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The paper should be organised into the following parts in this sequence: INTRODUCTION; METHOD; RESULTS; DISCUSSION; ACKNOWLEDGEMENTS; REFERENCES; TABLES; LEGENDS FOR FIGURES; FIGURES.

INTRODUCTION: Includes the purpose of the paper but does not include results or conclusions.

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CLOSING DATE

Papers for publication in the Australian Orthoptic Journal may be submitted to the Editor at any time up to 1st OCTOBER in the year prior to the next edition. This date may be extended on request to 31st October, providing an abstract of the proposed paper is received by the Editor before 1st October.

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EARLY DETECTION OF REDUCED VISION IN PRE SCHOOL CHILDREN IN AUSTRALIA — THE OAA PUBLIC RELATIONS CAMPAIGN

ANNE FITZGERALD, DipAppSci(Orth), MPH(Syd)

This year has presented the OAA with a number of challenges, not the least of which has been the proposed launch of the Association's public relations campaign.

Over the years we have all commented on the lack of public awareness of our profession despite the high clinical and research standards achieved by our members. As a profession we have been encouraged to address this situation both from within our own ranks and from outside but to date we have not succeeded with this task.

In recent months the OAA Council have discovered that increasing public awareness of orthoptics is more easily proposed than achieved. To make a start the Council appointed a public relations consultant, Mr Mike Lynskey and formulated an OAA Public Relations Committee.

With guidance from Mr Lynskey, it quickly became apparent to all concerned that it was not going to be possible to launch a public relations campaign without having a platform from which to launch it.

Thus, with the agreement of the Council, the Public Relations Committee have decided that the platform will be a nationwide study of the incidence of reduced vision in pre school children aged three years in Australia.

Amblyopia has been recognised for many hundreds of years yet, despite this, no accurate figures are available on the incidence of amblyopia in pre school children in Australia. In the 1989 Australian Census there were 277 000[1] pre school three year old children in this country yet we do not know their visual status.

The first recorded cases of amblyopia stem from the 16th century. In 1713 the first accurate clinical description emerged (from Le Cat[2]) and it was in 1742 that George Louis Leclerc, Comte de Buffon[3] was the first to document full time occlusion treatment for amblyopia.

Partial or part time occlusion in amblyopia treatment was first suggested by Erasmus Darwin,[4] the physician grandfather of Charles Darwin, in 1801.

In 1833 MacKenzie[5] recognised that part time occlusion together with close work was more beneficial than full time occlusions alone. Thus the place of accommodation in amblyopia treatment entered the debate.

We now know that the most intractable form of amblyopia is that suffered by congenital unilateral cataract patients, even when they are operated on the first weeks of life. However, the role of accommodation in amblyopia is still not fully understood.

The theories of the optimal form of treatment for amblyopia have changed over the years. For example, in the late 1800's and early 1900's, occlusion therapy fell out of favour with a group of influential ophthalmologists. They felt certain that amblyopia was a congenital defect that would not be affected by occlusion.[6,7,8,9]

Some authors suggested that occlusion therapy was simply unnecessary torture (Poulard[8] 1921) while others (Gifford and Bangeter) suggested

that occlusion caused physiological trauma such as stuttering.[9,10] (In a later publication in 1962 Bangeter[11] modified his view).

Amblyopia treatments have included pleoptics, penalisation, red filters and, more recently the Cam stimulator but, occlusion therapy has survived them all. Currently the debate at most conferences is not whether or not to use occlusion but rather whether or not it should be used on a full time basis.

A most recent study by Carolyn Calcutt, the OAA 1992 Patricia Lance Lecturer, has broadened the debate on occlusion yet again.[13] Her research has demonstrated that in countries where the commencement of education and thus the need for the use of accommodation for sustained periods of time is delayed until the end of the critical period, amblyopia is not likely to develop in infantile esotropes if they are NOT treated.

Our knowledge of physiology and thus our understanding of the basis of amblyopia have been greatly expanded by the work of researchers including Hubel and Wiesel, Ikeda and Blakemore.

Through their work we know that there are changes in the retina, LGN and the visual cortex as a result of deprivation.

However, this knowledge is constantly being challenged. In a publication in 1988, Colin Blakemore[14] clearly demonstrated that shifts in cortical ocular dominance columns sometimes showed no correlation with the degree of amblyopia. He reported that he could find no equivocal reason why a decrease in the area innovated by the deprived eye in layer IVc of the cortex or even the gross shift in the cortical ocular dominance column width should necessarily lead to the decrease in visual acuity found in the amblyopic eye.

Our knowledge of human amblyopia is also continually expanding through the use of new techniques for neurophysiological investigation of visual processing in the brain. Using positron emission tomography (PET), researchers have demonstrated that visual stimulation of eyes with strabismic or anisometropic amblyopia induces very low levels of glucose metabolism in the visual cortex compared to levels produced by non amblyopic eyes.[15] This provides another explanation for the reduction in the processing of visual information from the amblyopic eye.

With the advent of contrast sensitivity assessment and similar methods of assessment of visual function, we have been far better able to investigate and monitor amblyopia on a clinical level in humans.

Thanks to the work of clinicians like Prof Awaya, the OAA Guest Lecturer, and others we now know about the critical period for the development of human vision and of the vital importance of early detection and intervention in the treatment of amblyopia in children in developed countries.[16,17,18]

Today there is a mountain of literature which shows the effectiveness of occlusion therapy in the treatment of amblyopia. These studies have been subject to rigourous statistical analysis and they have shown without doubt that early intervention with this simple and non invasive therapy, preferably by the age of three, is far more effective than late intervention. By the age of eight amblyopia is virtually an incurable entity leaving the patient with unnecessary loss of vision in one and sometimes both eyes.

With the scientific and medical knowledge that we now have it is totally unacceptable that visual loss should remain undetected in any Australian child until such time as the child is routinely screened at school. By then valuable and irretreivable time for the treatment of amblyopia has been lost.

To convey this message to government, parents and the community as a whole the OAA need statistics. To this end the Public Relations Committee have proposed to Council that the Association embark on a research project encouraging all orthoptists in Australia to test pre school children attending Long Day Care Centres which are administered by the Commonwealth Department of Health, Housing and Community Services. It was considered that these centres should provide a broad socio economic cross section of the Australian population.

The statistics gathered by OAA members will demonstrate the incidence of visual loss, most

of which would otherwise have gone undetected until school age or later.

From this the OAA can lobby the Federal and State Governments to put strategies into place with the aim of early detection and treatment of amblyopia. It is envisaged that, with the help of Mr Lynskey, the research project results will be used to raise media attention to our profession.

To supplement this Orthoptists will also be asked to speak to their local community groups about amblyopia and the need for early intervention.

The committee aims to carry out the project in the first 6 months of 1993. Results will then be available for the beginning of the OAA's 50th anniversary celebrations at next years conference in Hobart. That conference will mark the launch of the public relations campaign.

It is only with media coverage based on sound research that our profession will attract the attention of the public and thus our share of the limited purse of public funds needed for future research and development.

In order for our profession to thrive into the next century we must continue to conduct research and we would be well advised to heed the following quotation from a 9th century philosopher;

> *"He who does not doubt, does not investigate, and he who does not investigate does not perceive, and he who does not perceive remains in blindness and error".*
>
> *Al-Ghazali (1058-1111)*

However, in 1992 it is not sufficient just to doubt, investigate and perceive, it is equally importantly to increase our public profile through the media and community groups and to this end each and every orthoptist must participate.

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ASSOCIATE PROFESSOR IN ORTHOPTICS — LA TROBE UNIVERSITY

In 1988, Alison Pitt, MEd(Melb), DBO(T), was appointed a Reader at La Trobe University. In 1992, the University instituted the title of Associate Professor for some academic staff at Reader level. This title has now been conferred on Alison Pitt. At the end of 1992, she was appointed as Chairperson for a further two years until 1995.

Alison completed her Diploma of British Orthoptics at the Birmingham and Midland Eye Hospital in 1972 and following a short period as locum orthoptist in Bradford worked as sole orthoptist in North Wales where she took responsibility for Orthoptic services across the whole of North Wales. After this, Alison took up a research orthoptist position in Uppsala, Sweden. She returned to undertake the Orthoptic Teachers' course which she completed in 1979 at the Coventry School of Orthoptics, which was then the highest orthoptic qualification offered in the UK. Alison worked as a teacher in Coventry for five years. During which time she was appointed as an examiner to the Orthoptic Board of the Council for Professions Supplementary to Medicine and worked on the National Orthoptic Curriculum Committee. In 1982, Alison was also appointed to the Coventry Bench as a lay magistrate and in her spare time took part in a programme for teenagers in institutional care.

In 1985 Alison came to Melbourne to take up the position as Head of School at the then Lincoln Institute of Health Sciences.

Since arriving in Australia, Alison has completed a Masters degree in Education at Melbourne University in 1990. In 1989, she was invited to give the Patrica Lance lecture, titled "Accommodation Deficits in a Group of Young Offenders". This paper demonstrated not only her interest in Orthoptics but also in adolescents from her previous work in the UK.

In addition to her position as Chairperson of the Division of Orthoptics, Alison teaches her speciality being the sensory aspects of ocular motility dysfunction. She has successfully developed the degree course in Orthoptics and has overseen the first year of the Honours programme. She is currently supervising five Masters students and the research projects of a number of undergraduate students. Alison has developed the research profile of the Division of Orthoptics not only by successful research grant applications but by actively developing academic staff research interests.

Within the School of Health Sciences, Alison works to develop overseas links and courses for all the disciplines, and is currently working on a proposal to begin Orthoptic training at the SNDT Women's University in Bombay, India.

Alison has also served the Orthoptic profession by being the President of the Victorian Branch of the OAA and has represented Victoria on both the Federal Council of the OAA and the OBA, including a period as chairman of the Examination Sub-Committee of the Orthoptic Board.

Alison is a most appropriate recipient of the title Associate Professor and the profession is very proud that she joins Elaine Cornell as one of the two Associate Professors of Orthoptics in Australia.

Shayne Brown

Australian Orthoptic Journal 1993; **29:** 1-7

The Patricia Lance Lecture, Sydney 1992

UNTREATED EARLY ONSET ESOTROPIA IN THE VISUAL ADULT

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Abstract

One hundred and forty two patients with a history of esotropia present before the age of six months who had had no previous ophthalmic examination, and who were aged eight years or more were examined in order to ascertain the presence or absence of DVD, manifest latent nystagmus and asymmetric OKN, the main features of essential infantile esotropia (ET).

Twenty nine patients showed no evidence of essential infantile esotropia, despite the history of onset of strabismus under the age of six months.

One hundred and thirteen cases were diagnosed as having essential infantile esotropia. There were 90 patients with manifest latent nystagmus, 67 with DVD and 97 with an asymmetric OKN response. Only eight cases had visual acuity in the non-fixing eye of less than 6/12. 'A' pattern esotropia was present in 72 cases and 'V' pattern elicited in 16 patients. There were no abnormal head postures.

It is suggested that the untreated condition of essential infantile esotropia should be studied before making definitive conclusions about the characteristics of the condition.

Key words: *'A' pattern, amblyopia, manifest latent nystagmus, DVD, asymmetric OKN.*

INTRODUCTION

Concomitant convergent strabismus with a reported onset before the age of six months is a frequently researched, well documented and easily identified phenomenon. The literature is full of erudite communications on the subject, investigating all aspects of the aetiology, and detailing the various manifestations and their implications for surgical and non-surgical management. The terminology has changed since it has been shown that the condition is not present from birth. The term congenital is discouraged and the terms early onset and infantile esotropia are variously used in the literature. In an attempt to further clarify the situation, von Noorden[1] has detailed the various types of early onset esotropia and suggested the preposition of 'essential' before infantile esotropia to describe those patients who conform to the syndrome described by Kommerell,[2] where a large esotropia is associated with manifest latent nystagmus and monocular opto-kinetic asymmetry. The presence of DVD is accepted as a further main feature of the syndrome. Previous clinical studies have concerned either neonates or infants in developed countries who have been documented from the age of presentation in the early days of their life, until their discharge as visual adults. These prospective studies have not taken into account several important factors; firstly that detailed assessment of eye movements in early infancy is not always possible; secondly that current methods of estimating the presence or absence of amblyopia in babies are less than accurate

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and finally whether the treatment which has been carried out has had any influence on the natural history of the condition.

METHODS AND MATERIALS

Between October 1986 and October 1989, all new patients referred to Groote Schuur Hospital in Cape Town and St John's Eye Hospital, Baragwanath with concomitant esotropia which was said to have been present before the age of six months, who had had no previous ophthalmic examination and who were aged eight years or more, were entered into the study. Also included were patients seen during a tour of the Sightsavers Team of the Bureau for the Prevention of Blindness to the homelands of Transkei and Kwazulu. A full orthoptic examination including cover test and ocular movements, fixation preference assessment, and prism cover test for near and distance, and on up and down gaze in order to determine the presence of 'A' and 'V' patterns, together with special reference to testing for manifest latent nystagmus, DVD, and asymmetric optokinetic nystagmus. Any abnormal head posture was noted. OKN was tested with a hand held drum which was rotated at 20 cycles per minute at approximately 30 centimetres from the patient. A visuscope was used to check fixation and to elicit manifest latent nystagmus if it had not been seen on cover testing, and a synoptophore was used to search for DVD if distance cover test with Spielmann occluder had failed to reveal its presence. Each patient was refracted following the instillation of cyclopentolate drops, and the media and fundi were examined. Where possible the patient was checked by a second coworker, and random sampling was undertaken to ensure continuity of test standards. Subsequently patients were checked again when they attended for surgery and post-operative data was obtained. All follow-up information on the patients was checked in August 1992.

RESULTS

One hundred and forty two patients fulfilled the criteria and were included in the series. There were 106 patients of African origin, 34 of mixed race, one Asian and one Caucasian.

The mean angle of deviation was 49.4 dioptres.

One hundred and thirteen (79.6%) patients showed one or more signs of essential infantile esotropia in that manifest latent nystagmus, DVD or asymmetric OKN had been elicited in the presence of a large esotropia of onset before six months of age.

Twenty nine (20.4%) cases reported a history of esotropia before the age of six months, but had none of the signs associated with essential infantile ET.

The incidence of amblyopia of more than two lines on the Snellen chart was 19.7% (28 cases).

The overall incidence of DVD was 47.1% (67 patients), of manifest latent nystagmus, 63.4% (90 cases) and asymmetric OKN was 68.3% (97 cases).

Seven and three-quarter per cent (11 patients) had an anomalous OKN response and large esotropia without manifest latent nystagmus or DVD.

'A' pattern esotropia occurred in 57% (81 cases) and 'V' pattern in 14.8% (21 patients).

No patient was reported to have an abnormal head posture, and there were no underactions of the lateral recti of more than minus one when assessed on a scale of one to four.

For further analysis the patients were then divided into two groups; Group A for those with one or more signs of essential infantile esotropia.

Group B for patients with other types of early onset esotropia. It cannot be assumed that the history was inaccurate in the 29 cases in Group B with no sign of essential infantile esotropia, and normal OKN. However, these patients did not fall into the well defined categories described by von Noorden[1] i.e. nystagmus blocking syndrome, VI nerve palsy, accommodative esotropia, infantile esotropia with CNS disorders, infantile esotropia with sensory disorders, Duane's retraction syndrome and Moebius syndrome. We suspect that they form a further group of early onset esotropes whose deviation occurs before the age of six months but after the maturation of the nasotemporal OKN response.

GROUP A

One hundred and thirteen cases with one or more signs of essential infantile ET were identified, of whom 90 were of African origin, and 21 of mixed race. There was one Asian and one Caucasian. The mean angle of deviation was 49.8 dioptres.

Latent Nystagmus, Asymmetric OKN and DVD

Ninety seven (85.8%) patients demonstrated an asymmetric OKN response, 67 (59.2%) showed DVD and in 90 (79.6%) latent nystagmus was elicited. Forty seven (41.5%) cases showed DVD, latent nystagmus and asymmetric OKN. Eighty (70.7%) patients with anomalous OKN were shown to have latent nystagmus, and 53 (47%) had DVD.

Fifty four (47.8%) patients had latent nystagmus and DVD but only eight of these (7.1%) cases were judged to have normal OKN responses. Eleven cases showed asymmetric OKN and esotropia without other signs of essential infantile ET.

Amblyopia and Fixation Preference

Fifty six (49.6%) patients showed no fixation preference and free alternation at first assessment.

Of the 57 (50.4%) cases with fixation preference, 22 (38.6%) had a difference in the visual acuity between the two eyes of more than two lines on the Snellen chart, but 49 (86%) of the 57 achieved vision in the non-preferred eye of 6/12 or better. There was significant unilateral refractive error in five of the eight amblyopic patients.

'A' and 'V' Patterns

Seventy two (63.7%) cases had 'A' pattern esotropia and 16 (14.1%) patients had a 'V' pattern.

GROUP B

Twenty nine cases were found to have no sign of essential infantile ET. Eight (27.5%) patients had amblyopia of more than two lines on the Snellen chart, but in only one case was there a relevant unilateral refractive error.

Nine (31%) cases demonstrated 'A' pattern ET and five (17.2%) had 'V' pattern ET.

DISCUSSION

Prospective studies of early onset esotropia in developed countries have a major advantage over a retrospective study such as this, in that the medical diagnosis is generally made very early in life and corroborated with photographic evidence. However, the great difficulty in accurate assessment of the young infant, and the natural desire to re-align the visual axes, at the earliest opportunity, for both cosmetic purposes and the possibility of obtaining some rudimentary post-operative binocularity, may well have obscured the natural history of the early onset esotropia.

Amblyopia and Fixation Preference

The original hypothesis was that children with untreated infantile esotropia are less likely to become amblyopic than their contemporaries who have early surgery. This is completely at odds with all the current literature on infantile esotropia. Von Noorden[1] describes amblyopia as a characteristic of essential infantile esotropia with an incidence of 35%, Hiles[3] suggests that up to 75% of the cases may need occlusion; Pratt-Johnson and Tillson[4] report an incidence of amblyopia of 50%, with 30% having visual acuity of less than 6/12 in the strabismic eye; Walker and Taylor[5] found 45% of their patients still amblyopic at final assessment and Dickey and Scott[6] found more than two lines difference in the visual acuity of 51% of their patients. Few authors specify, however, whether there was amblyopia before therapy commenced, or on what basis the diagnosis of amblyopia in infancy was made. The only paper which addresses the subject of pre- and post-operative amblyopia is that of Hoyt[7] who compared visual acuity before and after surgery and found only four (12.9%) patients out of 31 to have been amblyopic pre-operatively but that 19 (61%) developed amblyopia within the year after operation, thus suggesting that the post-operative cases were considerably more at risk of amblyopia than the untreated cases. In our study the incidence of amblyopia with a visual acuity in the fixing eye of less than 6/12 was only 7% in the untreated essential infantile esotropes. Therefore the un-

treated cases had a significantly lower incidence of amblyopia than their treated counterparts. Hoyt[8] has recently confirmed these findings in a series of untreated infantile esotropes who are mainly of Asian origin, unlike those in our series who are mainly African. This refutes the suggestion that the racial basis in our series affects the conclusions and confirms that the low incidence of amblyopia is characteristic of the condition.

However, some of the patients in the study did become amblyopic and the reason for this has to be investigated. The original examination noted in all cases not just the visual acuity but also the fixation preference, since this is the method most commonly used in the diagnosis of amblyopia in the pre-verbal infant. A study of the 113 patients with essential infantile ET reveals that although 56 patients showed free alternation, 11 of these patients did have a difference between the visual acuity of the two eyes, although in each case the visual acuity of the less good eye was more than 6/9.

Of the 57 cases with a fixation preference, 24 had equal visual acuity or visual acuity of 6/6 or more in the non-fixing eye, and a further 14 achieved 6/9 in the strabismic eye.

