
48 yrs	6 cms	Mild headaches, uncomfortable when reading and doing close work	Exercises commenced for physiotherapist to follow up	Lost to follow-up

Table 8. Case studies for field defects				
Age	**Field defect**	**Condition impact**	**Strategy**	**Outcome**
78 yrs	Left homonymous hemianopia	Aware of vision loss; no pursuit movements beyond midline	Three treatments with light-board and picture stimuli	Improved mobility and awareness reported by therapists
74 yrs	Right hemianopia, macular splitting	Nil	Discuss with family	Family aware of the patient's visual loss and take this into account when interacting with the patient
74 yrs	Left hemianopia, macular sparing	Blurred vision and transient vision loss	Four visits to teach compensation for field loss	Improved ability to direct intact seeing area to support safe mobility

Table 9. Case studies for visual neglect training				
Age	**Neglect**	**Condition impact**	**Strategy**	**Outcome**
75 yrs	Left	Only looks to left if asked; fix and follows only from right to left	Block right stimuli, four light-board treatments	Noticing objects on left; sees objects on both right and left presentation
74 yrs	Left	Unaware of left arm for physiotherapy; with line-bisection test, only responds to extreme right	Seven treatments with the light-board; red strip; describing complex pictures	"Huge improvement" reported by staff and patient, fully orientated to all parts of body and hospital environment and ADLs
74 yrs	Left, associated with partial left hemianopia	Eyes and head constantly to right and unaware of environment on left	16 treatments with light-board; red strip; describing complex pictures	Spontaneously looks to left occasionally, better response with physiotherapy, and eyes more in primary position

2. Optical Use and Interventions Outcomes

There were 49 optical interventions, of which 30 were referred for a prescription or update of glasses, as illustrated in Table 10. An additional 13 had left their glasses at home and relatives were requested to bring the glasses to hospital, and a further three patients were advised to wear the glasses they had with them. Four patients were recommended to either change their existing glasses from bifocal or multifocal to two separate pairs of glasses or to use their existing glasses correctly to enhance their visual comfort. Four patients were fitted with Fresnel prisms to enable them to regain BSV.

Table 10. Case studies using optical interventions				
Age	**Issue**	**Condition impact**	**Strategy**	**Outcome**
77 yrs	Bifocal segment too small	Glasses uncomfortable, had to be lifted to read	Two separate pairs of glasses	Patient very happy
90 yrs	Left glasses at home	Decreased distance vision RE 6/24, LE 3/60	Glasses brought from home	Improved vision R & LE 6/12
83 yrs	Not wearing glasses with pre-existing prism	Diplopia without glasses	Wear glasses full-time	Single vision with glasses
74 yrs	Diplopia	Vertical diplopia present	Prisms	Single vision in primary position

3. Occlusion Outcomes

Four patients were treated with occlusion to assist them to gain a single image and ocular comfort. The methods of occlusion used included a translucent filter, half-lens and total occlusion. The translucent filter was used because it was more cosmetically acceptable and for patients who

could tolerate the level of blockage provided by the filter. The half-lens occlusion was used on the lower segment of the spectacles where the deviation and diplopia were present, thus allowing the patient to capitalise on their binocular single vision when looking through the top of the glasses (Table 11). Total lens occlusion was used to overcome diplopia present in all positions of gaze.

Table 11. Case studies using occlusion

Age	Issue	Condition impact	Strategy	Outcome
74 yrs	Diplopia	No confidence in walking	Half-lens lower segment occlusion	Vary satisfied
82 yrs	Diplopia	Discomfort	Filter occlusion	Eyestrain, changed to prism

Two additional patients were treated as part of a medical procedure. One had a full ocular pad for a corneal ulcer and the other used tape to ensure lid closure in the presence of VII CN palsy with corneal exposure.

4. Supporting Compensatory Strategies Outcomes

There were five patients who required compensatory strategies, each of which took individual and different approaches. For instance, in two patients adaptations were used to allow visual comfort, and in one the patient was happy continuing as he had always done without clinical intervention (Table 12). Part of the management is this area links to observation and part to listening to the patient and their personal comfort issues.