Thus 19% of the alternators had an element of amblyopia and 66% of the cases with fixation preference had little or no amblyopia. This would suggest that fixation preference is not a reliable method of diagnosing amblyopia, and may well account for the seemingly high incidence of amblyopia suggested in studies conducted in infancy.

Nineteen patients failed to see 6/9 with the non-fixing eye, but 11 cases did achieve 6/12, and in six (54.5%) of the patients there was a significant degree of anisometropia.

There were eight patients with dense amblyopia ranging from 6/18 to light perception. Five (62.5%) cases had significant anisometropia, but there was no correlation between the amount of anisometropia and the depth of the amblyopia. There was significant astigmatic error in the strabismic eye in four of these patients.

Four patients had bilateral amblyopia due to bilateral uncorrected refractive error.

Uncorrected refractive error was therefore the cause of the amblyopia in 65% of the cases who achieved 6/12 or less in the non-preferred eye. The amblyopia in these patients would therefore appear to be of refractive rather than strabismic origin. It has been suggested that the patients with amblyopia in the absence of refractive error, may well have had an episode of infection or trauma during the sensitive period which could have resulted in stimulus deprivation amblyopia.

The important fact is, that out of 113 essential infantile esotropes, only 19 (16.8%) failed to achieve visual acuity of 6/9 in the squinting eye and only eight (7%) registered vision of less than 6/12 in the deviating eye.

Manifest Latent Nystagmus and DVD

The presence of latent or manifest latent nystagmus and DVD have long been recognised as integral elements of essential infantile ET. Lang[9] has suggested that the dominant feature of the infantile ET syndrome is latent nystagmus and Kommerell[2] and von Noorden[1] agree that it is a main feature of the condition. It is thought to originate as manifest nystagmus, often rotary in nature, and become latent with the passage of time, hence the manifest latent description. In this series there was an incidence of manifest latent nystagmus of 79.6%. Helveston[10] has suggested that manifest latent nystagmus is invariably found in conjunction with DVD, while Harcourt[11] describes an incidence of 73%. This study revealed DVD in association with manifest latent nystagmus in only 47.8% of the cases. This was due to a relatively low incidence of DVD in this group of patients, only 59.2%. It is possible that this low incidence is due to the difficulties of eliciting subtle amounts of DVD in the presence of very large esodeviations. DVD has been specifically searched for post-operatively where it was absent prior to surgery and four cases have been found. However, 18 patients with essential infantile ET without DVD did not attend for surgery or follow-up appointment.

The age at which DVD occurs is said to be between the ages of 18 months and three years, and it would be reasonable to surmise that many patients will have had surgery by this juncture. It is impossible to say whether the four patients

in our series who demonstrated DVD post-surgery did so as a reaction to the operation, or whether the removal of the gross esotropia revealed a pre-existing DVD. Parks[12] has suggested that DVD may diminish or disappear before the age of eight years. This could be an explanation for the low incidence in this series, although the suggestion has been refuted by most other authors.

Asymmetric OKN

The asymmetric OKN response in essential infantile ET has become accepted as an integral aspect of the condition. The aetiology is far from clear. Fitzgerald and Billson[13] found a high correlation between abnormalities of VERs suggestive of abnormal visual pathway projection and anomalous nasotemporal OKN. They suggested that patients with asymmetric OKN might be at risk of developing DVD. Atkinson,[14] amongst others has found that infants of two to three months of age demonstrate asymmetric OKN, which would suggest that the anomalous OKN response is a consequence of the strabismus disrupting the normal development of the pursuit system, rather than of a primary structural defect of the visual pathways. Nevertheless, as Demer and von Noorden[15] have shown, the presence of an asymmetric OKN response suggests an 85% chance that the onset of the strabismus was before the age of six months. We found an 85.8% incidence of asymmetric OKN in our study, which would appear to confirm the accuracy of the history as well as emphasising the importance of this phenomenon in essential infantile ET. With an incidence of only 59.2% of DVD, and only a 47% incidence associated with asymmetric OKN, it was not possible to confirm a relationship between asymmetric OKN and DVD. This study of the natural history seems to confirm that manifest latent nystagmus (79.6% incidence) and asymmetric OKN (85.8% incidence) are the two main features of essential infantile ET but that the signs of manifest latent nystagmus, DVD and asymmetric OKN, while frequently present, are not invariably linked.

'A' and 'V' Patterns

The association of 'A' pattern with essential infantile ET was first noted by Lang,[16] and the prevalence of an 'A' phenomenon has been reiterated on many occasions by Mein,[17] although most authors persist in attributing the upshoot in adduction seen in these patients to primary overaction of the inferior oblique. von Noorden[1] cited an incidence of 68% of inferior oblique overaction in patients examined during infancy, while Hiles[18] found 78% of his patients with overaction of one or both obliques. We found an incidence of 63.7% of 'A' patterns compared with an incidence of 14.1% of 'V' patterns. These figures correlate well with those of Harcourt and Mein[19] who cite an incidence of 60.4% of 'A' patterns and 25.5% of 'V' patterns in patients with DVD. It seems likely that in those series citing a high incidence of inferior oblique overaction and 'V' patterns, the upshoot on adduction which occurs with DVD has been attributed to a primary overaction of the inferior oblique muscle, and that the diagnosis of 'V' pattern has been made without comparison of the cover test, or prism cover test measurements, on up and down gaze. It should be noted therefore that an upshoot on adduction is not necessarily an inferior oblique overaction, but may be the first manifestation of DVD, and 'A' and 'V' patterns diagnosed only when accurate assessment of the esotropia on up and down gaze can be made. The fact that the upshoot in adduction may be elicited at an early age may necessitate also reconsideration of the age at which DVD occurs. It is unlikely that the high incidence of 'A' patterns in this group of essential infantile esotropes is due to the 74.6% incidence of patients of African origin. We have not been able to elicit clinical evidence to support this hypothesis, and it is likely that there could be wide variations in the types and manifestations of strabismus in Africa. The IOA survey in 1983 suggested a higher incidence of esodeviations in patients of African origin, but while in southern Africa there does appear to be a prevalence of esotropia, patients originating from other areas often present with large exodeviations, with associated 'V' patterns.

Compensatory Head Posture

Harcourt and Mein[18] report an incidence of compensatory head posture in essential infantile

ET of 75%. It is thought to be motivated either by the attempt to fix in adduction to obtain the best visual acuity by minimising the nystagmus, or possibly to compensate for the restriction of abduction, or DVD. It is frequently of variable nature and is often seen only on testing the visual acuity. We found no patient with a compensatory head posture. The only possible explanation is that whilst the majority of our patients were literate, education probably commenced at a much later age than in the developed countries, and there would have been much later exposure to TV and reading matter. It is possible that in these patients the manifest component of their manifest latent nystagmus had disappeared long before they needed accurate vision.

Post-Operative Results

Of the complete group of 142 patients a total of 35.2% did not attend for surgery, and in the essential infantile ET group this rose to 37.2% (42 cases). This may be attributed to the fact that in this patient group cosmesis is not a problem and that the family may have been reassured with regard to the vision at the first visit. Of the patients who did consent to operation, a satisfactory cosmetic result was claimed in all cases. The final angle of deviation varied from 10 dioptres of exotropia to 25 dioptres of esotropia, the majority falling within the five to 15 dioptre range. No patient complained of diplopia following operation. No patient achieved binocular vision following surgery. The incidence of binocularity in patients who have had early surgery is low, and although there have been reports in the literature of adults with long-standing esotropia achieving binocularity with so called peripheral fusion, and monofixation syndrome following surgery for strabismus as adults, it is very unlikely that any worthwhile binocular vision could be elicited in patients who have squinted since the first few months of life.

Whilst accepting that the patients in this group, by having late surgery, have foregone any chance of developing binocularity, it has to be remembered that the underprivileged child or the child in Third World with essential infantile esotropia is being put at risk of developing amblyopia if operated early, and improving cosmesis is of secondary importance to the retention of good visual acuity in each eye.

CONCLUSIONS

The cases of essential infantile ET who remained untreated until visual adulthood demonstrated a high incidence of manifest latent nystagmus and asymmetric OKN, which appear to confirm these phenomena as the characteristic features of the condition. The correlation between manifest latent nystagmus and DVD of more than 70% is not confirmed by this study. Examination of post-operative data has shown that subtle amounts of DVD have been elicited after surgery but the high failure rate of patients in attending for operation means that conclusions cannot be drawn over the appearance of DVD post-operatively. It is not possible to hypothesise as to whether, in some cases, the DVD had been present in the past and had disappeared before the initial examination. The reported age of onset of DVD may well have to be reconsidered if the upshoot on adduction which has been attributed to inferior oblique overaction in infancy, can be proved to be due to the first manifestation of the DVD.

This series has demonstrated clearly that amblyopia is not a characteristic of essential infantile ET. The amblyopia which has occurred in this series has been the result of uncorrected refractive error in over 65% of the cases. It is possible that, with a population such as we have studied, there may well be an element of stimulus deprivation amblyopia in some of the amplyopes without refractive error. The amblyopia which occurs in the treated cases is a direct result of the surgical intervention.

The incidence of 'A' patterns in 63.7% compared with 'V' patterns in only 14.1% confirms that an 'A' pattern is an important feature of the condition and re-emphasises the fact that the upshoot in adduction should not be attributed to inferior oblique overaction, and a 'V' pattern should be diagnosed only when confirmed by measurements in up and down gaze.

The absence of a compensatory head posture and lateral rectus weakness are interesting. The

head posture described by other authors is invariably associated with visual tasks; it is possible that a lack of specific visual stimulation at an early age has resulted in its non-appearance.

Finally it is to be hoped that this study will have been seen to emphasise that the management of essential infantile ET in the developed countries which is based on the necessity of removing the cosmetic aberration as soon as viable, should be modified when treating patients in the developing countries, where access to medical facilities is limited. For the underprivileged, cosmesis may not be a problem and retention of good visual acuity in both eyes, is the first priority. This study has shown that patients who can demonstrate one or more characteristics of essential infantile ET, have an alternating esotropia or equal visual acuity and absence of unilateral or high bilateral refractive error are at very low risk of developing amblyopia if left untreated. Whilst they may not achieve binocular single vision if the surgery is carried out later in life there is a low risk of diplopia.

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VISUAL AGNOSIA — AN UPDATE ON DISORDERS OF VISUAL RECOGNITION

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Emmie Russell Prize 1992

Abstract

Normally, we can recognise objects around us at a glance. However, selective brain damage can cause visual agnosia. Patients with this disorder are unable to recognise familiar objects, despite normal visual acuity. Although they can see well enough to accurately describe parts of the object, they cannot recognise what the object is or what it may be used for. What is more puzzling is that, when patients are allowed to hold the object or hear its characteristic sound, they can often identify it immediately. The study of this disorder has led to considerable progress in our understanding of the various ways visual processing can break down. A review is presented of the different types of visual agnosia which can occur, and implications for normal visual object recognition.

Key words: *Visual object recognition, apperceptive agnosia, associative agnosia.*

INTRODUCTION

Visual object recognition appears to be a natural and effortless achievement. Normally, we can "apprehend the meaning of objects, our prior associations with them, and their uses, from vision" with no difficulty at all.[1] However, damage to the posterior regions of the brain can cause visual agnosia. Visual agnosia is a recognition disorder that is not due to a primary sensory impairment, language deficit or intellectual deterioration.[2] Essentially this refers to patients who can still *see* objects but are unable to recognise what they are. When patients are allowed to feel the object or hear its characteristic sound they can usually identify it immediately.

Visual agnosia has been surrounded by much scepticism and controversy. For many years investigators argued that visual agnosia did not exist. For example, Bender and Feldman[3] claimed that all reported cases of visual agnosia could be explained by visual field defects and/or mental deterioration. However, current research indicates that visual agnosia not only exists, but is a complex disorder caused by impairment to different stages of visual object recognition.[1]

Although significant advances have occurred in our understanding of how visual recognition breaks down, there is often confusion in its clinical assessment and diagnosis. Patients with visual recognition disturbances typically present with vague visual problems following injury to the brain. For example, patients may complain that "everything is slightly out of focus" or simply "I can't see". These patients are referred to the orthoptist and the ophthalmologist for visual assessment. To establish the diagnosis of visual agnosia it is critical to rule out the existence of any sensory impairment. Therefore the orthoptist has a role in the assessment of visual agnosia and should be aware of why and how this disorder occurs.

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The aim of this paper is to review two potential explanations of visual agnosia: the traditional model and the more recent multistage model. This review will focus on how the underlying processes of normal visual object recognition can breakdown to cause visual agnosia.

TRADITIONAL MODEL OF VISUAL AGNOSIA

The earliest account of visual agnosia was proposed by Lissaeur in 1890.[4] He proposed that two different stages are necessary for object recognition: apperceptive and associative. The apperceptive stage involves deriving conscious awareness of the sensory impression of an object. The associative stage involves establishing the meaning of the object by linking it to previous recollections and experiences.

(1) *Apperceptive agnosia.* Patients with a defect in the apperceptive stage are unable to recognise differences that distinguish two similar objects. They cannot name, match or copy pictures of objects or simple shapes. However, they can identify colours and appreciate changes in light intensity, as well as detect direction of movement.

(2) *Associative agnosia.* Patients with a defect in the associative stage are able to perceive objects reasonably well as they can match and copy objects correctly. However, they are then unable to recognise any of these objects. These patients are considered to have normal perception "that has somehow been stripped of its meaning".[5]

MULTISTAGE MODEL OF VISUAL AGNOSIA

Although Lissauer's model of visual agnosia has remained in use for over a hundred years, it is now regarded as inadequate. The increasing number of reported case studies indicates that the clinical presentation of visual agnosia varies considerably from one patient to another.[6] Therefore it is argued that Lissauer's two stage model conceals the diversity of patients' symptoms and the complexity of underlying processes involved in visual object recognition.

According to Humphreys and Riddoch's model[1,7,8] there are 5 different stages or levels of visual object recognition plus a final 'matching' process:

1. The first stage involves extracting form information, including global object shape and local object features.
2. The next stage utilises the above information to obtain a retinotopic object description, that is, a description coded according to the image on the retina. This allows the geometric features of the object to be "binded" together into a perceptual whole.
3. After integrating all of the above information, a non-retinotopic object description is abstracted. In other words, orientation is assigned to the whole object so that it can be recognised from different viewpoints.
4. The fourth stage is concerned with accessing or using form knowledge, that is, stored information about the typical structure of objects. Essentially, this means the persons memory of an object's shape is matched against the object description obtained from Stage 3.
5. The next stage involves accessing semantic knowledge, that is, a person's knowledge or stored information about the meaning and function of objects.
6. Finally, a 'matching' process occurs between the object description (derived from stages 1, 2 and 3) and the persons memory of that object's shape and function (derived from stages 4 and 5).

According to this multistage model of visual object recognition, visual agnosia can be fractured into various subtypes. Each type of visual agnosia can be explained by an impairment to a particular stage of visual object recognition.

(1) *Impaired shape processing.* This refers to patients who have difficulty recognising objects as well as copying or matching simple shapes. Yet, like patients with associative agnosia, they have intact perception of colour, light and movement. Impaired shape processing is usually caused by multiple lesions in the occipital cortex due to carbon monoxide poisoning.[9] These lesions are thought to produce undetected minute blindspots scattered throughout the patient's visual fields.

(2) *Impaired integration processes or loss of stereoscopic vision.* These patients have intact form information as they can match and copy simple objects correctly. However, they have problems with tasks requiring integrated form information. For example, patients can see the detail of objects clearly but do not recognise the object as a whole. This may be due to an impairment in integrating 2-dimensional shape information or due to the loss of depth perception. It typically occurs following bilateral damage in the posterior regions of the brain.
(3) *Impaired transformation processes.* Patients with this disorder can perform simple matching tasks and identify objects seen from a conventional view, but cannot recognise objects seen from unusual angles. For example, they can easily recognise a bucket from the more conventional side view, but not when it is viewed from directly above. Impaired transformation processes tend to be associated with damage to the parietal lobe.
(4) *Impaired object form knowledge.* Such patients can match and copy objects with no difficulty at all, but nevertheless have problems distinguishing between familiar and novel objects as well as drawing from memory. This occurs because patients can no longer remember what previously familiar objects look like.
(5) *Impaired semantic knowledge.* These patients have no difficulty with visual processing up to this point; they can match and copy objects correctly as well as recognise objects seen from unusual angles. However, they are unable to perform tasks which require classification on the basis of functional characteristics. For example, patients' cannot match semantically related objects such as a light bulb and a candle. This occurs because patients cannot remember the function of the object or any previous associations with the object. Such an impairment tends to occur after bilateral temporal lobe damage.
(6) *Impaired access to semantics.* These patients, like associative agnosics, perform normally on all tests of visual matching and copying as well as on verbal tests of object knowledge. Nevertheless, they still have difficulty recognising objects, especially when surrounded by other structurally and semantically similar objects. For example, patients cannot recognise a horse if it is surrounded by other animals such as a cow or a pig. Problems recalling knowledge about an object's shape and meaning tends to be associated with posterior left hemisphere lesions.

DISCUSSION

This review indicates that disruption to the various stages of visual object recognition can result in many different types of visual agnosia. Clearly the hierarchical nature of normal visual object recognition is reflected in the clinical presentation of this disorder. In its severest form, patients may present as legally blind as they are unable to match, copy or recognise even simple objects. In its mildest form, patients may present as essentially 'normal' on most matching and copying tasks but nevertheless have problems recognising objects in specific situations.

The possibility of impaired shape processing is raised if the patient cannot recognise or match letters, shapes and pictures, demonstrates normal visual acuity using objective measures such as the Catford Drum and Stycar Balls and also has intact colour, light and movement perception. These patients experience significant problems with daily functioning. They cannot recognise even simple objects by vision yet can by other modalities such as touch and sound. The possibility of impaired integration processes is raised if patients have poor stereopsis and no other sensory defect. Such an impairment will cause difficulty with daily functioning as these patients typically adopt a slow feature-by-feature analysis of their environment since they cannot perceive objects as a whole.

An impairment to any of the other stages of visual object recognition is unlikely to be detected during the orthoptic assessment. These higher levels of visual processing are investigated by the neuropsychologist using a range of specific tests. They may involve identifying conventional and unusual photographs of objects (transformation processes), distinguishing between novel and familiar objects (accessing form knowledge), classifying objects according to its functional use (semantic knowledge) and distinguishing between

similar objects (access to semantics). The possibility of disruption to the higher stages of visual object recognition is raised if patients present with complaints of visual problems despite normal visual acuity and intact elementary sensations, as well as a history of occipital, parietal or temporal lobe damage, usually due to a stroke, tumour or head injury. Many of these patients function reasonably well and only experience problems recognising objects in particular situations.

CONCLUSION

The term 'visual agnosia' is a convenient shorthand expression that is used to describe patients who despite normal visual acuity, cannot recognise objects by vision yet can by other modalities such as touch and sound. What is often overlooked is that this term actually refers to a heterogenous group of patients. It is now evident that different types of visual agnosia can occur when different stages of visual object recognition break down. There is no doubt that orthoptists are able to identify patients with an impairment to the early stages of visual recognition concerned with shape processing and integration processes. However, orthoptists should also be aware of the other types of visual agnosia and indicate when higher levels of visual object recognition need further investigation.

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A COMPUTER GENERATED METHOD OF TRAINING ECCENTRIC VIEWING

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Abstract

Visual rehabilitation programs typically require a wide selection of source materials capable of being rapidly accessed and, as required, conveniently interchanged. Computer technology has facilitated the creation, storage and access of data, in both text and graphic image form. Consistent with this, computer technology has the potential to provide an efficient medium for visual rehabilitation programs. A computer program has been developed to assist in training persons with central field loss to eccentrically view. The program has four components: a module to establish the eccentric viewing point at an appropriate angle and degree of eccentricity; an exercise module to reinforce the chosen eccentric viewing position; a module to encourage the application of eccentric viewing technique to visual tasks other than reading including exercises for clients unable to appreciate print material; a module comprising advanced reading material to extend reading skills for appropriate persons. Initial clinical trials indicate that this computer program provides a successful method of teaching eccentric viewing technique.

Key words: *Computer-based rehabilitation, eccentric viewing training, central field loss.*

INTRODUCTION

Pathology of the macular region is associated with a reduction in visual acuity which can cause significant functional handicap, loss of independence and emotional distress. These outcomes are of particular significance to the elderly, an age group which has a high incidence of macular disease.[1,2] The impact of vision loss associated with macular degeneration can be ameliorated with appropriate rehabilitation programs.[3,4] The clinical experience of one of the researchers (KF) indicates that effective rehabilitation programs must comply with certain criteria:

- The materials used to train refixation should consist of high resolution characters against a background which provides optimum contrast.
- A range of resource materials should be available; conventionally such resources have consisted of print or pictures of varying size which are presented to the client.
- The materials must be readily accessible and be able to be manipulated in terms of character size and format.

Storage of such resources makes demands on space and reproduction of resources to provide a variety of sizes often results in a compromise of contrast and legibility.

The personal microcomputer has many applications including data storage and manipulation.

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Computers have been used within the vision related health sciences in various applications such as assessment tools[5,6] and as a medium for presenting therapy.[7,8]

Computers have also been widely used as a means of providing information to visually-impaired people. The computer has certain features which correspond to the requirements of assessment and rehabilitation of visually-impaired persons. These include the ability to store large amounts of data in a format which is easy to access; the ability to manipulate the data on display and to interchange data efficiently; and the availability of high resolution monitors that permit display of data in a clear high contrast manner. The personal computer should provide an effective medium for the presentation of visual rehabilitation programs. Based on the need to find a more efficient means of providing eccentric viewing training and the recognised features of the personal computer, a software package has been developed to assist persons with macular degeneration to enhance their visual function by the accurate relocation of fixation.

MATERIALS AND METHODS

The computer used for development and initial trials was an IBM compatible PC 386 with super VGA monitor. The trial program was developed based on the information gained from visually-impaired clients in relation to the legibility of letters and graphics and from clinical orthoptists in relation to the development of the user interface. Design features such as ease of movement from one screen of information back to the previous screen or forward to the next screen, ease of transfer from a module back to the main menu, footnote displays of prompts and availability of a help function were addressed in the development of the program.

Preliminary clinical trials were carried out in the Visual Rehabilitation Clinic, La Trobe University, with four legally blind clients who had central field loss due to age-related macular degeneration or Leber's optic atrophy. Each training session was typically 45 minutes duration. The training sessions were conducted by one of the researchers (KF), who is the co-ordinator of the Visual Rehabilitation Clinic. Permission from clients for their participation in the trials was sought by informed consent following discussion of the computer package.

The computer program consists of four modules:

- A module to establish the eccentric viewing point at an appropriate degree of eccentricity.
- An exercise module to reinforce the chosen eccentric viewing position.
- A module to encourage application of eccentric viewing technique to visual tasks other than reading. This module also provides exercises for clients unable to appreciate print material.
- A module of advanced reading material to extend reading skills for appropriate persons.

Selection of a module is facilitated by the use of a menu. Following module selection a sub-menu enables definition of the parameters of exercise materials specific to the requirements of each client.

Module 1

The sub-menu allows the clinician to choose print size, viewing angle and eccentric position in relation to the fovea. Once these parameters are set, an image of a letter, number or simple shape as determined by the clinician appears on the screen. The position of the image is in the appropriate area of field to ensure stimulation of the most efficient eccentric viewing position. This position will have been previously identified by the clinician in pre-training assessment. A second image provides a reference point to assist the client to appreciate the size of the refixation movement required to attain the eccentric viewing position.

Module 2

The sub-menu allows the clinician to define various parameters, including degree of eccentricity in relation to the primary position; viewing angle; number of letters in a word stimulus; and print size. Words of chosen size and length appear on the screen at the designated position to stimulate the most viable section of retina.

Module 3

This module consists of 6/60 and 6/36 equivalent size shapes which can be generated at various locations on the screen. Using the sub-menu the clinician can choose the size of the shape and whether the shape will be randomly generated by the computer or selected by the clinician. The location of the shape on the screen can be randomly generated or predetermined by the clinician.

Random generation and location of shapes encourages the client to search the field of view to find an object and then to use the eccentric viewing technique to identify that object. The facility for the clinician to determine the shape and its location on the screen provides exercises to increase awareness of a particular area of the visual field or to become familiar with specific shapes.

The shapes that form the library for this module may also be used for persons unable to cope with print material. The variety of shape size and contrast ensures some images that all clients can see in order to develop their confidence. The library of shapes also includes some images that are more difficult to recognise or have more detailed components. This inclusion was intended to provide a means of challenging the skill levels of more advanced clients.

Module 4

This module consists of narrative material, graded in length and varying in content. The sub-menu provides a choice of: print size (N18 or N14); the number of lines to be displayed (1 through to 12 lines); narrative length and narrative content. These choices allow narrative of appropriate length and content to be chosen for each client. The ability to display a given number of lines of text assists clients who experience the "crowding" effect in the initial stages of text reading when using eccentric viewing technique.

RESULTS AND DISCUSSION

Results of the initial evaluation trials with clients of the Visual Rehabilitation Clinic, La Trobe University are summarised in Table 1 and described below. The trials were carried out over a period of 12 months.

TABLE 1
Summary of results from clinical trials

Client	Pre VA	Post VA	No. Sessions
KM	N36	N14	24
LW	N48	N14 words N18 text	6
MW	N48	N24	9
RB	N36	N14	17

Client KM

Diagnosis: Age-related macular degeneration with a near visual acuity of N36 assessed with a Curpax near acuity test without optical aids.

Eccentric viewing training had commenced with the conventional print exercises using an eccentric viewing angle of 4° in dextro-elevation. Progress was slower than anticipated and KM was losing confidence. When the computer-based training program became available, it was decided that this method would be of benefit to this client. The computer-based training method commenced with the graphic image exercises from module 3 as a strategy to restore client confidence. The 6/60 and 6/36 equivalent shapes were easily recognised by KM. Achieving this recognition gave the client confidence in her ability to use residual sight and reinforced the fact that some sight remained. As she became more confident, module 2 was commenced using three letter words of N24 size. The client was able to read these words although she confused letters that were similar in shape. The strategy was varied to commence each session with graphic images from module 3 to establish confidence and was followed by words from module 2. At each successive session, words of increasing length were presented. At the completion of 24 sessions, KM is working from module 4 and reading N14 size text. Long words and those composed of similar shaped letters still provide a challenge, but she is reading confidently and with a reasonable level of fluency.

Client LW

Diagnosis: Age-related macular degeneration with a near visual acuity of N48 assessed by the

Curpax near visual acuity test without optical aids.

Training commenced with the computer package using an eccentric viewing angle of 3° in elevation. Training commenced with the graphic images of module 3 as the client lacked confidence in the accuracy of her residual vision. The graphic images were readily identified and the client progressed to the more visually demanding exercises of module 2. At the completion of six sessions, LW was reading N14 words and N18 text. This rate of improvement in six sessions is unusual using conventional training techniques based on the extensive clinical experience of one of the researchers (KF). At this stage, the client chose to discontinue training because of transport problems. Near visual acuity could probably have been improved with further training. However, a significant reduction in print size was achieved in a small number of sessions.

Client MW

Diagnosis: Age-related macular degeneration with near visual acuity of less than N48 assessed on the Curpax near visual acuity test without optical aids.

Training commenced using the computer package and an eccentric viewing angle of 2° in depression. This client is not interested in reading but is very keen to use her sight for craft related activities. Initially only the graphic image exercises from module 3 were used until the shapes were confidently and accurately identified. As MW gained confidence with module 3, she requested to try the word exercises from module 2. At the time of writing she had completed nine training sessions and was reading N24 size words accurately and fluently.

Client RB

Diagnosis: Leber's optic atrophy with a near visual acuity of N36 assessed on the Curpax near acuity test without magnifying aids.

Previously, RB had spent many sessions using conventional training methods. This client has not found it easy to use one eccentric viewing position and has alternated between potential eccentric viewing positions of elevation and depression. His pathology has progressed since training commenced and he loses confidence easily.

When the computer based training became available, and following consultation with client RB, the decision was made to change to this method of training. Computer training commenced with module 1 to establish an eccentric viewing point. The eccentric viewing position was established at 14° in depression. It was noted that RB could identify the shapes but not the letters presented in module 1. Based on this information, module 3 was used in preference to module 2 as a means of reinforcing the chosen eccentric viewing point. Initially RB could identify the bold shapes at 6/60 and after two further sessions, he was able to identify 6/60 and 6/36 shapes with ease. Once RB had gained confidence through success with this module, he was able to return to module 2 and read N24 words. At the completion of the training program over a period of 17 sessions, RB was working from module 4 and reading N14 print size. He no longer alternated between elevation and depression, but settled on one eccentric viewing area and realised he was able to reduce the angle he had originally used to 5° depression. RB progressed well having gained confidence from his success with module 3.

SUMMARY

Client responses during the trials to date suggest that the computer program is viable as a method of training eccentric viewing. The preliminary results suggest that computer-based eccentric viewing training can provide an alternative to conventional training methods by achieving performance at a level and rate that, for the clients tested, exceeds one that would have been predicted by the clinician involved in the testing. The access to exercises which are not print dependent has been an apparent advantage to some clients and has facilitated progress which was previously not being achieved through use of conventional training.

An important component of further trials is to assess whether these preliminary results can be sustained in a different context, in which the

clinicians involved in the testing have not been involved in the development of the program. Because of the small sample size of the client base in the preliminary testing, further evaluation trials must be undertaken to investigate the versatility of the program with a wider range and greater number of clients in a variety of settings. An important component of these further trials will be obtaining feedback from both clients and clinicians with a view to modifying, as appropriate, the user interface.

ACKNOWLEDGEMENT

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THE EFFECTS OF AEROBIC EXERCISE ON INTRAOCULAR PRESSURE

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Abstract

Intraocular pressure (IOP) measurements were taken on nine subjects at rest and at two standardised workload levels after aerobic exercise was performed on a bicycle ergometer. The mean resting IOP was 15.22 mmHg and after four minutes of cycling at 55% of the subjects maximal aerobic capacity (VO2 max), the mean IOP dropped to 12.25 mmHg. A further four minutes of cycling at 75% of VO2 max showed a further drop to a mean IOP of 10.30 mmHg. The results of this study confirm the observation of other studies, that moderate aerobic exercise significantly lowers IOP. The implications of aerobic exercise reducing IOP in glaucoma patients is discussed.

Key words: *Non-contact tonometry, submaximal workload, glaucoma, workload intensity.*

INTRODUCTION

Reduction in intraocular pressure (IOP) related to physical exertion has been well documented.[1-6,8] It has been shown that this effect is inversely proportional to the workload intensity.[1,6] Krejci[8] reported on 17 subjects pedalling at four progressive workloads (25%, 50%, 75%, 100% of maximal aerobic exercise capacity). IOP measurements were taken when a standard heart rate level was reached for each subject. The IOP was shown to decrease from 16.6 mmHg to 12.1 mmHg in the right eye and from 17.3 mmHg to 13.3 mmHg in the left eye. Other studies have found that IOP can change with body position,[9] varies diurnally[10] and that the numerical value can be altered by the tonometry technique itself.[11]

Shapiro and Shoenfeld[7] stated that many earlier studies disregarded the influence of body position, diurnal variation, and tonometry techniques on IOP values. They concluded that when these factors are taken into consideration, and only the exercise workload level is considered, there is only a relatively small and insignificant decrease in IOP secondary to aerobic exercise.

The purpose of this study is to examine the effect of aerobic exercise on intraocular pressure using an experimental design that standardises the variables that influence IOP and also the intensity of physical exercise.

This paper was based on a research project completed as part of an undergraduate degree by Pierre Elmurr in the School of Orthoptics, Faculty of Health Sciences, University of Sydney in 1992.

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METHOD

Five male and four female sedentary students aged between 20-31 years (mean age 22.4) from Sydney University were voluntarily recruited to the study. After consenting to the experiment all subjects completed a health status questionnaire (parQ test). This questionnaire is designed to identify people for whom physical activity is inappropriate. All nine subjects were able to participate in the study and none had been previously involved in a regular exercise programme.

Aerobic exercise was performed on a bicycle ergometer. This was chosen because this form of activity is well accepted as a means of calibrating levels of physical exercise and has the flexibility of examining intraocular pressure during exercise.[7] The first two stages of the experiment were designed to ensure that each subject exercised at a standardised level. Intraocular pressure measurements were taken in the final third stage using a Keelar Pulsair non-contact tonometer.

Stage 1: Determination of relationship between heart rate, volume of oxygen, and power output (watts).

Subjects were required to perform an incremented exercise test on a ergoline cycle ergometer (model 800s) for 12 minutes. Four power outputs were used increasing by 25 watts every three minutes after commencing at 50 watts. The subjects were required to cycle at 60 RPM at all times. The heart rate was measured at rest, and every three minutes in conjunction with the increasing power output. Electrocardiograph (ECG) and respiratory gas exchange were monitored throughout testing. Figure 1 illustrates the exercise physiology set-up. Gas analysis for concentration of carbon dioxide (CO2) and oxygen (O2) in expired gas was measured using amateur gas analysers and were recorded on a online computer system.

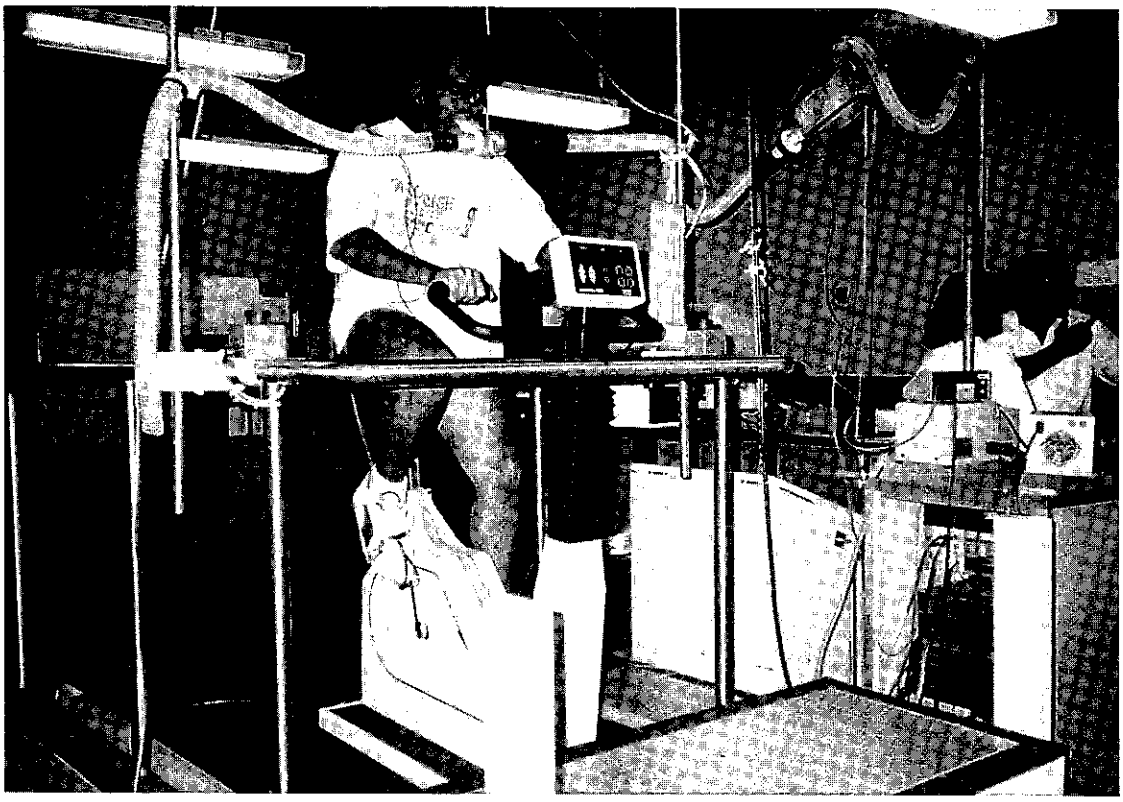

Figure 1. Stage 1 & 2: Exercise physiology set up for determination of maximal oxygen uptake (VO2 max).

Stage 2: Determination of maximal oxygen uptake. (VO2 max)

A two minute rest period was allowed, then another incremented procedure was performed. Subjects cycled with power output increased by 25 watts every minute, after commencing at 50 watts until volitional fatigue occurred. Heart rate was monitored every minute to coincide with incrementation. ECG and respiratory gas exchange were monitored as mentioned above. This procedure determines the VO2 max, which is the maximal aerobic capacity that an individual can sustain under exercise conditions before volitional fatigue. Calculations of VO2 max were performed on an online computer system.

The results from the first two stages were correlated graphically to determine the two submaximal workload intensities to be adopted in stage 3, and were set at 55% and 75% of VO2 max. Figure 2 demonstrates how these levels were determined for one subject. The four VO2 numerical values taken in stage one, every three minutes, were plotted against the equivalent power output (A at 3 min, B at 6 min, C at 9 min and D at 12 min). The VO2 max value determined in stage two was also plotted on the graph. (Figure 2 VO2 max = 26.2) The workload levels to be used in stage three were determined by calculating 55% and 75% of the VO2 max and reading from the graph the equivalent power output. For example in figure 2, 75% of 26.2 is 19.65 which corresponds to a workload level of 100 watts for that subject. This procedure was followed for all nine subjects.

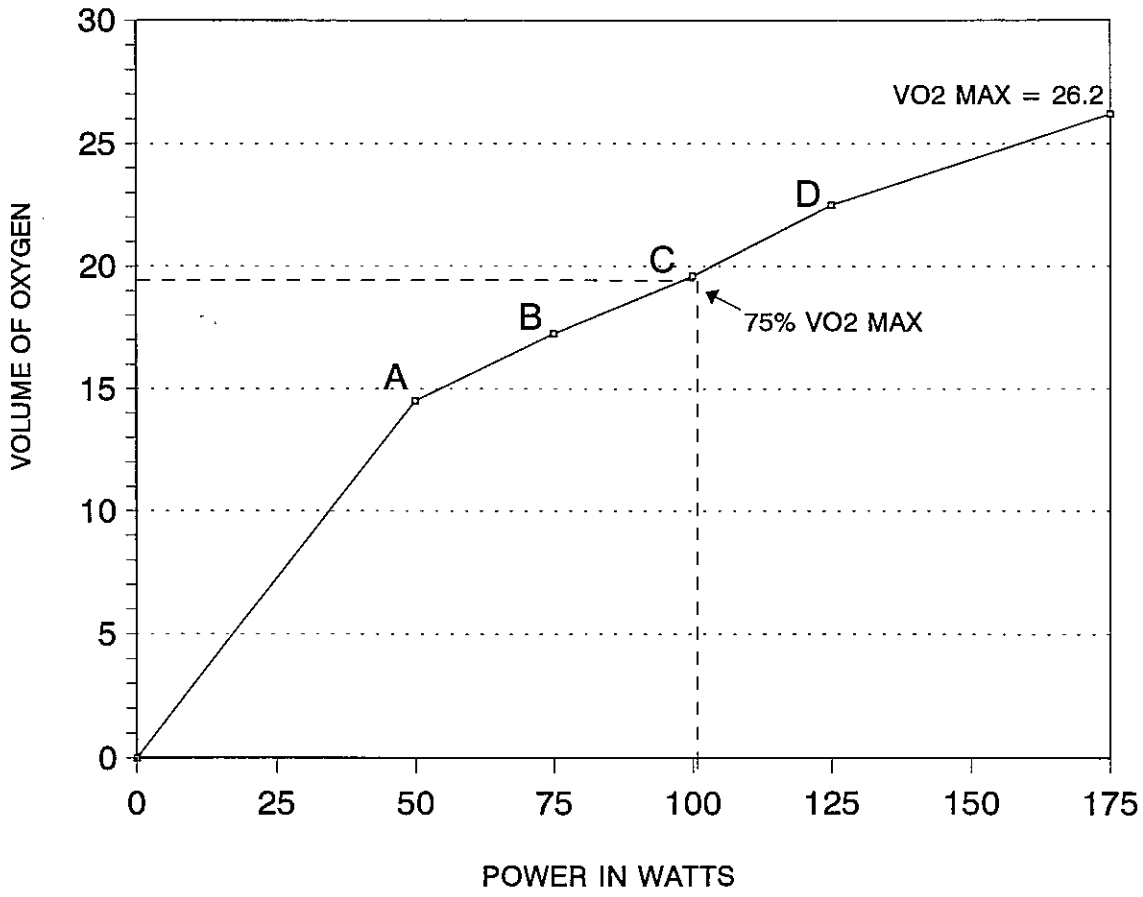

Figure 2: Determination of submaximal workloads.