Table 12. Case studies demonstrating compensatory strategies

Age	Condition impact	Strategy	Outcome
87 yrs	Nystagmus in primary position	Lift chin to have stable vision	Patient expressed satisfaction
70 yrs	Vertical gaze defect, could not use bifocals	Support to continue lifting glasses and/or get single focus lenses	Pleased to be advised to adapt glasses
83 yrs	Constant diplopia, which was present prior to the stroke	Leave alone	Patient expressed satisfaction

DISCUSSION

Patients post stroke have complex issues of physical defects such as hemiparesis, difficulty swallowing, communication issues and vision defects, as well as cognitive problems such as confusion and apathy. They have to contend with the changed environment of the hospital and respond to a variety of health care practitioners each with a different role to play in their recovery process. In this environment the best visual status will assist the patient to respond.

Based on the outcomes of the 2008 report "The orthoptist and the management of visual problems in inpatients with stroke",[10] fifty percent of the participants in the study had interventions recommended, which is a large number of people identified with vision problems. Strategies to achieve the best visual status may be as simple as bringing glasses from home and using them, using glasses correctly or seeking modification of the current lens format into two separate pairs of glasses. In addition an explanation to the patient about their vision problem, its impact and how to adapt, can empower the patient to use their eyes more effectively and achieve a better outcome from the rehabilitation process. It is often surprising that seemingly simple orthoptist-directed actions can change the patient's attitude and level of cooperation. There is also a strong sense of satisfaction to observe a patient with improved ocular function performing leisure activities such as watching television and reading. In addition, to receive feedback from other health care practitioners about improvement in participation in rehabilitation processes following eye care intervention is extremely rewarding.

This study has revealed that treatment strategies can be effective when they are orthoptist-directed, with supervision and follow-up by an orthoptist or other health care practitioner. This was demonstrated in the area of scanning for field loss and neglect where close and regular supervision of the patients resulted in improvement. Conversely, treatment for convergence deficiency was generally not effective, likely due to contributory factors including stroke-related dementia and cognitive damage preventing awareness of the purpose and benefits of the treatment, fatigue, lack of sustained concentration and patient discharge from the hospital before the completion of treatment. Therapeutic approaches therefore have variable outcomes. However, as can be seen in the case studies, patients do benefit and having some negative outcomes should not deter the orthoptist from implementing strategies. Consequently, it is important that the therapeutic interventions should be set at a level dictated by the capabilities of the patient.

Whilst there are studies that discuss the various approaches and outcomes for treating field defects and visual neglect, there are no reports on the other interventions. The outcomes in this study do have limitations because they are reported as either general subjective patient responses or observed improvements in patient behaviours and responses to other activities. There is a need for larger randomised controlled studies of patients undertaking active treatment strategies with objective measurement through tools such as quality of life questionnaires. There is also a need to measure the impact of improved vision responses on the patient's ability to interact with

other health care services, which may then decrease the length of stay in hospital and the impact on total health expenditure. Outcomes can then guide practitioners in their role in the area of intervention for eye care in the field of stroke.

CONCLUSION

The outcomes from the study do not measure the impact of orthoptic intervention in monetary terms but through the case studies have demonstrated increasing cooperation and decreased frustration from patients whilst performing daily tasks, therapy activities and their response to interpersonal interaction. The orthoptist is well placed to provide practical support directly to patients, in turn assisting the rehabilitation process in terms of service delivery and time management.

A future study into more objective measurement of outcomes plus time and cost savings is recommended.

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Two Case Studies: Eccentric Fixation and Amblyopia - A Challenge to the Treating Practitioner

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ABSTRACT

Two cases of eccentric fixation in the presence of strabismic and anisometropic amblyopia are presented, both of which failed to respond to therapeutic efforts. A brief account of past and present treatment modalities used in the management of eccentric fixation is provided, including a discussion as to the limitations and efficacy of each. Analysis of the literature reveals that regardless of the treatment method employed, a population of "incurable" patients exist who fail to improve despite treatment efforts. Treatment outcome is dependent upon a multitude of factors and the potential reasons as to why this sub-group of patients with eccentric fixation fail to show improvement are discussed. This paper serves to highlight the challenges that such cases pose to the treating eye care practitioner and encourages the need for further research in this area; an area where little is known even today.