Stage 3: IOP measurement.

One week later subjects were required to sit on the ergoline bike and IOP measurements were taken at rest. Four minutes of cycling was commenced with a workload equivalent to 55% of each subject's VO2 max. Four consecutive IOP measurements were taken at the end of the 4th minute. A rest period was allowed until heart rate came within 15 bpm of resting heart rate then another four minutes of cycling was commenced with a workload set at 75% of each subject's VO2 max. IOP measurements were again taken at the end of the 4th minute. Figure 3 demonstrates how the IOP measurements were taken.

The above procedure took 15 minutes to complete and all subjects were tested at the same time of day. Heart rate was monitored every minute, blood pressure before and after each four minute interval, and ECG was monitored throughout the procedure.

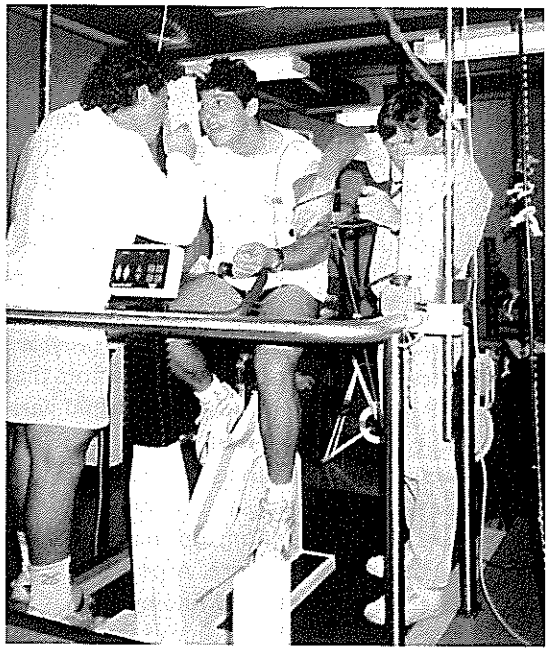

Figure 3: Measurement of IOP during stage 3.

RESULTS

The mean heart rate and blood pressure levels obtained in stage three indicated that an appropriate level of aerobic exercise was achieved for the testing procedure.

When IOP was compared between eyes of the same subject no statistical difference was found. This enabled the measurement of IOP for right and left eyes to be combined for each subject in the statistical analysis. A two paired T test was used and alpha was set at the conventional level of .05.

Mean resting IOP was 15.22 mmHg. At the end of the first workload level (55% of VO2 max) the mean IOP had dropped to 12.25 mmHg. At the end of the second workload level (75% of VO2 max) the mean IOP had dropped to 10.30 mmHg. Table 1 tabulates these results. The decrease in IOP from rest to the first workload level: 55% VO2 max ($t = 6.49$), from rest to the 2nd workload level: 75% VO2 max ($t = 10.61$), and from the first to the second workload level ($t = 7.91$) were all found to be statistically significant ($p < .0001$ level).

DISCUSSION

It is well known that a change in body position can cause a change in IOP.[9] All measurements in this study were taken in the sitting position whilst the exercise was performed so that the effect of body position on IOP was controlled.

Over the course of the day IOP varies an average of 3 mmHg to 6 mmHg in normal individuals.[13,14] These diurnal changes can occur over as short a period as 20 minutes.[15] All measurements were taken at the same time of day for all subjects and the testing procedure took no longer than 15 minutes to complete. Taking four consecutive measurements of each eye has been shown to produce accurate and precise results.[16] Non-contact tonometry was used in preference to contact tonometry as the latter tends to distort

TABLE 1
Mean IOP measurement at rest, 55% and 75% of VO2 max

Exercise Intensity	Mean IOP (mmHg)	Standard Deviation	Difference in IOP (mmHg)
Rest	15.22	2.34	2.97
55% VO2 max	12.25	2.13	
Rest	15.22	2.34	4.91
75% VO2 max	10.30	1.94	
55% VO2 max	12.25	2.13	1.94
75% VO2 max	10.30	1.94	

results because the instrument itself alters the steady state condition of the eye during repeated IOP measurements.[17,18]

Reduction of IOP following aerobic exercise noted in this study confirms previous findings.[1-6,8] A quantitative comparison between the results of this study with previous work is difficult because of the differences in methods used. The form of aerobic activity, workload intensities used, and type of tonometry measurement vary in each study. Additionally some previous research designs have not controlled factors that affect IOP levels, such as posture, diurnal variation and tonometry technique.

It is interesting to note that Shapiro et al.[7] who controlled similar variables to this study, did not find a significant relationship between IOP reduction and aerobic exercise. A possible explanation for these contradictory findings is that the intensity levels set for the aerobic exercise workload were too low to produce a significant drop in IOP. This highlights the apparently important relationship between IOP and aerobic exercise intensity.

Passo, Goldberg, Elliot, and Buskirk[19] investigated the implication of a reduction in IOP with exercise. Nine sedentary subjects suspected of having glaucoma were observed before and after three months of aerobic exercise training. The mean IOP had decreased by 4.6 mmHg at the end of the conditioning period. With cessation of exercise IOP returned to elevated preconditioned levels by three weeks. They concluded that regular aerobic exercise is associated with a reduction in elevated IOP and may represent an effective non-pharmacologic intervention for patients suspected of having glaucoma. It is important to recognise that recent physical exertion may mask increased IOP in patients presenting for routine examination. They suggest that an exercise history be taken to determine IOP dynamics more accurately. Changes in exercise status should also be monitored in routine glaucoma care, just as changes in pharmacologic therapy are made.

The physiological mechanisms responsible for the reduction of IOP are not clear. It has been suggested[2-4,6-8] that IOP decreases when blood pH and venous pressure decrease and CO2, lactate and osmolarity increase. Aerobic training has been shown to reduce catecholamine levels, especially norepinephrine concentrations.[20] Regular exercise also enhances the parasympathetic-sympathetic input ratio at rest, increasing relative acetylcholine-norepinephrine release.[20-22] These autonomic changes that accompany regular exercise may favour reduced IOP. These two mechanisms appear to be related to improved facility of outflow through the anterior chamber within the eye.

CONCLUSION

It appears that some physiological mechanism is responsible for IOP reduction immediately following aerobic exercise. Further study is required to understand the mechanism of action of this acute change and the role of aerobic exercise as a non-pharmacological alternative therapy to lowering IOP in glaucoma patients.

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CONTRAST SENSITIVITY AND VISUAL ACUITY AFTER EXCIMER LASER TREATMENT FOR MYOPIA — PRELIMINARY FINDINGS ON 15 PATIENTS

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Abstract

This paper examines the preliminary effects of excimer laser treatment on visual function in 15 patients who had myopia ranging from −1.75 to −6.00 dioptres.
The use of the excimer laser to correct refractive error is discussed. Visual function was assessed using a Snellen's acuity test as well as the Vector Vision CSV 1000 contrast sensitivity test. All tests were performed without and then with additional glare using the Mentor Brightness Acuity Tester (BAT). Visual function was assessed prior to excimer treatment then post treatment at three months and the patients will be followed up again at six and 12 months. The visual function at three months post excimer is discussed in this paper.
Key words: *Excimer laser, photorefractive keratectomy (PTK), myopia, contrast sensitivity, Vector Vision CSV 1000, visual acuity.*

INTRODUCTION

In 1983 Trokel reported on the use of the Excimer laser for corneal surgery.[1] He showed that this ultraviolet laser allowed the precise removal of corneal tissue through an unusual laser/tissue interaction. The procedure, involving the use of the excimer laser to alter the refractive error of the eye is now known as photorefractive keratectomy[2] (PRK) as light energy (photo) is used to excise (ectomy) a portion of the cornea (kerato). (The therapeutic treatment of corneal opacities using the excimer laser is known as phototherapeutic keratectomy; PTK).

When performing PRK surgery the excimer laser actually ABLATES (or removes) a very thin layer of the central corneal stroma having the effect of flattening the central cornea thus reducing its refractive power. As a result, parallel rays of light are bent less and are able to come into focus on the fovea of the myopic eye. Initially, post excimer, the patient is made hypermetropic but this settles over time, resulting in a refractive error that is closer to (or at) emmetropia. As the excimer removes such a small depth of corneal tissue the structural integrity of the eye remains intact.

The name excimer is derived from the first two and last syllables of the term excited dimer.[3] An excited dimer is two atoms of inert gases, in this case argon and fluoride, which form a temporary and unstable molecule when forced together

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under extremely high pressure and high voltage. The unstable molecule (or the dimer) decays very readily giving off an emission of an individual photon of 193 nm far UV light. The particular properties of this wavelength of laser light make it suitable to use to decompose the cornea.

The emitted photons (or pulses) are of extremely short wavelength light thus they contain large amounts of energy. The photons are directed onto the cornea by a series of lenses and mirrors housed inside the laser.

Prior to commencement the epithelial layer of the cornea is marked then scraped away thus the photons ablate firstly the exposed Bowman's layer then corneal stroma. When a photon from the excimer laser hits the surface of the cornea the following occurs;

- the photon is absorbed by the corneal tissue it hits
- the energy from the photon (6.4 electron volts) exceeds the binding voltage of the corneal tissue carbon-carbon bonds thus the carbon molecule bonds breakdown removing the corneal tissue[4,5]
- the breaking of the molecular bonds occurs so quickly, (a few picoseconds; ie 1×10^{-12}), and

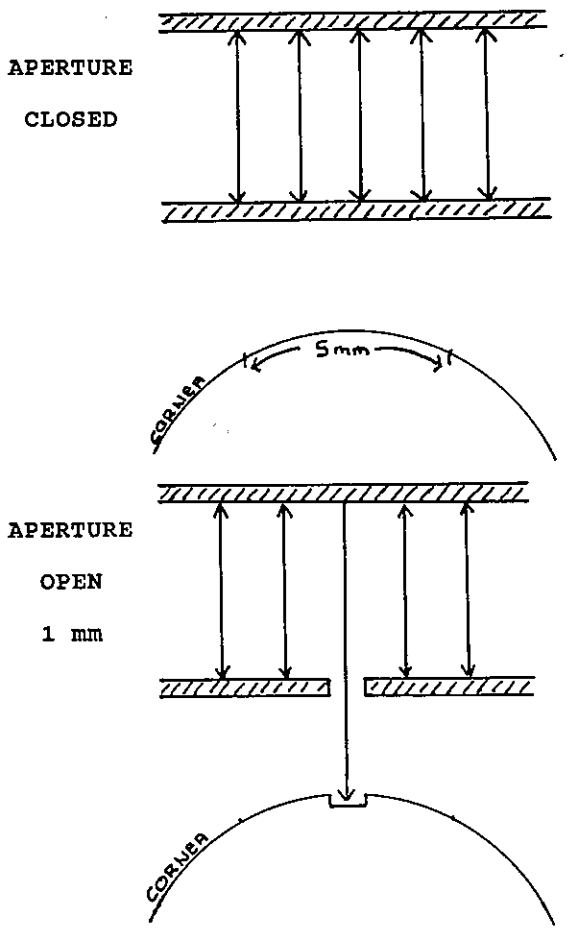

Figure 1: Excimer laser; aperture closed (top). Aperture open 1 mm allowing photons of 193 nm light to pass through onto the cornea ablating corneal stroma (bottom).

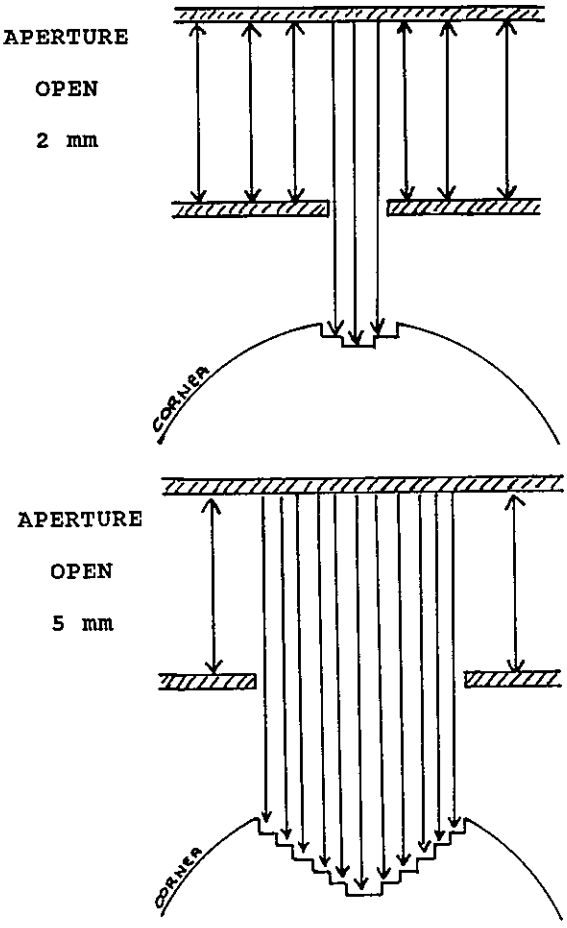

Figure 2: Excimer laser; aperture open 2 mm allowing more photons to pass onto the cornea creating a series of steps into the cornea (top). Aperture open 5 mm (maximum opening) allowing more photons to pass onto the cornea creating a deeper stepped ablation into the cornea (bottom).

the photon is so powerful that the corneal molecular fragments are ejected from the surface of the cornea at supersonic speed of 1000 to 3000 meters per second, carrying excess energy with them.[6]

- Each photon from the excimer removes between 0.1 and 0.5 micrometers or microns of tissue (1 micrometer = 1×10^{-6} meters) with no burning or cutting thus there is no adjacent tissue damage.[6] The tissue removal is precise to within 0.25 microns per pulse.

By the end of the procedure a 5 mm diameter of corneal tissue has been precisely removed by a series of photons passing through a gradually increasing aperture opening. The photons leave a series of steps in the cornea thus the section that is removed is deeper in the centre (see Figures 1 and 2). The steps are smoothed out by epithelial regrowth. (When the excimer laser is used for therapeutic purposes; PTK, the aperture remains fully open so that the tissue is removed evenly within the ablation zone). The excimer causes very little damage to the surronding tissue.

The ablation procedure is accompanied by a strong burning smell, similar to an intensive smell of burning hair. This is thought to be because the airborne particles ejected from the corneal surface are similar types of particles to those given off by thermally damaged biological tissue in the form of smoke but the excimer does NOT burn.[5]

The excimer laser thus differs from the other lasers commonly used in ophthalmology because its beams are absorbed by the cornea causing decomposition of a predetermined depth of corneal molecules without burning.

The aim of the current study was to examine the effect of excimer laser treatment for myopia on different aspects of visual function pre and post excimer treatment. This paper reports on the visual function of 15 patients three months post excimer. Visual function six and 12 months post excimer will be the subject of a later publication.

METHODS

A group of 15 patients ranging in age from 21 years to 57 years who had been followed up for three months post excimer treatment have been included in these preliminary results. All patients had myopia ranging from −1.75 to −6.00 dioptres (spherical equivalent) prior to being treated with the excimer (see Table 1).

Prior to treatment all patients had visual acuity (VA) assessed monocularly with a Snellen's chart at six meters, a logMAR chart at three meters and one third of a meter and an OPSM near vision chart. The distance vision tests were performed with and without optimal correction and with and without glare.

Contrast sensitivity function was assessed using the Vector Vision CSV 1000 at eight feet. The CSV 1000 chart is back lit for constant illumination. The illumination varied with ambient room light. This test was performed with and without optimal correction and with and without glare.

The CSV 1000 test consists of four different rows; each containing eight pairs of targets (numbered one to eight across the row). The targets decrease in contrast across the row. The spatial frequency of the pairs of targets in each row increases down the chart (that is, the stripes in each pair of targets become narrower). The spatial frequencies in each row are, row A = 3; row B = 6; row C = 12 and row D = 18 cpd.

Patients were instructed to tell the examiner whether the stripes appeared in the top target or the bottom target in each pair or if there were no stripes. For each eye the number of the target with the minimum contrast at which stripes were seen

TABLE 1
Refractive error; pre excimer and post excimer laser treatment

Case No	Age	Sex	Refractive error	
			Pre	Post
1	23	M	−2.50	+0.25
2	27	F	−3.25	+0.25
3	41	F	−3.75	+2.25
4	32	F	−6.00	+0.75
5	32	M	−3.50	+0.75
6	27	M	−1.75	+0.25
7	29	F	−2.75	+0.50
8	45	F	−4.75	+0.25
9	21	F	−4.75	−1.00
10	32	F	−5.25	−0.75
11	33	M	−4.25	+0.25
12	24	M	−3.00	+0.50
13	57	M	−3.75	+0.50
14	34	F	−6.00	+1.00
15	39	F	−1.75	−0.25

in each row was recorded. Thus contrast sensitivity was recorded as a number between one (maximum contrast; the worst score) and eight (minimum contrast; the best possible score) or zero was recorded if no stripes were seen on a given row.

The effect of glare on the vision tests and the contrast sensitivity test was assessed using the Mentor Brightness Acuity Tester (BAT) glare test at maximum power (400 foot lamberts).

The mean scores for VA and contrast sensitivity were calculated for all patients. Pre excimer VA and contrast sensitivity mean scores were then compared to post excimer scores using a t test to see it there was any significant difference in scores. When the p value was less than 0.01 the difference in scores was considered to be significant.

Immediately following the visual assessment the patients had excimer laser treatment (Summit Laser) to correct their myopia. Each patient was checked by their ophthalmologist two days after excimer treatment to ensure that the cornea had reepithelized. Patients were then seen weekly extending to monthly by their ophthalmologist. Subjective retinoscopies were conducted, the haze was graded and the IOP was measured.

All of the above mentioned visual function tests were repeated on all the patients after the excimer at six weeks, three and six months. They will also be tested at 12 and 24 months.

RESULTS

(i) REFRACTIVE ERROR: — Three months post excimer the refractive errors ranged from +2.25 to −1.00. Ten of the 15 patients were between +0.75 and +0.25. Twelve of the 15 were hypermetropic (see Table 1).

(ii) HAZE: — Post excimer all patients had corneal haze ranging from extremely mild to moderate. This suggested that all the corneas were still recovering.

All visual function results are stated as being either BEST CORRECTED (that is the scores with the full refractive correction) or UNCORRECTED (that is the scores without any correction). For statistical analysis Snellen's VA was divided into categories as follows; 6/5⁺, 6/5, 6/6⁺, 6/6, 6/9⁺, 6/9 and so on up to 1/60.

TABLE 2
Snellen's visual acuity; uncorrected pre excimer compared to post excimer and best corrected pre excimer compared to post excimer

Case No	Uncorrected VA		Best Corrected VA	
	Pre	Post	Pre	Post
1	3/60⁺	6/9⁺	6/6⁺	6/6
2	6/24	6/9	6/6	6/6
3	3/60	6/24⁺	6/9	6/9
4	2/60	6/12⁺	6/6⁺	6/5
5	3/60⁺	6/18	6/5	6/5
6	3/36	6/6⁺	6/5	6/5⁺
7	1/60	6/9	6/6⁺	6/6⁺
8	3/60⁺	6/9⁺	6/9⁺	6/9⁺
9	2/60	6/9	6/6	6/6⁺
10	6/60	6/9	6/6⁺	6/6
11	3/60	6/6⁺	6/5	6/6⁺
12	5/60	6/9⁺	6/5⁺	6/5
13	6/60	6/6	6/9	6/6
14	3/60⁺	6/9	6/6⁺	6/6
15	6/60	6/6⁺	6/9⁺	6/6
Mean	4/60	6/9⁺	6/6⁺	6/6⁺

(iii) VISUAL FUNCTION WITHOUT GLARE (pre excimer compared to post excimer).

(a) BEST CORRECTED: — The best corrected Snellen's VA pre excimer was 6/6 or better in all but four patients (who were all 6/9⁺ or 6/9; see Table 2. There was no significant difference in pre excimer compared to post excimer best corrected VA ($t = -1.11$, $p = 0.298$). All but two (Cases 3 and 8) were 6/6 or better post excimer. Both these cases had pre excimer VA of less than 6/6.

When the pre excimer best corrected contrast sensitivity scores were compared to post excimer best corrected scores the post excimer scores were all slightly reduced but there was no significant difference in scores (see Table 3).

TABLE 3
Effect of excimer laser on best corrected contrast sensitivity scores. Pre excimer compared to post excimer; (mean scores, standard deviations, t and p values)

Row	Contrast sensitivity score				t value	p value
	Pre excimer		Post excimer			
	Mean	SD	Mean	SD		
A	4.7	(1.418)	4.5	(1.269)	0.43	0.678
B	5.3	(0.949)	4.7	(1.494)	1.41	0.193
C	5.1	(0.876)	4.7	(1.494)	0.60	0.565
D	5.7	(1.337)	4.4	(2.171)	1.49	0.169

(b) UNCORRECTED: — The pre excimer uncorrected VA ranged from 1/60 to 6/24 with only four patients having 6/60 or better. (The mean uncorrected VA was 4/60; see Table 1).

The uncorrected VA at the three months post excimer visit was significantly improved in all cases (mean VA 6/9⁺; $t=16.51$, $p=0.0001$). Scores were as follows; seven of the 15 patients had 6/9⁺ or better, six of the 15 patients had 6/12⁺ or 6/9, (Cases 2, 4, 7, 9, 10, 14) and two of the 15 patients had 6/18 or less, (Cases 3 and 5; see Table 2).

At the time of writing this paper the six month data was available on four of the six patients with 6/12⁺ or 6/9, (Cases 2, 4, 7, 9). All four had improved to 6/6 or better by six months. Six month data was also available on both of the cases who had VA of 6/18 or less at three months. One had improved to 6/5 (no refractive error; Case 5). The other patient (Case 3; who had a best corrected VA of 6/9 pre excimer) still had 6/18 vision.

Pre excimer contrast sensitivity was not assessed without correction as most patients could not see the contrast sensitivity chart without their correction.

(c) BEST CORRECTED PRE EXCIMER COMPARED TO UNCORRECTED POST EXCIMER VISUAL FUNCTION: — As the aim of excimer treatment is to enable the patient to see and function normally without the aid of glasses or contact lenses the results of the pre excimer visual function with best correction were compared to the post excimer uncorrected visual function.

There was a minimal decrease in scores from pre excimer best corrected VA (mean 6/6⁺, SD 6/9⁺ to 6/5) to post excimer uncorrected VA (mean 6/9⁺, SD 6/6⁺ to 6/12) at three months (see Table 2). This decrease was NOT significant ($t=-3.12$, $p=0.008$).

Statistical analysis of pre excimer best corrected contrast sensitivity compared with post excimer uncorrected contrast sensitivity revealed that contrast sensitivity was significantly worse post excimer in rows C and D (high spatial frequencies) at three months post excimer (see Table 4). As this finding must be reflecting the residual refractive error present in all patients three months post excimer no meaningful conclusions can be drawn at this stage.

TABLE 4
Effect of excimer laser on contrast sensitivity scores. Pre excimer best corrected scores compared to post excimer uncorrected scores; (mean scores, standard deviations t and p values)

Row	Contrast sensitivity score (Best corrected) (Pre excimer		(Uncorrected) Post excimer		t value	p value
	Mean	SD	Mean	SD		
A	4.6	(1.183)	4.1	(1.710)	1.17	0.262
B	5.1	(0.915)	3.9	(1.981)	2.50	0.025
C	5.0	(1.069)	3.5	(1.885)	3.15	0.007
D	5.4	(1.454)	3.6	(2.230)	3.11	0.008

(iv) VISUAL FUNCTION WITH GLARE

(a) GLARE EFFECT ON BEST CORRECTED SCORES: — When comparing results pre excimer without glare to pre excimer with glare VA scores remained at a mean of 6/6⁺ with and without glare ($t=-0.72$, $p=0.486$) thus glare had no effect on VA. Contrast sensitivity showed that scores without glare were significantly reduced in all rows; (see Table 5).

Post excimer 10 cases were tested with best correction and glare (Cases 2, 7-15). When comparing the results post excimer without glare to post excimer with glare VA, scores were

TABLE 5
Effect of glare on contrast sensitivity scores. Best corrected pre excimer and post excimer scores; (mean scores, standard deviations t and p values)

Row	Contrast sensitivity score Without glare		With glare		t value	p value
	Mean	SD	Mean	SD		
Pre excimer						
A	4.6	(1.183)	2.8	(1.781)	3.02	0.009
B	5.1	(0.915)	2.9	(1.685)	4.08	0.001
C	5.0	(1.069)	2.7	(1.944)	4.28	0.001
D	5.4	(1.454)	2.5	(2.134)	4.56	0.001
Post excimer						
A	4.5	(1.269)	3.4	(0.843)	3.16	0.012
B	4.7	(1.494)	3.9	(1.524)	2.45	0.037
C	4.7	(1.494)	3.0	(1.700)	5.67	0.001
D	4.4	(2.171)	2.7	(1.418)	2.68	0.025

marginally improved with glare but this improvement was NOT significant (mean with 6/6⁺ and without glare 6/6; $t = -1.35$, $p = 0.209$) for these 10 cases. Contrast sensitivity scores were significantly reduced in rows A and C. In rows B and D the scores were almost significant. As there were only 10 cases it is possible that, with more cases, the reductions will become significant (see Table 5).

(b) GLARE EFFECT ON BEST CORRECTED PRE EXCIMER COMPARED TO POST EXCIMER SCORES: — VA was not significantly altered by glare when pre excimer VA with glare (mean 6/6, SD 6/5 to 6/9⁺) was compared to post excimer VA with glare (mean 6/6, SD 6/6⁺ to 6/9; $t = -1.11$, $p = 0.298$).

Contrast sensitivity scores pre excimer with best correction and glare were slightly lower than the post excimer scores with best correction and glare. This was not significant in any rows, however, in rows A and B it was almost significant. Once again the small population may be affecting the figures (see Table 6).

(c) GLARE EFFECT ON UNCORRECTED PRE EXCIMER COMPARED TO POST EXCIMER SCORES: — Uncorrected VA and contrast sensitivity scores using the glare test were not statistically analysed as the addition of glare created a pinhole effect overcoming the refractive error.

(iv) LogMAR RESULTS: — LogMAR results have not been analysed for this study.

(v) NEAR VISUAL ACUITY: — The results of near VA were unaffected by excimer laser treatment with all the patients achieving exactly the same near VA post excimer as pre excimer. However, patients frequently took a long time to focus on the N5 chart.

TABLE 6
Effect of glare on contrast sensitivity. Best corrected contrast sensitivity; (mean scores, standard deviations t and p values). Pre excimer compared to post excimer

Row	Contrast sensitivity score				t value	p value
	Pre excimer		Post excimer			
	Mean	SD	Mean	SD		
A	4.7	(1.418)	4.5	(1.269)	0.43	0.678
B	5.3	(0.949)	4.7	(1.494)	1.41	0.193
C	5.1	(0.876)	4.7	(1.494)	0.60	0.565
D	5.7	(1.337)	4.4	(2.171)	1.49	0.169

DISCUSSION

During the first three months post excimer laser a corneal haze was apparent in all cases. This was thought to be reflecting corneal stroma recovery. Currently the corneal haze is being monitored to ascertain whether or not the amount of haze will prove to be a useful indicator of the rate of regression towards emmetropia; the greater the haze the more the regression.

It is usual for patients to have some degree of hypermetropia in the first few weeks post excimer which regresses with time. The majority of the patients in this study were slightly hypermetropic in the first few weeks. The regression process naturally steepened the patient's cornea taking the eyes back towards emmetropia or even myopia in some cases after three months. No patients in this study were emmetropic at three months post excimer.

With the use of topical corticosteroid eye drops the different rate of regression that occurred from patient to patient was compensated for. Thus if a patient was regressing too quickly (i.e. was rapidly approaching emmetropia or had returned to myopia) steroid drops were used to slow the regression rate. Alternately if a patient was remaining at a constant level of hypermetropia which was too high they were taken off steroids to allow for more regression.

In all cases three months after treatment the patient's myopia had been reduced to a significant degree. After six months most were progressing towards emmetropia (these findings will be the subject of a later publication).

A recent Multicentre study[7] reported that, in all 31 patients, best corrected VA scores were within one line of their pre excimer best corrected VA scores three months post excimer treatment. However no statistical analysis was given. The findings from the present study demonstrated statistically that there was no difference in the best corrected VA scores pre excimer compared to post excimer. Even the slight decrease in the mean VA

when post excimer uncorrected VA scores were compared to pre excimer best corrected scores was not significant.

The introduction of glare pre excimer and post excimer did not significantly affect VA scores in this study. Even when comparing the pre excimer scores with glare, to the post excimer scores with glare there was no significant difference on VA scores. Thus in this study VA assessment using the Snellen chart, even with additional glare, proved to be too gross an assessment to detect the effects of excimer treatment on visual function.

The Multicentre study[7,8] also reported on contrast sensitivity scores using the Pelli-Robson test and the Vistech MCTS test. (Pelli-Robson results cannot be compared to the results of this current study as the Pelli-Robson test was designed to detect peak contrast while being relatively insensitive to defocus. The MCTS test, like the Vector CSV 1000 tests contrast sensitivity function at high spatial frequencies thus is very sensitive to defocus).[9] The results of the Multicentre study[7] and a subsequent letter to the editor of Archives of Ophthalmology[8] stated that no significant difference was found between contrast sensitivity with the BAT glare test set at maximum illumination three months after excimer treatment. They did not state whether contrast sensitivity was tested with or without correction thus it is not possible to make a comparison between the results of their study and the results of the present study.

In the present study, three months after excimer treatment there was a slight decrease in best corrected contrast sensitivity scores when comparing pre excimer scores to post excimer scores. This finding was not significant.

Contrast sensitivity scores were reduced with the introduction of glare pre excimer (that is, glare reduced the performance on contrast sensitivity BEFORE excimer treatment was carried out) thus contrast sensitivity scores were reduced with glare irrespective of excimer treatment. The same finding occurred post excimer. As a result, contrast sensitivity scores with glare post excimer compared to those without glare were not useful in terms of monitoring recovery of visual function. The analysis of pre excimer compared to post excimer best corrected contrast sensitivity scores was also not conclusive. These findings conflict with those reported by Hogan et al.[10] who demonstrated that 17 patients tested with the Vistech MCTS contrast sensitivity test had decreased scores when tested with glare post excimer laser.

In discussion of the Multicentre study,[7] Adamsons[8] commented that the contrast sensitivity results reported were not detailed enough. She went on to state that " . . . it is imperative that its (*excimer laser's*) effect on visual function be evaluated as rigourously as possible. This evaluation should include not only contrast sensitivity and glare testing, but also subjective evaluation by the patient of his or her visual function using a standardised questionnaire".[8] It is hoped that the present study will fulfil these goals after twelve months.

Subjective comments from patients in the present study revealed they were not at all worried about the post excimer haze or small degrees of residual refractive error. The fact that they could see without glasses was overwhelming in terms of patient satisfaction with the procedure. The only complaint made by some of the patients was discomfort experienced from the glare from oncoming headlights when driving at night. Most felt that this was "a small price to pay" in return for not having to wear glasses. Seiler[11] also reported that many of the 255 patients in his study complained of post operative glare which did not interfere with their activities in the long term.

CONCLUSIONS

As stromal recovery is reported to occur for up to two years it is difficult to draw any meaningful conclusions from the present study at this early stage. By following the patients for 12 months (or 24 months if necessary) it is hoped this study will document any subtle effects on visual function.

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CONTRAST SENSITIVITY IN ADOLESCENTS WITH DIABETES*

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Abstract

Due to the nature of diabetic retinopathy, early detection of visual dysfunction is important in diabetic patients. It has been demonstrated in the literature that contrast sensitivity testing can be used as an early index of change in the retina of diabetic patients. Most studies in this area have been conducted on adults. Forty nine diabetic subjects aged 12 to 18 years with normal visual acuity had contrast sensitivity assessed on the Vistech VCTS 6500. Their results were compared to a group of 202 age matched normal subjects. The results of this study reveal that adolescent diabetics show a decrease in contrast sensitivity function, when results are subject to statistical analysis, however the loss in contrast sensitivity would not necessarily be detected in a clinical situation.

Key words: *Contrast sensitivity, diabetes, adolescents, diabetic retinopathy, Vistech.*

INTRODUCTION

Diabetes Mellitus is a metabolic disease that results in an increase in blood glucose concentration due to a lack of insulin or the body opposing the action of insulin.[1] There are two forms of diabetes; insulin dependant diabetes mellitus (IDDM) and non insulin dependant diabetes mellitus (NIDDM).

Initial symptoms of diabetes include excessive urine production, excessive thirst, weight loss and tiredness. These symptoms can be controlled by altering diet (NIDDM) or by the use of insulin (IDDM). However, changes in the eyes of diabetic patients (diabetic retinopathy) can occur even when the symptoms are controlled.

Early diabetic retinopathy is thought to occur as a result of changes in the small blood vessels of the retina leading to a loss of their structural integrity. With progression of the disease changes to the retinal blood vessels include thickening and weakening of capillary walls. These changes lead to microaneurysms, haemorrhages, oedema, hard exudates and cotton wool spots. This is known as non proliferative diabetic retinopathy. Later the patients may progress to proliferative retinopathy consisting of either maculopathy and/or neovascularisation.[2]

As a result of the above-mentioned changes visual function can be disturbed in diabetic patients, even in the early stages of the non proliferative diabetic retinopathy.[3]

Recent literature has revealed that contrast sensitivity may be affected much earlier than visual acuity in patients with diabetic retinopathy.[4-9]

*This paper was based on an Honours Thesis completed as part of an undergraduate degree by Janine Munns in the School of Orthoptics, Faculty of Health Sciences, University of Sydney in 1992.

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The literature has suggested that there is a correlation between contrast sensitivity loss and the severity of retinopathy.[4-6] Two studies have suggested that the degree of loss visual function, as revealed by contrast sensitivity tests, may be related to the duration of diabetic retinopathy.[4,7] The majority of the studies include adult subjects only whilst others encompass a wide age range.

The present study reports on the contrast sensitivity function of adolescent IDDM patients aged 12 to 18 years. The aim was to ascertain if contrast sensitivity function was abnormal and, if so was the abnormality indicative of early visual dysfunction.

Previous research has shown that contrast sensitivity alters with monocular or binocular testing,[10] age[11-14] and sex.[15] As a result, in the present study non diabetic subjects have been compared to IDDM patients of the same age, monocular and binocular scores have been compared and a comparison of overall scores for each sex has been made.

METHOD

Summary of Groups Studied

Two groups of adolescents aged 12 to 18 years were studied.

Group I consisted of 49 subjects (18 males, 31 females) with IDDM with or without diabetic retinopathy (see Table 1). The mean age of this group was 14.8 years. The average disease duration was 7.8 years. Subjects were collected over six months from the Royal Alexandra Hospital for Children, in Sydney.

Group II consisted of 202 non-diabetic subjects (96 males, 106 females) from a secondary school in the north west of Sydney tested over one week. The mean age of the control group was 14.7 years.

TABLE 1
Number of IDDM subjects with and without retinopathy

Eye	No Retinopathy	Mild → Mod Background Retinopathy	Ungradable
Right	25	21	3
Left	30	17	2

Study Design and Selection of Study Sample

Each subject's visual acuity was assessed monocularly at 6 m and 1/3 m using a Snellen's chart. Cover test and Lang's Stereo Test were also performed as contrast sensitivity may also be affected by amblyopia,[16] refractive error and astigmatism.[17] Criteria for inclusion in the study was an uncorrected monocular visual acuity of 6/6 or better and N5, no strabismus and 550 seconds of arc on the Lang Stereo Test.

If the subject met all the criteria outlined above, he or she was then assessed using the Vistech VCTS 6500 at 3.05 metres.

Contrast Sensitivity Assessment

The Vistech VCTS 6500 was used to assess contrast in this study. This test was designed to assess contrast sensitivity using gratings of various spatial frequency and different contrasts under a specified illumination.

The Vistech test consists of 40 striped sine wave gratings divided into rows A to E which range from high to low contrast (see Diagram 1). Subjects were asked to identify the orientation of the gratings in each target they could see in each row in turn and to state when they could not see the gratings. The orientation of the gratings was randomised along each row.

The minimum contrast at which each grating could be seen (the contrast threshold), was recorded.

The luminance was checked with the light meter provided with the test and was kept constant from one area of the chart to another and also from one testing session to the next. Each subject was seated 3.05 metres (10 feet) from the chart.

The test was carried out monocularly and binocularly (as previous studies have demonstrated a difference in monocular and binocular scores[10]). The order of testing right eye, left eye or binocularly first was randomised by computer prior to the commencement of the study.