Keywords: eccentric fixation, amblyopia, strabismus, anisometropia, visual outcome

INTRODUCTION

Amblyopia is a leading cause of monocular vision impairment.[1] Known causes of amblyopia include ocular misalignment (strabismus) and a difference in uncorrected refractive error between the two eyes (anisometropia).[2] Mixed strabismus and anisometropia is reported as the cause of amblyopia in 37% to 43% of cases.[3-7] In the majority of instances, amblyopia can be successfully treated via means of occlusion therapy, atropine penalisation, spectacle correction or a combination of these.[4,6,8-10] However, not infrequently the treatment of amblyopia is made challenging by the presence of non-central fixation in the amblyopic eye. Eccentric fixation has been reported by Cüppers (1958) and Von Noorden (1970) to exist in as many as 35% to 44% of cases of amblyopia and thus constitutes a considerable factor in the management of this condition.[11]

Fixation typically involves the purposeful imaging of an object of interest on the fovea - the part of the retina in most individuals that possesses the highest resolving power and holds principal visual direction.[12-14] However, in individuals with eccentric fixation a reorganisation of retinal motor values occurs such that a retinal point other than the fovea assumes principal visual direction.[11,15,16] This occurs despite a reduction in the level of visual acuity achievable with the eccentric retinal locus compared to that permitted by the fovea.[13,15,16] The visuscope, born out of earlier improvements in diagnostic armamentarium, made possible the identification of such anomalies in fixation and upon its invention contributed to eccentric fixation no longer being considered a rare phenomenon in amblyopic eyes.[17] The visuscope is still commonly used by practitioners today and represents an invaluable tool in the diagnosis and classification of eccentric fixation.[18]

Despite the therapeutic management of eccentric fixation being the subject of much controversy in the past, the restoration of central fixation and subsequent reversal of amblyopia is generally achieved in most cases today.[1] However, a minority of patients still remain unresponsive to treatment.[19] In such cases, an investigation as to the factors impinging upon the prognosis is warranted and can aid in making a clinical decision about when to cease treatment.

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CASE REPORTS

Case Report 1

Master C presented at age 2 years and 3 months with a right esotropia present since birth. No known family history was reported and general health was otherwise unremarkable. Cover testing confirmed a constant right esotropia, with RSO-- and RIO++ revealed on ocular movement testing. Unaided visual acuity was assessed and no response to any of the single Kay Pictures could be elicited from the right eye at 0.5 metres, with marked objection to left cover. Visual acuity in the left eye was 3/5 unaided. Non-central fixation of the right eye was diagnosed on visuoscopy, wandering in the area of the disc. Left fixation was central. Cycloplegic refraction revealed hypermetropic anisometropia and glasses were prescribed, RE +4.50 DS and LE +2.50 DS. No other clinically significant findings were apparent.

In a three-month follow-up visit, right amblyopia was seen to persist, with no improvement in vision. Left occlusion was then prescribed for four to six hours per day for one month. Over the following nine months, when left occlusion was prescribed for six hours per day, the family reported that he mostly wore it for four to five hours a day, with generally good compliance. Regular review showed right fixation remained eccentric midway between the fovea and disc, with a negligible improvement in vision, stabilising at LE 3/30 and RE 3/4.5. The esotropia remained stable at 25 to 30 prism dioptres.

At this stage of the treatment, Master C began demonstrating social withdrawal, becoming uncharacteristically solemn and lacking motivation to play or venture outdoors. Master C's parents were advised to taper occlusion and strabismus surgery was to be considered.

Case Report 2

Miss D presented at age 4 years and 3 months with a right esotropia present since birth. General health was otherwise unremarkable. Past ocular history included weekly atropine penalisation for eight months and no prior surgery or occlusion therapy. Cover testing revealed a moderate right esotropia not taking up fixation, measuring 25 prism dioptres by Krimsky reflections. Ocular movements were full. Visual acuity with glasses was RE 2/60 (Sheridan Gardiner singles) and LE 3/4.8 (matching logMAR). Visuoscopy revealed unsteady eccentric fixation in the amblyopic eye and central fixation in the left. Hypermetropic anisometropia was present and spectacle prescription was increased to RE +9.00/-1.25 x 180° and LE +2.50/-1.25 x 175° at this visit.