Statistical Analysis

In both groups the mean, maximum and minimum scores, range, and standard deviation

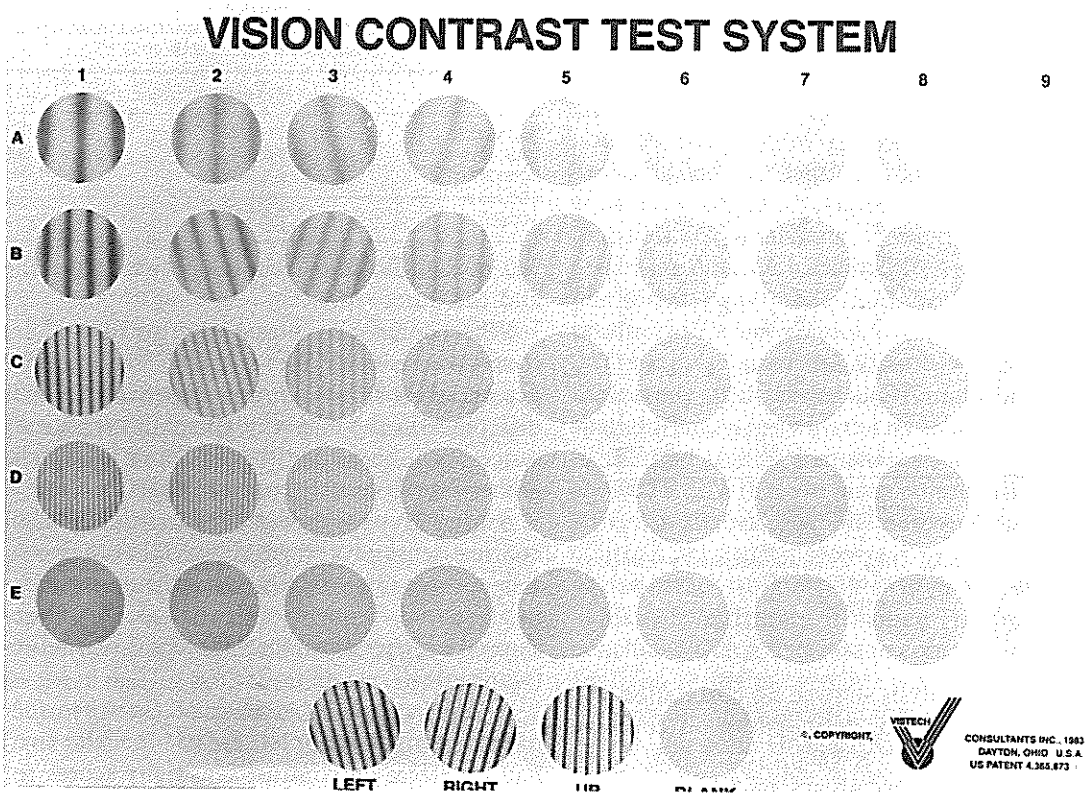

Diagram 1: Vistech VCTS 6500 chart

were calculated for all contrast sensitivity scores. The 5th and 95th percentiles were also calculated to enable direct comparison with the range of normals provided by Vistech. *T*-tests were also performed to compare the means of contrast sensitivity scores. The null hypothesis was that there would be no difference between the means in each group.

A correlation coefficient was calculated to look at the effect of diabetic retinopathy and the duration of the disease on contrast sensitivity function.

A one-way Analysis of Variance (ANOVA) was used to assess if the order of testing each subject's right eye, left eye or both eyes first had any influence on the contrast sensitivity scores for both the diabetic group and the control group.

Significance levels for all statistical tests was $p = 0.01$.

RESULTS

Figure 1 shows the range of normal contrast sensitivity scores using 5th and 95th percentiles for both groups. (For clarity of presentation, only the scores of the left eyes are shown in Figure 1). Diabetic scores falling within the control group range are considered to be normal. The diabetic group show a decrease in scores for the mid to high spatial frequencies, that is 6, 12 and 18 cycles per degree (cpd).

The results of the series of *t*-tests to analyse if there was any difference between the mean contrast sensitivity scores of both groups for each spatial frequency shows a significant decrease in all spatial frequencies for the right eyes and left eyes (see Table 2). Binocularly there was no difference between the two groups in rows A ($p = 0.06$) and B ($p = 0.413$). Otherwise findings were similar to the monocular results.

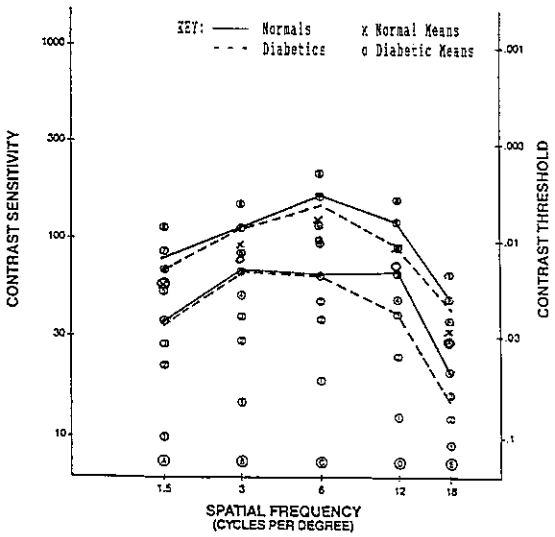

Figure 1: Monocular (LE) Contrast Sensitivity Function Curve on the Vistech VCTS 6500 test; Control versus diabetic subjects.

Statistical analysis showed no correlation for the effect of diabetic retinopathy on contrast sensitivity function for each eye.

The duration of diabetes had a significant effect on binocular scores in Row D ($r = -0.38$, $p = 0.004$). Likewise, a significant relationship was also found for the contrast sensitivity scores of the left eye in row E; ($r = -0.42$, $p < 0.01$). For all scores in row E some correlation coefficient values were significant and others were marginally significant (see Table 3; the negative 'r' values suggest the correlation is weak). There was no significant correlation between duration and the other rows.

TABLE 2
T-test for difference in mean contrast sensitivity scores of control group versus diabetic group; right and left eyes

Row	*t*	*p*
Right Eye		
A	2.88	0.004
B	4.60	0.001
C	4.27	0.001
D	4.87	0.001
E	4.00	0.001
Left Eye		
A	2.54	0.013
B	2.64	0.009
C	6.49	0.001
D	4.63	0.001
E	2.79	0.007

TABLE 3
Correlation showing duration of disease versus contrast sensitivity scores for row E (18 cpd)

	r Value	*p*
Right Eye	−0.23	0.056
Left Eye	−0.42	0.001
Binocular	−0.31	0.015

When comparing monocular and binocular contrast sensitivity scores using 5th and 95th percentiles for both groups there was a subtle difference in the mid to high spatial frequency range with binocular scores being minimally higher (see Figure 2; control group results).

A *t*-test comparing monocular and binocular contrast sensitivity scores showed that there was a significant difference between the mean scores in all spatial frequencies. The binocular scores showed a higher mean than the monocular scores (see Table 4).

A one-way ANOVA, revealed that the order of testing (right eye or left eye or binocular first) had no effect on contrast sensitivity scores for the control group or the diabetic group.

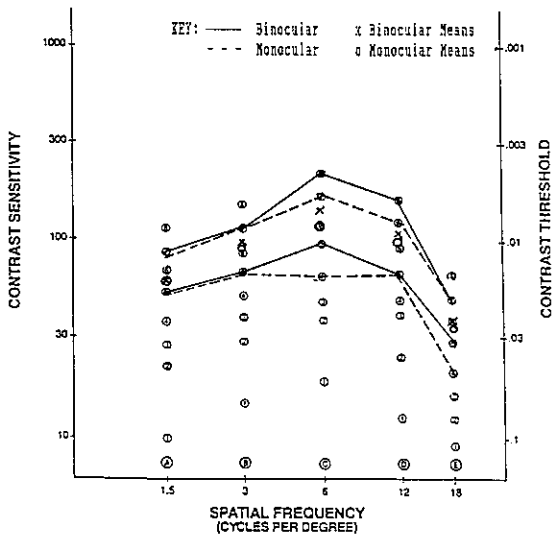

Figure 2: Control Group Contrast Sensitivity Function Curve on the Vistech VCTS 6500 test; Binocular versus Right Eye scores.

TABLE 4
T-test for contrast sensitivity scores of control group; right eye versus binocular

Row	*t*	*p*
A	− 4.39	<0.0001
B	− 4.87	<0.0001
C	−10.56	<0.0001
D	− 9.46	<0.0001
E	− 8.96	<0.0001

The effect of sex on contrast sensitivity scores was assessed using a *t*-test. The control group demonstrated that boys tended to score higher than girls for rows B, C and D ($p<0.01$). There was no effect of sex in the diabetic group.

There was a significant difference between the mean visual acuity for both groups. The diabetic group showed a significantly lower mean for both the right visual acuity and the left visual acuity.

There was no significant difference between the mean age for both the diabetic group and the control group. This was calculated by means of a *t*-test; $p=0.75$.

DISCUSSION

This study has demonstrated that adolescents with diabetes show a significantly reduced mean monocular contrast sensitivity for all rows when compared to age matched non-diabetics. When assessed binocularly they show reduced mean contrast sensitivity scores in the mid to high spatial frequencies only (rows C, D and E).

However, when comparing the contrast sensitivity functions of the diabetic group to the control group using 5th and 95th percentiles, the difference between the contrast sensitivity scores of both groups was not as great as comparing the scores using a *t*-test. At most, 5th and 95th percentiles show a slight decrease in the mid to high spatial frequency range. Unfortunately the score sheets provided with the Vistech VCTS only give 5th and 95th percentiles (means are not supplied). Therefore, clinically, the Vistech only requires the examiner to compare a patient's results to a normal population as marked by 5th and 95th percentiles to determine if scores are abnormal. Although statistically, diabetics may show reduced contrast sensitivity scores, clinically this will not always be apparent.

Investigators have found high spatial frequency loss,[5] low spatial frequency loss,[6] and non-selective spatial frequency loss,[7,8] when assessing contrast sensitivity in diabetics. Sokol et al[4] even found no contrast sensitivity loss in diabetics. Comparisons of contrast sensitivity results are difficult because different procedures, methods, equipment and techniques may produce different results.

Trick et al[7] used the Vistech VCTS 6500 in their study of diabetics and found a loss in all spatial frequencies with the most significant loss in the mid spatial frequency range. They also showed a difference in mean contrast sensitivity scores in diabetics when compared to normal contrast sensitivity scores. The results of the present study support Trick's findings.

Many investigators, including Trick et al[7] have concluded that a decrease in contrast sensitivity function seems to correlate with the severity of retinopathy.[4-9] However, other authors have not found that contrast sensitivity decreases before the presence of visible retinopathy in the diabetic eye.[4,6,7]

The findings in the present study indicate no relationship between the contrast sensitivity scores of diabetic subjects and the presence or absence of diabetic retinopathy. Della Sala et al[6] found no relationship of contrast sensitivity function to retinal pathology unless the diabetic retinopathy was gross. Trick et al[7] found a significant difference between the diabetics with retinopathy and the control group at 6.0 and 12.0 cpd. The subjects in this study had only mild to moderate background retinopathy or no retinopathy, which may account for the fact that no correlation was found.

The present study did show a correlation between contrast sensitivity function and the duration of diabetes for some scores in the high spatial frequency, (row E). This indicated that those subjects who had diabetes for the longest duration tended to score worse in row E. Sokol et al[4] reported a relationship between duration of diabetes and performance on contrast sensitivity tests similar to the relationship found in

the present study. They also reported a correlation between the presence or absence of retinopathy, the duration of diabetes and performance on contrast sensitivity tests. A negative correlation between contrast sensitivity function in row C and duration of diabetes was observed by Trick et al.[7]

One of the requirements for all subjects in this study was that they have a visual acuity of 6/6 or better. However, the diabetic group showed a significant reduction in visual acuity when compared to the control group, even though all subjects had to have what is considered to be "normal visual acuity". Although this visual reduction was revealed statistically, in a clinical situation this subtle difference would not be detected as a result of the gross nature of the Snellen type charts with many charts not testing to 6/4. As an aside it is interesting to note that many clinicians consider 6/6 to be normal visual acuity. These two groups of subjects clearly demonstrate that 6/5 can readily be reached in this age group. Similar findings have been reported in studies on younger age groups of Australian children.[18]

The diabetic group revealed a reduction on contrast sensitivity testing that would be more likely to be detected if scores were statistically analysed. However, as this does not occur in a clinical setting, contrast sensitivity scores would not necessarily alert the clinician to visual dysfunction.

Ross et al[10] reported a slight improvement in contrast sensitivity scores in adults when tested binocularly rather than monocularly. On comparison of binocular scores to monocular scores the results of the present study support Ross' findings. For binocular scores, all spatial frequencies were significantly higher than for monocular scores. As diabetic retinopathy may affect one eye substantially more than the other, these results suggest that, when assessing contrast sensitivity function of diabetics, monocular assessment gives the clinician more information about the individual visual function of each eye. It is also vitally important to compare all results to monocular age-corrected normals.

One finding of interest was that boys' contrast sensitivity scores were minimally better than those of girls when assessed with the Vistech VCTS 6500. A similar finding was reported by Fitzgerald[15] in a study of 606 visually normal children aged 6 to 12 years. Fitzgerald stated that, if there was to be an effect of sex on the outcome, one would have anticipated that girls would have scored better than boys. This is because most other studies on child development show that female children are more developed than male children of the same age.[19]

CONCLUSION

Statistical analysis of results in the present study reveal that adolescent diabetics show a decrease in contrast sensitivity function. Unfortunately the majority of the loss in contrast sensitivity would not be detected in a clinical situation.

The fact that no relationship was found between contrast sensitivity function and diabetic retinopathy, and that contrast sensitivity scores were not always abnormal in patients with known diabetic retinopathy, suggests that the Vistech VCTS 6500 Contrast Sensitivity Test is not suitable to predict which patients have sub clinical diabetic retinopathy or when diabetic adolescents are likely to develop retinopathy.

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PELLI-ROBSON CONTRAST SENSITIVITY ON 122 CHILDREN AGED SIX TO TWELVE YEARS

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Abstract

The importance of comparing the results of contrast sensitivity tests to age related normals has been demonstrated in numerous studies. To date no studies have shown normal results on children using the Pelli-Robson chart.
This study outlines the Pelli-Robson test and gives the results for 122 visually normal children aged between six and twelve years.
Results demonstrated a mean score on the Pelli-Robson chart of 1.861 log units in children of this age group. These findings are similar to those reported for young adults.
Key words: *Pelli-Robson, contrast sensitivity in children, age related normals.*

INTRODUCTION

For many years visual acuity has been routinely assessed using a Snellen's vision chart or its equivalent. The shortcoming with this procedure is that the visual world contains very few high contrast small figures made up of sharp black boarders on a white background. Much of our visual world is comprised of subtle shades of low contrast, different sized objects. Over the last decade it has become possible to measure such low contrast by using contrast sensitivity tests.

Contrast sensitivity testing is a technique which provides a measure of visual sensitivity. There are a number of contrast sensitivity tests commercially available which are quick and simple to administer. These include the Vistech VCTS charts, Vistech Multi Contrast test system (MCTS), Vector Vision CSV 1000 test and the American Optical Test Plates or Arden Gratings. These tests use sine wave gratings, (striped lines) to test contrast sensitivity. These gratings are presented at different orientations and at contrasts varying from almost 100% (black on white) to 'shades of grey'. The width of the gratings is expressed as the number of cycles per degree (cpd) subtended at the nodal point of the eye (Figure 1). This width is known as the spatial frequency.

Grating widths range from high spatial frequencies (for example 18 cpd) which are narrow striped lines to low spatial frequencies which are very widely spaced striped lines (for

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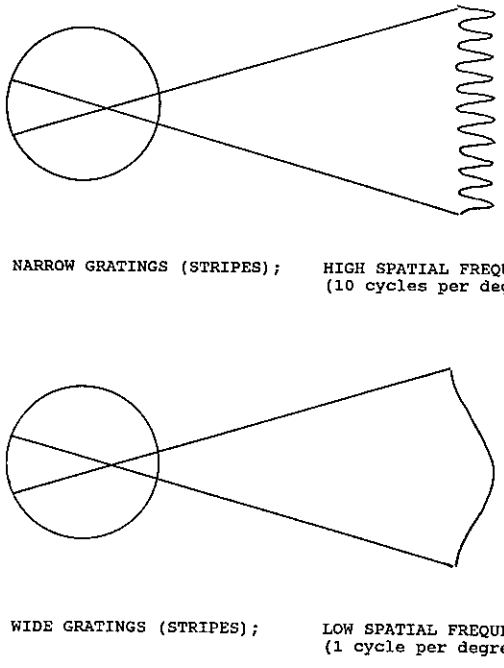

Figure 1: Spatial Frequency. The width of a grating is determined by the number of cycles of the grating per degree (cpd) subtended at the nodal point of the eye. Top: High spatial frequency narrow gratings. Bottom: Low spatial frequency wide gratings.

example 1.5 cpd). For any given grating size (spatial frequency) there is a level of contrast known as contrast threshold below which the grating is invisible. Thus contrast threshold represents the minimum contrast at which that size grating is visible.

When performing the above mentioned contrast sensitivity tests the clinician is measuring the contrast threshold at each different spatial frequency giving a range of results. When plotted on a score sheet a contrast sensitivity function curve is created (Figure 2).

In humans contrast sensitivity is maximum when tested using gratings with a spatial frequency of 6 cpd.[1] This is known as mid to low spatial frequency. Conventional visual assessment using Snellen's charts only give an indication of the patients ability to see high spatial frequencies (at very high contrasts) and they give no information about mid and low spatial frequencies.[1] To obtain accurate measurement of high spatial frequencies, refractive errors must be fully corrected. High spatial frequency gives the clinician information about sight when reading for example.[1] Mid and low spatial frequencies provide information about the visibility of larger objects such as face recognition and the patient's orientation and mobility vision.[2,3,4]

The major difference between the above mentioned contrast sensitivity tests and the Pelli-Robson chart is that the Pelli-Robson chart uses letters of constant size (low spatial frequency; 1 to 2 cpd)[5] rather than gratings that decrease in size. Hence the Pelli-Robson chart gives a single measure of contrast sensitivity (known as peak contrast sensitivity) rather than a contrast sensitivity function curve as seen in Figure 2.

Pelli et al[5] argued that letters were preferable to grating targets because letters were more familiar to patients. This is especially relevant when testing children. Pelli also suggested that, as letters consisted of a mixture of both vertical and horizontal square wave gratings and oblique and curved contours, more of the visual system was tested than the contrast sensitivity tests mentioned above which use sine wave gratings at particular orientations.

The reason that Pelli et al[5] used constant and relatively large sized letters was based on the

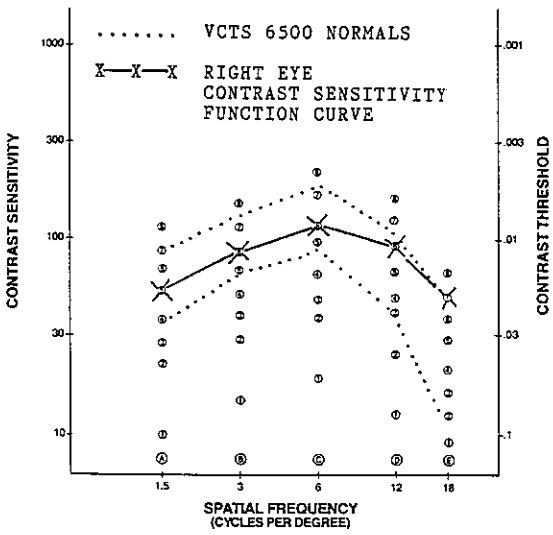

Figure 2: Contrast Sensitivity Function Curve on the Vistech VCTS 6500 test.

finding by Legge et al[6] that contrast sensitivity is reduced at high spatial frequency in subjects with normal vision. As a result, Pelli et al[5] argued that to ascertain if patients with normal vision have abnormal contrast sensitivity, only larger targets (lower spatial frequency) were needed.

The literature reveals that contrast sensitivity is an important diagnostic tool for assessing visual deficit in a number of ocular conditions including amblyopia[7,8,9] refractive error and astigmatism,[10] glaucoma,[11] cataract,[12] macular disease,[13] multiple sclerosis,[14] optic neuritis,[15] corneal oedema[16] and cerebral lesions.[17] In a number of these diseases the visual acuity remains normal.

More recent publications have demonstrated the effect of age on the results of contrast sensitivity tests.[18-29] Searching the literature for population normal scores for the Pelli-Robson chart revealed one publication in which scores for visually normal young adults (mean age 22.5 years; SD ± 4.3 years) and older adults (mean age 70.2 years; SD ± 6.7 years) were reported.[30] The mean score was 1.88 log units or above for young adults and 1.65 log units or above for the older subjects. No studies using the Pelli-Robson chart on children were found on CD Rom Medline searching.

The aim of this paper therefore was to ascertain normal Pelli-Robson chart contrast sensitivity scores for children aged between six and twelve years old.