Minimal change in vision was noted in a subsequent examination three months later. Left atropine penalisation was then prescribed twice per week with one hour of left occlusion per day. Over the following 11 months, poor treatment compliance was reported and Miss D failed to attend several visits. Despite the family being advised on consecutive occasions as to the importance of occlusion therapy, the patient completed irregular and ill-sustained periods of patching and atropine occlusion.

Upon regular review, right fixation remained eccentric and unsteady, midway between fovea and disc. At age 5 years and 5 months limited improvement in visual acuity was observed, RE 3/18 (Sheridan Gardiner singles) and LE 3/3 (logMAR chart). Only one letter on the 3/30 line was seen when visual acuity in the right eye was tested using the crowded chart. At this stage, the patient was advised to increase left occlusion to four or more hours per day and continue left atropine twice per week until no further improvement in vision could be attained.

DISCUSSION

Despite eccentric fixation representing a common phenomenon in young patients with strabismus or severe unilateral retinal image blur, little is known about the exact aetiology of this fixation anomaly. Some have proposed that a causal relationship exists between abnormal retinal correspondence (ARC) and eccentric fixation, whereby the facultative change in principal visual direction that occurs under binocular conditions in ARC can progress to become obligatory under monocular viewing and thus manifests as eccentric fixation.[20] However, this theory has been refuted on the basis that the angle of anomaly and angle of eccentricity are not always equal.[13,21] Others suggest that eccentric fixation is the direct result of a non-organic defect in foveal function brought about by sensory inhibition.[20,22] Allegedly, the reduction in foveal function experienced through suppression is occasionally carried to such an extent that the resolving power of the fovea is reduced to a level below that of the surrounding retina.[23] The incentive is then for the amblyopic eye to fixate eccentrically. However, this mechanism remains questionable as frequently the visual acuity at the fovea remains superior to that at the locus of eccentric fixation in amblyopic eyes.[24,25]

Many different treatment modalities for eccentric fixation have been employed in the past, including pleoptics, red filter treatment, inverse prisms, inverse occlusion and direct occlusion. All reported success, with 60% to 98% of cases showing improvement,[17,18,26-33] however, none were without their limitations.[26,29,30,34,35] Pleoptics involved dazzling the peripheral retina of the amblyopic eye with a high intensity light, then stimulating the fovea with prolonged sessions of intermittent flashing, followed by exercises to restore foveal straight-ahead projection.[16] Generally only suitable in children aged 7 years or older, it was popular following its initiation but later abandoned due to inconsistent results and the treatment proving both expensive and arduous.[16,36-38] Similarly, others reporting on the efficacy of red filter treatment, where the sound eye was occluded and a red filter placed over the amblyopic

eye to preferentially stimulate the cone photoreceptors,[26] noted that central fixation could be restored to normal in some instances but that maximum visual acuity was not usually obtained.[27,37] Inverse prisms, designed to force the eye to make a fixation movement so that the fovea is in a straight-ahead position, were used in conjunction with hand-eye activities to retrain the principal visual direction.[39] However, this method demanded significant patience and adherence to detail on behalf of the patient, and successful results often required up to nine or ten months in difficult cases.[39] Inverse occlusion was advocated by Bangerter (1953) and Cüppers (1958) on the premise that occlusion of the eccentrically fixing eye could disrupt the links connecting the fovea of the sound eye with the eccentric retinal locus of the deviated eye. However, this method has been largely replaced by conventional occlusion; with the aim that occlusion of the normally fixing eye will result in a re-establishment of central fixation and improvement of vision in the amblyopic eye.[17,19,36]

On researching the literature, the authors have found minimal reported studies or mention of eccentric fixation since the 1970s, a fact also noted by others.[3] Mainstream treatments used in contemporary practice aim to promote the function of the amblyopic eye and encourage central fixation by restricting, usually through direct occlusion or atropine penalisation, the competitive advantage of the fellow eye.[1,19] These methods are commonly preceded by spectacle correction, with the aim of first minimising retinal image blur owing to uncorrected refractive error.[1] Recently, the valuable role of refractive correction in the treatment of amblyopia was reported.[4,9,40,41] Refractive correction alone has been reported to improve visual acuity an average of 2.9 lines and result in resolution of amblyopia in at least one-third of 3 to 7 year-old children with untreated anisometropic amblyopia.[4]