METHODS

(a) Patient Selection

One hundred and twenty two school children aged between six and twelve years (mean age 8.1; SD ± 1.4 years) had their contrast sensitivity assessed using the Pelli-Robson chart. There were 56 males and 66 females. The children were part of a larger study of children being tested to establish normal levels for the Vector Vision CSV 1000 contrast sensitivity test. (The results of the CSV 1000 test will be the subject of another publication currently in preparation.)[31]

All children attended the same primary school in Western Sydney and their parents had given written permission for their children to be assessed. Children were selected at random from their classrooms. In order to be included in the study all children had to have normal visual acuity (6/6 or better in either eye) without glasses on both the Snellen chart and the logMAR chart, no strabismus and a 550 seconds of arc on the Lang stereo test. Testing was performed with natural pupil size.

(b) Pelli-Robson Chart

The Pelli-Robson chart consists of eight lines of letters. There are two groups of three letters ('triplets') on each line. Letters in each individual 'triplet' are the same contrast. The 'triplet' in the top left hand corner of the chart has the highest contrast (100% contrast) and the 'triplet' in the bottom right hand corner has the lowest contrast (0.9% contrast).[32] The contrast in each successive group decreases by a factor of $1/\sqrt{2}$ (or 0.15 log units) from the top left to the bottom right corner. The letters are all the same size (4.9 cm × 4.9 cm; that is slightly smaller than a Snellen 6/36 letter). When viewed at a distance of one meter each letter subtends 1.5 degrees at the nodal point of the eye. It has been suggested by Pelli et al[5] that the spatial frequency of each letter is between one and two cpd although, as Elliot et al[30] point out this has not been experimentally verified.

The letters used in the Pelli-Robson chart are Sloan letters C, D, H, K, N, O, R, S, V, Z. With the exception of the letter C, all the letters have very high legibility.[33] (The letter C was found to be easily confused with the letter O). Letters are printed on both sides A (chart 4K) and B (chart 2K) of the Pelli-Robson chart. As Elliot et al[30] demonstrated that there was no statistically significant difference between scores on sides A and B all subjects were tested on side A (chart 4K) in this study.

When looking at Figure 3, a Pelli-Robson score sheet, the letters on the second bottom row on the left, for example, 'KCH' have a contrast of 1.80 log units. Those on the right 'ODK' have a contrast of 1.95 log units. (The score sheet shows all the letter 'triplets' on side A of the Pelli-Robson chart printed at 100% contrast. The contrast threshold (log unit) is printed in the margin next to each 'triplet' of letters).

PELLI-ROBSON CONTRAST SENSITIVITY TEST

0.00 V R S	K D R	0.15
0.30 N H C	S O K	0.45
0.60 S C N	O Z V	0.75
0.90 C N H	Z O K	1.05
1.20 N O D	V H R	1.35
1.50 C D N	Z S V	1.65
1.80 K C H	O D K	1.95
2.10 R S Z	H V R	2.25

Right Eye

Log Contrast Sensitivity: _____

Acuity: _____

Correction: _____

Pupil Diameter: _____ mm

0.00 V R S	K D R	0.15
0.30 N H C	S O K	0.45
0.60 S C N	O Z V	0.75
0.90 C N H	Z O K	1.05
1.20 N O D	V H R	1.35
1.50 C D N	Z S V	1.65
1.80 K C H	O D K	1.95
2.10 R S Z	H V R	2.25

Binocular

Log Contrast Sensitivity: _____

Acuity: _____

0.00 V R S	K D R	0.15
0.30 N H C	S O K	0.45
0.60 S C N	O Z V	0.75
0.90 C N H	Z O K	1.05
1.20 N O D	V H R	1.35
1.50 C D N	Z S V	1.65
1.80 K C H	O D K	1.95
2.10 R S Z	H V R	2.25

Left Eye

Log Contrast Sensitivity: _____

Acuity: _____

Correction: _____

Pupil Diameter: _____ mm

Name: _____

Age, Sex: _____

Diagnosis: _____

Medications: _____

Date: _____

Examiner: _____

Comments: _____

PELLI-ROBSON CONTRAST SENSITIVITY CHART 4K. The above log contrast sensitivities are correct to within ±0.05 at the time of calibration of the chart. Copyright © 1988 by Metropia Ltd. Made by Metropia Ltd in U.K. Reorder Cat. No. 7002252 from Clement Clarke Inc, 3128-D East 17th Avenue, Columbus, OH 43219, U.S.A., (800)-848-8923, or Clement Clarke International Ltd, 15 Wigmore Street, London W1H 9LA, U.K., (01)-5808053.

Figure 3: Pelli-Robson Score Sheet. (The numbers in the margins next to each 'triplet' of letters gives the log contrast sensitivity corresponding to the letters in the group. For example, the letters 'KCH' have a score of 1.80 log units.)

(c) Testing Procedure

The Pelli-Robson test was administered in the same way as a Snellen's chart in that the patient was asked to identify the letters in each line of the test. The patients were tested binocularly standing one meter from the chart.

Patients were instructed to read the letters across each row. Children were encouraged to look carefully for letters towards the bottom of the chart. The lowest contrast 'triplets' in which at least two of the three letters were named correctly was recorded as the contrast threshold (as per directions in the manual). Results were recorded on score sheets. The contrast threshold is recorded as the log of the reciprocal of contrast sensitivity. As previously mentioned, on the score sheet this value is printed in the margin next to the 'triplets' of letters (Figure 3).

The light level used for the testing was 85 cd/m^2 which is the level suggested in the manual.

(d) Statistical Methods

For all the visual acuity and contrast sensitivity scores the mean, standard deviation and range were calculated. An analysis of variance (ANOVA) was used to determine the effect of age on scores and a correlation analysis was then done to look for any linear association between age and score. The effect of sex on contrast sensitivity scores was determined by a t test on the population means. The level of probability used was $p<0.05$.

RESULTS

VISUAL ACUITY: The mean visual acuity was 6/5$^+$ for both the right and the left eye with >67% of eyes scoring 6/5. There was no statistically significant difference between the two eyes.

PELLI-ROBSON: The mean contrast threshold was 1.861 + 0.98 log units (second bottom row) with scores ranging from a low of 1.65 log units to a high of 2.10 log units (Table 1).

TABLE 1
Pelli-Robson chart; range and distribution of scores

Score (log units)	Number of children
1.65	9
1.80	56
1.95	52
2.10	2

ANOVA revealed that there was no significant difference in scores for children aged 8 years and over. However, the scores for children aged six and seven years were minimally significantly worse than those aged 8 and over (Table 2).

TABLE 2
Mean scores for age group 6-7 years and age group 8-12 years

Age group (years)	Number of children	Mean score (log units)
6-7	73	1.8452 + 0.96
8-12	49	1.8852 + 0.97

The results of the correlation coefficient analysis demonstrated that there was no linear relationship between the scores from the two age groups ($r = 0.1807$). This suggested that the difference in scores, although significant, was so small that it would not be clinically detectable.

SEX: The t test revealed that the sex of the child did not affect the score. The males ($n = 56$) mean score was 1.8616 log units and females ($n = 66$) mean score was 1.8614 log units; $p = 0.989$.

DISCUSSION

When comparing the results of the present study to the results previously reported by Elliot et al on visually normal adults using the Pelli-Robson chart[30] it is apparent that the scores obtained by the children (mean age 8.1 years) in the present study were similar to the results reported on young adults (mean age 22.5 years). The six to twelve year old children in the present study had a mean Pelli-Robson score of 1.86 + 0.98 log units and the young adults in Elliot's study had a mean Pelli-Robson score 1.88 + 0.88 log units. Thus the present study supports Elliot's finding that the majority of normal patients under the age of 50 will score 1.80 log units or better.

The finding of a minimal effect of age on scores for six and seven year old patients (Table 2) must not be ignored but, as the sample of patients of that age was relatively small, further investigation should be conducted to confirm this.

Previous studies of children's contrast sensitivity have demonstrated that it was essential to use only age related normals as mean scores for children differed from those for adults.[18-29] However, it may be that when measuring peak contrast sensitivity using the Pelli-Robson chart rather than mapping out a contrast sensitivity function curve (using other contrast sensitivity tests), age is not as critical.

Alternately, using letters rather than sine wave gratings is subjectively easier for patients as letters are more familiar[5] therefore when using letters the children's scores may be more easily able to mimic the scores from young adults.

By using the relatively large letters, the Pelli-Robson chart detects contrast while being insensitive to defocus. The Pelli-Robson test measures contrast sensitivity irrespective of the retinal image quality and it is especially insensitive to optical blur.[34] Conversely, the contrast sensitivity tests like the Vistech test were designed to detect the loss of contrast sensitivity that is associated with defocused images due to either optical defocus or pathology[34] as they incorporate high contrast sensitivity gratings. The studies reporting the effect of age on contrast sensitivity using the Vistech contrast sensitivity test found that age had a more marked effect on contrast sensitivity at high spatial frequency,[28,29,35] a spatial frequency which the Pelli-Robson chart does not test.

In this study the sex of the child had no effect on the score. Once again this differs from previous reports where male children have performed better than female children in this age group.[28,29,35]

CONCLUSIONS

This study demonstrated that the mean Pelli-Robson chart score for 122 children aged between six and 12 years was 1.861 log units. This finding was similar to that reported by Elliot et al for visually normal young adults.

The Pelli-Robson chart is quick and easy to administer, especially when compared to the other readily available contrast sensitivity tests. This is because young children are very familiar with letters and the task of naming letters. The Pelli-Robson chart takes less time to administer than the Vistech VCTS test of the Vector vision test. However clinicians must remember that the Pelli-Robson test was not designed to be sensitive to retinal image quality and it is especially insensitive to optical blur.

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ALCOHOL AND VISUAL FUNCTION — AN OVERVIEW

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Abstract

The effects of alcohol on complex hand-eye co-ordination are well known. Research on the ability of alcohol to degrade performance of the visual system, while not extensive, nevertheless allows for critical analysis. Review of this literature indicates that the ocular motor components of visual function are consistently and dose-dependently influenced by alcohol. There is some evidence of impairment of visual acuity and visual field, however the data with respect to the levels at which the deficit is apparent are not clear-cut. There is a need to specify functional implications on tasks requiring hand-eye co-ordination.

Key words: *Vision, ocular motor function, impairment.*

Alcohol is commonly known to affect cognitive and motor performance. Intoxication, even at moderate levels, can impair the ability of an affected person to perform both skilled hand-eye co-ordination tasks such as driving, piloting a plane or operating machinery, and simple daily tasks. The relative contributions of impairment in the perceptual and motor systems to alcohol-induced performance deficits is unknown. In the last 60 years there have been a number of studies of the susceptibility to alcohol of both the sensory and fine motor aspects of visual function. This paper summarises the literature in relation to changes in visual ability at various blood alcohol levels (BALs) and considers the functional implications of these changes to the visual system.

Alcohol is a small water-soluble molecule that penetrates cell membranes at the same rate as water. When administered orally, alcohol is rapidly absorbed into the circulation by diffusion across the gastric and intestinal mucosa.[1] Being a small, readily absorbed molecule that easily crosses membranes, means that alcohol has the potential to and does affect a wide variety of physiological systems.[2] The major site of action of alcohol is the central nervous system (CNS)[2] where clear effects are apparent in those structures that are involved in highly integrated functions such as the reticular activating system.[3] Starmer[4] argued that, broadly, the effects of alcohol can be:

> *"conceptualised as involving alterations of the afferent input from the sense organs and/or changes in the CNS, which confer a potential for disruption of the analysis of sensory*

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information and the control of intricate movement patterns'' (pp103-104).

Many types of tasks have been used to measure the effect of alcohol on cognitive and psychomotor performance. The common finding of these studies has been that there is significant and dose-dependent impairment of performance although differences exist between tasks as to the extent of the impairment.[2,4-7]

Published studies on the influence of alcohol on the visual system commenced around the late 1930s and early 1940s. In one study, copious amounts of spirits were consumed in the relaxed setting of a cocktail party and participants were tested for a variety of changes in their visual function over the course of the evening.[8] Fortunately, more recent studies have adopted more rigorous approaches to experimental design, subject selection and statistical analysis.

Vision and Alcohol

Static visual acuity has been found to be resistant to alcohol, even when low contrast stimuli have been used. No conclusive impairment of static visual acuity has been obtained at low BALs.[9] Some impairment of static vision is found at moderate BALs.[10-13] However a number of researchers have found no change even at moderate and high BALs.[14,15] Only one study has investigated dynamic visual acuity, the impairment of which is arguably more important in driving. Honegger, Kampschulte and Klein[16] used a visual tracking device in which a single letter was projected on a screen and then rotated in a circle at selected speeds. In this study it was shown that dynamic visual acuity is significantly reduced while alcohol levels are rising and begins to improve once BAL starts to decrease. Subjects with low BALs and no reported subjective feelings of intoxication, had significantly impaired dynamic visual acuity.

Restriction of the visual field could be considered to present a major hazard to driving. Except at relatively high doses of alcohol (0.10%) there does not appear to be any appreciable reduction in the extent of the lateral visual field.[8,15,17,18] Interest has moved to visual field examination tasks that require divided attention, that is time-sharing of the fovea and an extra foveal task more or less simultaneously and the processing of information. Von Wright and Mikkonen[19] found significant performance deficits at a BAL of approximately 0.05% when tracking and visual recognition tasks were combined. Moskowitz et al[14] examined the detection of peripheral stimuli. Alcohol was found to impair the central processing of peripheral visual information when processing of that information conflicted with processing of information from other sources. The deficit appears to be in the ability to divide attention and process information. It was concluded that the effect of alcohol on peripheral vision was a function of the information load on central vision.

Dark adaptation, in terms of detection of low contrast targets, is not impaired at low or moderate BALs.[14,20] Reduction of glare resistance has been cited as a potential driving hazard especially at night, although no studies provide strong evidence for a consistent influence of alcohol upon glare tolerance, resistance or receovery.[10,12]

Critical flicker fusion refers to the transition point at which a rapidly flickering light source is first perceived as continuous. This function has been used as an index of the temporal resolution of the visual system as well as an indicator of central nervous system function. The literature offers differing conclusions concerning the effects of moderate levels of alcohol. The results indicate impairment at moderate to high BALs,[21,22] but below these levels the findings are inconsistent. The concern here is that in the general population the range of normal results has not been clearly defined.

The literature is inconsistent on the impact of alcohol on accommodation, with one report of a decreased stimulus AC/A ratio[23] and controversy regarding the change to accommodative amplitude.[15,24]

Ocular Motor Function and Alcohol

The literature is consistent in reporting that the effects of low doses of alcohol are apparently capable of producing marked decrements in

ocular motor function. Numerous studies have reported a significant esophoric shift in distance measurement and an exophoric shift for near measurement.[8,11,13-15,20,25-28] Change at distance is usually greater than at near and can be seen even with low BALs. Vertical heterophoria is not induced or altered.[11,14,27]

Deterioration of motor and sensory fusional ability results from the ingestion of alcohol, and this is manifested as a loss of abduction power.[11,14,23,26,28] The decrement of the convergence near point is associated with a reduction in the fusion ability as reported by several studies.[13,15,23,28]

Defects in binocular co-ordination could be expected to cause impairment of depth perception Wist et al[29] reported that moderate BALs were associated with a significant increase in fixation disparity, but stereoacuity was unaffected at this level. Hill and Toffolon[15] confirmed this finding showing no significant change in stereoacuity in their study.

There are few reports in the literature regarding the influence of alcohol on the saccadic and smooth pursuit eye movement systems prior to 1974. A typical saccade is initiated approximately 180 milliseconds after the target stimulus is activated and peak velocity is rapidly achieved. Normal subjects can miss the target by undershooting or overshooting which necessitates a second corrective saccade. Peak velocity increases with the amplitude of the movement, reaching a maximum at saccades of approximately 30 degrees. These properties make saccades the fastest and best controlled movements of which the body is capable.

A number of recent studies have found the tendency for saccadic peak velocity for saccades of 20 degree amplitude to be reduced by between 7 and 25% at BALs of 0.05% and above.[28,30-33] Methodological and instrumentation limitations have made definitive measurement of changes in latency difficult, however Katoh[33] has reported an increase in the latency of saccades of between 8 and 17%. According to Wilkinson et al,[30] smooth eye movements become jerky after alcohol with catch up saccades being required to continue the pursuit eye movements.

The presence of nystagmus after alcohol ingestion is well known and in fact forms the basis of a roadside sobriety testing device which has been recently piloted in a number of states in the USA.[34] Howells[35] examined alcohol-induced nystagmus and reported that all subjects demonstrated nystagmus after 50 mL of absolute alcohol but at varying durations post ingestion. Seedorff[26] accounted for nystagmus in terms of the action of ethanol on the vestibular system and cerebellum.

DISCUSSION

From this review of the literature it can be seen that the visual and ocular functions most consistently and significantly influenced are specific components of the ocular motor system. There is a change to the static deviation with an increase in esophoria at distance, and increase in exophoria at near. Motor fusional reserves and the ability to converge at near range are impaired. In addition, there are reported changes in saccadic latency and velocity and a reduction in ability to conduct smooth pursuit eye movements.

It can be argued on the basis of these results that the reported nystagmus is either a direct result of decrements to the saccadic and smooth pursuit systems, or that the measured and reported changes in these functions of eye movement are simply manifestations of the nystagmus. Whichever it is, it is not possible simply on the basis of these results to specify the affected pathway for eye movement, nor is it possible to determine the functional effect on motor behaviour without additional performance testing. It is reasonable to assume that the oscillopsia known to be produced by acquired nystagmus would almost certainly lead to reduced visually driven motor performance, but there is little evidence in the literature.

The ocular motor system exists to allow shifts in visual direction and to maintain comfortable binocular single vision and smooth conjugate eye movement control. Whether a change in the heterophoria position will alter judgement perceptions as Wilson and Mitchell[13] have suggested is unclear given the available data. The slowing of a 20 degree horizontal saccade to a peripheral stimulus

may mean the difference between a quick response to danger or not, but is yet to be proven. The point at which the alcohol dose dependent decrement in the binocular system will mean an impairment in binocular performance is also yet to be established.

The other issues that need to be addressed are the complex perceptual and cognitive aspects of visuomotor activity before any meaningful decisions regarding the functional implications of ocular motor change can be determined. It would appear that the studies conducted by Moskowitz et al[14] requiring a divided attention response to visual field testing move closer to incorporating visuomotor aspects of function with perceptual responses also. Complex tasks such as driving place demands not only on the visual act but on the perceptual ability of the driver who in turn must produce a set of appropriate motor responses and behaviours. To extrapolate a direct result on the ability to drive or produce any other set of behaviours from the available data on visual function is clearly inappropriate and methodologically unsound. Many of the studies do attempt to do this even if only by inference.

CONCLUSION

The functional implications of the observed changes in the visual system as a result of alcohol ingestion have yet to be established. Hand-eye co-ordination tasks involve the interaction of a number of physical and cognitive systems so isolating the influence of visual function on these tasks would be very difficult. Without further studies which address the complex inter-relationships between afferent and efferent systems it can only be hypothesised that these observed changes will influence the overall performance decrement of the intoxicated person.

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VISION AND OCULAR FUNCTIONS IN THE OLDER DRIVER

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Abstract

The driving ability of the older driver is often controversial and many driver licencing bodies recheck the vision of older drivers prior to renewal of their drivers licence. Currently in Victoria, the vision of drivers is not rechecked routinely on renewal of a drivers licence. This study presents information on the visual status of 201 older drivers in Victoria using an automated vision screener. It was found that 91.5% of those screened had an unacceptable result to one or more of the tests of ocular function. By examining the incidence of ocular dysfunction in this group, the acceptability of the present Victorian regulations will be considered.

Key words: *Vision screener, driving, vision, ocular function.*

INTRODUCTION

Recent demographic trends indicate that the number of aged people is increasing in some countries. Accordingly, the number of older drivers on the roads is also increasing. It has been discussed by Klein[1] that advancing age, particularly after the age of 50, brings about a number of physiological changes including the decline of vision and other ocular functions. As a result of these changes Klein further points out that there is continued need to evaluate the affect of declining visual performance and driving ability, given the high relationship between road accidents and older drivers.[1] These links have caused many driver licencing authorities to consider ways in which the licencing of older drivers can be regulated.