In modern practice, direct occlusion for the treatment of severe amblyopia is now advised regardless of the type of fixation, provided that the patient is still within the plastic age of visual development.[19] It has been demonstrated that six hours of occlusion per day produces a similar outcome to full-time occlusion in severe amblyopia,[5] or even two hours occlusion with atropine penalisation.[6] Two hours of daily patching combined with one hour of near activities has also been shown to modestly improve amblyopia associated with strabismus, anisometropia, or both, in children aged 3 to 7 years old.[10] Earlier studies of children with eccentric fixation who were successfully treated, reported that central fixation and maximal visual acuity was generally achieved within three to four months of full-time occlusion, with a small number of children being occluded for up to nine months.[17,30,31,35] More recently, dose-response studies have shown that vision improvement reaches a plateau around 100 cumulative hours,[7] or at 200 hours, with minimal improvement after 400 hours.[3] Maximum improvement occurs within the first three to four months,[3,5,6] but further improvement can be demonstrated up to six months.[6] It has also been suggested that strabismus surgery disrupts the eccentric fixation due to proprioceptive and innervational influences, but that this seldom results in a spontaneous improvement in vision.[17,18]

Despite being the treatment method of choice, occlusion therapy is not without its limitations. The implications of occlusion on the psycho-social well-being of patients, including peer victimisation,[42,43] social stigma and subsequent alterations in self-concept,[44] have been documented. The ill effects of which were beginning to manifest in Master C following prolonged and intensive treatment. For this reason, atropine penalisation is often favoured over occlusion for the decreased social burden inflicted and has been shown to produce similar treatment outcomes to patching.[8,45]

Regardless of the treatment modality employed, a minority of patients fail to improve in spite of therapeutic efforts,[26,29,30,34,35] as in the cases of Master C and Miss D. These patients, referred to in the literature as "lost cases," are only revealed when no positive response to considerable attempts at management can be elicited.[19] Historically, red filter treatment had a non-responder rate of 13% to 53%,[26,27,37] with one study reporting that 80% of cases did not maintain any gain in visual acuity.[38] However, of note is that all of these studies employed small sample sizes. Studies involving pleoptics reported a non-responder rate of between 28% and 35%,[18,32] with one study finding no long-term benefit in 45% of patients.[46] Furthermore, 10% to 33% of subjects undergoing direct occlusion, either with or without a preparatory period of inverse occlusion, failed to achieve central fixation.[17,35,37] Inverse occlusion was found to be ineffective and not recommended for young children[30,35] and it has even been suggested that it represents a waste of valuable time, detracting from the critical treatment period.[17] Comparison of treatment outcomes is complicated by the lack of definition of the eccentric fixation and the vague and varied definitions of cure or improvement. However, analysis of the reported cases showed that those with peripheral, steady eccentric fixation were the least likely to improve.[17,18,28,32] Herein, in apparent non-responders the dilemma lies in determining whether further treatment would prove futile and thus a clinical decision about when to discontinue treatment must be made. In such instances, an investigation as to the factors affecting treatment outcome is warranted.

The restoration of central fixation and success of amblyopia treatment is contingent upon several factors. Final visual outcome is dependent upon the type of fixation present, with central wandering fixation holding a better functional prognosis than steady, well-entrenched eccentric fixation.[19,34,38] Visual acuity potential is greater if fixation is nearer the fovea,[47] with the hypothesis that occlusion improves the amblyopia component but that residual visual

acuity is dependent on the eccentric retinal point.[7] The severity of amblyopia can also dictate treatment success, with well-established amblyopia and those with poor visual acuity at diagnosis often proving more treatment-resistant.[5,7,48,49] Best-corrected vision of less than 6/12 at the time of initial treatment and a difference in visual acuity between the two eyes of four or more lines have been identified as risk factors for treatment failure.[48] The degree of anisometropia is also highly significant in predicting final visual outcome in patients, with higher degrees of anisometropia having a negative effect on treatment outcome.[4,7,48,49] Stewart et al found that children with eccentric fixation responded significantly less to refractive correction, and that those with severe mixed amblyopia and eccentric fixation had significantly greater residual amblyopia after six hours per day of occlusion therapy.[7] Owing to each of the above factors, the success of treatment in both of the current patients may have been compromised.

The success of treatment is not only limited to factors pertaining to the patient's visual status, but extends to include more readily modifiable influences. A long interval between the onset of squint and the implementation of effective treatment can negatively affect prognosis.[16,28] Thus, prompt diagnosis and early treatment is imperative.[34] In addition, non-compliance is a known and frequently reported influence on treatment outcome, with poor compliance lending to a reduction in treatment success.[7,48] Miss D in particular was diagnosed much later and demonstrated reduced compliance which could have contributed, at least in part, to poor treatment outcome. The reasons for non-compliance are infinitely broad but poor parental cooperation is often a key factor underlying non-compliance in children.[19] Thus, it is recommended that a full and detailed explanation of the importance and reasoning behind treatment be consistently issued to parents in an effort to gain their cooperation and understanding if therapy is to be effective.[28,48]

In reviewing the given cases, Master C was compliant with occlusion over a ten-month period and has certainly received the optimal dose of occlusion with minimal change. It would appear that surgery is the next option and atropine penalisation could be attempted as this would avoid the psycho-social problems now in evidence. In the same way Miss D, who has never been compliant is at least continuing with atropine as maintenance occlusion. Given that they are both non-responsive to occlusion, this level of maintenance occlusion may give some benefit with minimal side-effects during the sensitive period.

Indeed, eccentric fixation remains an area where knowledge is limited and further research is required in order to understand its mechanisms and why this sub-group of patients fail to improve despite concerted treatment efforts. The authors hypothesise that there may be some sub-clinical foveal pathology present in these patients which drives the incentive for them to fixate eccentrically. However, evidence to support this theory has not yet been found.

CONCLUSION

Whilst eccentric fixation in accompaniment with amblyopia is not uncommon, the pathogenesis of this fixation anomaly remains unclear. A myriad of treatments have been implemented in the past, each carrying specific limitations and different levels of efficacy. Whilst the treatment methods of choice used today are often effective in restoring central fixation and ameliorating amblyopia, they do not guarantee success in every patient. Ultimate success is contingent upon many factors, some of which are known and can be easily identified in a given case, and others which may not be immediately apparent. The latter makes the decision about when to cease treatment difficult and a clinical judgement must be made about whether maximum vision is likely to have been achieved and thus any continued treatment futile. Until such a time when more is known, in patients who fail to respond to conventional treatment, the clinician can at best conduct a thorough examination into the factors potentially impinging upon prognosis; be it non-compliance, well-established eccentric fixation or otherwise. These factors can then help guide the clinician in making an appropriate decision about when to cease treatment. Indeed, further research in this field is necessary and encouraged; as currently it still stands to represent an area about which relatively little is known.

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Named Lectures, Prizes and Awards of Orthoptics Australia

THE PATRICIA LANCE LECTURE

1988	Elaine Cornell	Home exercises in orthoptic treatment
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	The Glaucoma Inheritance Study in Tasmania (GIST)
1997	Robin Wilkinson	Heredity and strabismus
1998	Pierre Elmurr	The visual system and sports perfomance
1999	Kerry Fitzmaurice	Research: A journey of innovation or rediscovery?
2005	Kathryn Rose	The Sydney Myopia Study: Implications for evidence based practice and public health
2006	Frank Martin	Reading difficulties in children – evidence base in relation to aetiology and management
2008	Stephen Vale	A vision for orthoptics: An outsider's perspective
2009	Michael Coote	An eye on the future
2010	John Crompton	The pupil: More than the aperture of the iris diaphragm

THE EMMIE RUSSELL PRIZE

1957	Margaret Kirkland	Aspects of vertical deviation
1959	Marion Carroll	Monocular stimulation in the treatment of amblyopia exanopsia
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 convergent 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 Cort	Nystagmus blocking syndrome
1980	Sandra Tait	Foveal abnormalities in ametropic amblyopia
1981	Anne Fitzgerald	Assessment of visual field anomalies using the visually evoked response
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