A worldwide review of visual standards for drivers conducted by Charman[2] in 1985, revealed that many countries recheck the vision of older drivers as a means of regulating the renewal of licences. In Australia, except for Victoria and the Northern Territory, the vision of drivers over the age of 70 is tested prior to renewal of a licence. Currently in Victoria drivers of a private vehicle are only required to have 6/12 vision in the better eye and 6/60 vision in the worse eye. It is the responsibility of the driver to report to the licencing body any physical changes, such as reduced or impaired vision.

When attempting to calculate the accident risk for drivers a number of factors need to be considered such as the drivers age, the number of accidents and the number of miles driven. Research by Waller[3] and Barr[4] indicates that the accident risk for the older driver is higher than for any other group of drivers. This is despite the fact that older drivers avoid demanding driving situations such as heavy traffic or night driving. Keltner and Johnson[5] found that the driver over age 65 presented with the highest risk value. Barr[4] further found that an increase in the likelihood for injury, or for a fatality following an accident, occurs in the elderly driver.

Research into the vision of drivers has found that many drivers have a level of vision that is less

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than the recommended level and that age is linked to this decline. Guest and Jennings[6] surveyed Victorian drivers presenting to optometric practices and found that 12.3% could not reach the recommended visual standard and that 32.2% of those over the age of 70 were below the standard. A survey of the vision of some 503 Brisbane drivers attending specialist, but not eye outpatient departments, performed by McConnell et al[7] found that 8% failed to meet that states vision requirement and that 55% in this unsatisfactory group were aged between 56-75 years.

However when attempting to determine the visual capacity of drivers it is preferable to survey a population of drivers with no bias towards possible dysfunction as might be the case in these studies. For this reason, it was decided to study a group of older drivers in a non-clinical setting using an automated vision screener to test a range of ocular functions including visual acuity. The value of vision screeners when examining ocular functions in a large population is widely accepted and according to Unger[8] they are used in many countries to assess the visual capacity of drivers. The aims of this study were:

1. to provide information on the visual status of a group of drivers over the age of 50 using the automated Keystone View VS 11 Vision Screener
2. to determine if the visual capacity of these drivers complies with the standard recommended for drivers in the state of Victoria
3. to consider if re-assessment of older drivers vision should be performed at the time of licence renewal
4. to identify the incidence of ocular dysfunction in this age group as detected by a vision screener.

METHOD

Subjects

All volunteers screened on the vision screener were over the age of 50. They held a current Victorian drivers licence for a private vehicle and had driven in the week prior to testing.

Apparatus

All candidates were screened using the Keystone View VS II Vision Screener. This instrument allows screening of the following ocular functions:

- visual acuity, right, left and both eyes together
- heterophoria, horizontal and vertical
- fusional ability
- stereopsis

Each of these functions were tested for near (40 cm) and far (6 m). This instrument also assesses colour vision and horizontal peripheral visual fields. A description of this vision screener has previously been outlined.[9]

Procedure

The drivers were screened at one of five venues, which were either a metropolitan bowling club or Senior Citizens club. One tester operated the instrument at all locations and testing took between 10-15 minutes for each individual. The testing was performed between June and August 1992. Candidates were tested with glasses if they drove with their glasses, even if this was not a condition of their driving licence.

RESULTS

Two hundred and one drivers were assessed on the screener, 109 males and 92 females. Their ages ranged from 50 to 87 years, the average age being 66.9 years. Table 1 shows the age profile of those screened.

Vision

Vision was tested monocularly and with both eyes open at 6 m and 40 cm. It was found that 53 subjects (26.4%) had 6/6 vision in the right eye and 56 subjects (27.9%) had 6/6 vision in the left eye. With both eyes open 77 subjects (38.3%) demonstrated 6/6 vision. Seventy eight subjects (38.8%) had 6/6 vision in at least one eye.

TABLE 1
Age profile of sample $n = 201$

Age	Number	%
50-59	46	22.9
60-69	72	35.8
70+	83	41.3

The testing of near vision revealed that 37 subjects (18.4%) had 6/6 equivalent in the right eye and 33 subjects (16.4%) in the left. With both eyes open 45 subjects (22.4%) had 6/6 equivalent.

Heterophoria

The test of heterophoria type and measurement revealed that 184 of those screened (91.5%) were within the normal limits for horizontal heterophoria for far, and 183 (91%) for vertical heterophoria.

At near, 166 candidates (82.6%) were within normal limits for horizontal heterophoria and 174 candidates (86.6%) for vertical heterophoria. Table 2 shows the types and numbers of heterophoria found on those screened.

TABLE 2
Number and types of heterophorias

	Far		Near	
Horizontal phoria	eso	20	eso	8
	orthophoric	129	orthophoric	58
	exo	35	exo	100
	suppression	14	suppression	11
	eso >6Δ	3*	eso >4Δ	3*
	exo >4Δ	0	exo >6Δ	21*
Vertical phoria	R/L	1	1	
	orthophoric	107	82	
	L/R	75	91	
	suppression	14	11	
	>1ΔR/L	1*	1*	
	>1ΔL/R	3*	15*	

*Unsatisfactory.

Fusion

A capacity for fusion was demonstrated in 171 candidates (85.1%) for far, and for near in 142 candidates (70.6%).

Stereopsis

Stereopsis for far was within the acceptable range for 75 candidates (37.3%) and for near in 47 candidates (23.4%).

Colour

The results to the colour discrimination test were acceptable for 159 candidates (79.1%) for red/green and for 137 candidates (68.2%) for blue/violet.

Horizontal Peripheral Vision

The assessment of horizontal peripheral vision screens up to 85° on the temporal side and 45° on the nasal side and was satisfactory in 167 candidates (83%).

Suppression

A group of 20 candidates (9.95%) exhibited suppression during testing in one or more tests. In this group 18 candidates (90%) had unacceptable fusion results. Unacceptable levels of stereopsis were found in all candidates with suppression, 14 candidates (70%) having no detectable stereopsis for near and far.

SUMMARY OF RESULTS

A review of the vision results and the performance to each ocular function test was undertaken to ascertain the significance of the results in these older drivers. The results to distance vision testing were examined to see how many of those screened were driving with a level of vision that was below the VicRoads recommended standard. It was found that 18 subjects (8.9%) had less than 6/12 vision in their better eye. Consequently, it could be suggested that these drivers should not be driving as they would in all probability fail a VicRoads eye sight test. The results of vision testing for the three major age groups showed that of those who had a level of vision below that recommended for a drivers licence in Victoria, 16 candidates (88.8%) were over 60 years of age. These results support the fact that the older the driver the greater the need to monitor the level of vision (see Table 3).

A review of the drivers performance to each test of ocular function shows that 184 of those screened (91.5%) had unacceptable results in one or more tests. In this group, 160 candidates (79.6%) had unacceptable results in two or more tests and 130 candidates (64.6%) had unaccept-

TABLE 3
Age of drivers failing vision standard

50-59	2	11.2%
60-69	8	44.4%
70+	8	44.4%
	18	100%

able results in three or more tests. Furthermore, 66 candidates (32.8%) had unacceptable results in half or more of the ocular tests.

A comparison between the results in this study to those found in young learner drivers highlights the poor performance of these older drivers[9] (see Table 4). The previous study screened 727 driver licence applicants using the same automated vision screener and a comparison of the two sets of data shows that the number of unacceptable results is much greater in the older drivers. The results of all tests of ocular function as tested on the automated vision screener show that the numbers of drivers in this older age group who had unsatisfactory results is much higher.

The findings in this study regarding the visual status of older drivers are of concern as they indicate that the majority of those screened had evidence of visual and ocular dysfunction that could contribute to difficulties when driving. Further research into the effects of reduced visual and ocular performance on driving need to be carried out. The results provide support for the introduction of retesting of drivers vision on renewal of a drivers licence, particularly for those drivers over the age of 60.

TABLE 4
Number of drivers with unacceptable test results

	Older Drivers $n=201$	Younger Drivers $n=727$
≤1 test	184 (91.5%)	20.4%
≤2 tests	160 (79.6%)	14%
≤3 tests	130 (64.6%)	4.1%

CONCLUSION

The results of screening a group of older drivers with an automated vision screener showed:

- that the majority of drivers, 61.7% did not have 6/6 vision in at least one eye
- that 8.9% of those screened demonstrated a level of vision below the standard recommended to hold a drivers licence
- that in this fail group, 88.8% were over 60 years of age
- that assessment of drivers vision should be performed at the time of renewal of a drivers licence. The results indicate that the vision retest could be required only in drivers over the age of 60 as this group represents those with the greatest level of reduced vision
- that the incidence of ocular dysfunction in this group of older adults was considerably high, with 91.5% exhibiting an unacceptable result to one or more tests of ocular function
- that licencing authorities could consider the value of examining a wide range of ocular functions in drivers.

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DIPLOPIA AND DRIVING

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Abstract

The effects of diplopia on driving skills are described, based on the responses of subjects in two groups. Group 1 (12 subjects) had artifically produced diplopia. Group 2 (three subjects) had diplopia associated with a medical condition. Results showed that diplopia reduces the accuracy and judgement skills of drivers regardless of the cause of the diplopia.
Key words: *Diplopia, driving skills.*

INTRODUCTION

Diplopia is described by Duke Elder[1] as having an affect that is "usually so disturbing that the simultaneous activities of the eyes cannot be tolerated". In the complex activity of driving where sensory and motor functions are required to work in harmony and at a high level, diplopia must affect driving ability.

Little has been recorded about the effects that diplopia has on the driving skill. Currently the presence of diplopia does not preclude a driver from holding a driver's licence.

METHOD

In order to investigate the effects that diplopia has on driving skills the responses of two groups are reported. The first had diplopia artifically created with the use of press-on prisms on plano glasses (normal group) and their driving skills in the presence of the diplopia was evaluated. The second group comprised three subjects who were being evaluated for their ability to drive and who had diplopia caused by a medical condition or injury.

ARTIFICIAL DIPLOPIA (Normal Group)

Diplopia was artifically produced in 12 subjects who had normal binocular single vision and then each persons' driving skill was tested over a set driving route.

The 12 subjects in the study were six males and six females between the ages of 18 and 30 years.

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All subjects were physically fit, had held a licence for at least one year and had driven regularly. Each subject was given an eight dioptre fresnel prism placed base in on both the left and right lens of a pair of plano glasses. These prisms caused artificial horizontal diplopia which could not be fused.

Each subject drove over two courses which had been selected for their similarity. Each course had three right corners, three left corners, straight stretches of road, the need to pass parked as well as moving vehicles and a laned section of road. All subjects commenced with course one. Half the subjects drove course one with diplopia created by the prisms and then course two without the prisms so that they had binocular single vision. The other half of the subjects drove course one with binocular single vision and course two with diplopia.

All subjects were assessed under the same conditions which included driving in a car that was unfamiliar to them; in lighting conditions that were constant, i.e. full sunlight; and on quiet back streets of a Sydney suburb. To ensure the safety of each subject the test was carried out in a car which had dual controls and in the presence of a fully qualified driving instructor whose role was to take over control of the car in the likelihood of any danger, collision or loss of control. All subjects were informed that they had the right to terminate the testing procedure at any time.

The test procedure involved each subject driving the test route whilst using binocular single vision. This took five minutes and enabled them to become familiar with the car. They were then directed by the driving instructor to follow the test route and to maintain a constant speed. Two responses were observed:

1. *Driving Speed*
   Three speed levels were recorded during each of the following activities, right hand turns, left hand turns, driving on a straight stretch of road, driving past parked vehicles and driving past moving vehicles. The speed levels for each activity were averaged in order to compare each driver's response in the diplopic and single vision state. If the subject closed one eye, stopped when it was unnecessary or refused to continue with the course they were given a speed score of zero.
2. *Driving Accuracy*
   This was recorded in two situations. The first situation was along 500 metres of a two lane road whilst maintaining a constant speed of 50 km/h. Each time the driver deviated from the lane one point was deducted from a total score of 10. This score was changed into a percentage and the response compared between the diplopic and binocular single vision state.

The second situation examined the ability of the driver to weave the car around six witches hats. The hats were placed in a straight line with a gap between each hat. The driver had to maintain a constant speed of between 15-20 km/h and make no errors. A point score system was used. One point was deducted from a total of six, when either a hat was hit or the driver failed to successfully drive around a hat.

If at any stage the subject shut one eye to overcome the diplopia their score was recorded as zero.

RESULTS (Normal Group)

The speed at which all the subjects drove for each assessed activity was averaged and the results of speed with diplopia was compared to the average speed with binocular single vision. Table 1 and Figure 1 show the results. In all situations the speed at which the driver travelled when diplopia was present was less than the speed at which the same driver travelled when binocular single vision was present. In each situation the difference was statistically significant with the t test showing the p value to be 0.00001.

TABLE 1
Comparison of speed with and without diplopia

	Corners	Straight ahead	Parked vehicles	Moving vehicles
Speed with single vision km/h	22.06	60.05	52.22	50.84
Speed with diplopia km/h	10.53	31.87	15.87	16.72

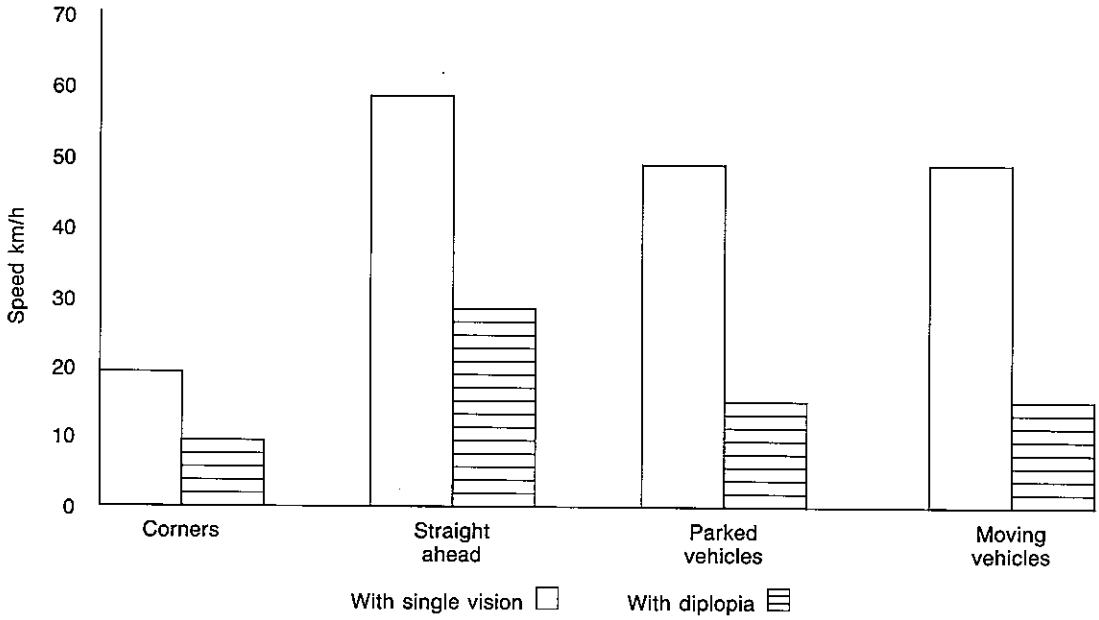

Figure 1: Comparison of speed with and without diplopia.

Accuracy was determined by the ability to perform each of two tasks without error. In the first task of weaving around the witches hats, each driver gained six out of six possible points when they were using binocular single vision. An average score of five out of six possible points was scored when the subjects performed the task in the presence of diplopia. The difference was shown by the t test to have a p value of 0.0069.

In the second task the ability to remain within marked lanes over a 500 metre straight stretch of road was tested. Each driver gained 10 points out of a possible score of 10, when they used binocular single vision. In the presence of diplopia the average score dropped to 4.5. By the t test the p value was 0.00001.

DIPLOPIA IN DRIVER REHABILITATION

It is fully recognised that the experimental situation does not represent the circumstances that occurs in clinical practice for patients with diplopia caused by ocular motor nerve or extra ocular muscle damage. For instance in the experimental situation prisms were used to produce the diplopia and these produced a reduction in vision to approximately 6/9. Additionally the diplopia was not associated with any illness or injury so did not have any accompanying disorientation related to reduced bodily function. It is also unusual for a person with diplopia which has just occurred to immediately drive. Normally some adaptation time would occur. Therefore these factors raised doubt about the true effect that diplopia will have in the driving situation as well as the need to test drivers' abilities in the presence of diplopia.

Three subjects presented to the Driver Rehabilitation Centre at Cumberland College as part of the regular assessment programme for drivers with physical handicap. Each one had diplopia. The results of their on-road tests are proposed as evidence of how diplopia may affect driving skills.

SUMMARY OF THE OCULAR CONDITIONS OF THE SUBJECTS WITH DIPLOPIA

Subject 1

Aged 23 years, had a medical history of removal of an astrocytoma from the right side of the cerebellum.

Visual acuity • right 6/12, left 6/9, binocularly 6/9
Visual fields • normal
Ocular condition • major muscle underaction of left superior oblique; nystagmus, left beating in the primary position and elevation, rotary on right and left gaze, less on extreme depression; saccades hypometric and "zig-zag" on elevation
Symptoms • DIPLOPIA constant for eight months, maximal in the field of the left superior oblique.

Subject 2
Aged 16 years, had a medical history of head injury.
Visual acuity • right 6/4, left 6/5, binocularly 6/4
Visual fields • normal
Ocular condition • left esotropia and left hypotropia; nystagmus on elevation, right beating to the right and left beating to the left; saccades, "zig-zag" movement on elevation
Symptoms • DIPLOPIA in all positions of gaze for 12 months.

Subject 3
Aged 71 years, medical history of cerebrovascular accident and cardiac problems.
Visual acuity • right 6/9, left 6/6, binocularly 6/6
Visual fields • normal
Ocular condition • major muscle underaction, right superior rectus compensated with head posture to right shoulder
Symptoms • DIPLOPIA, present on right gaze for at least 12 months; difficulty looking at the dashboard; problems with "changes in the level of the road surface".

During the on-road assessment overall speed levels were noted, for instance if the client constantly exceeded or travelled well below the limit. Averaged responses for specific activities were not recorded and therefore speed patterns in these clients could not be directly compared to the patterns reported in the normal group.

Accuracy in driving performance was routinely noted in each client during the on-road assessment, particularly with reference to judgement and positioning.

Judgement was noted in relation to:
- timing, when braking at corners and at intersections, which was either too early or too late
- timing accuracy for lane change.

Positioning was noted in relation to:
- the car being kept to the left of the centre of the road
- staying within marked lanes
- placement of the car when turning corners
- placement of the car at intersections.

RESULTS

Table 2 summarises the problems experienced by each subject.

Judgement errors included braking too late when turning into corners, braking for no reason, premature changing of position to avoid a parked car, merging too late and, braking too early or too late at intersections.

Positioning problems included turning on the wrong side of a silent cop, driving too close to the gutter, losing position on lane change and steering into the gutter after turning corners and at curves.

Other problems encountered included failure to check the blind spot on the side of the diplopia and failure to identify a sign on the side of the diplopia.

The problems experienced when driving were eliminated when the diplopia was corrected by

TABLE 2
Driving problems in the presence of diplopia

Driving problems	Case 1	Case 2	Case 3
Judgement errors	At corners Premature lane change	Intersection	Right merge late
Positioning errors	Wrong side silent cop Lost position on lane change	At corners and curves	
Other			Could not check: — Blind spot to right — Signs on right

either prisms or a graded filter over the non dominant eye. The severity of the problems encountered by the clients was in proportion to the existence of the diplopia. For instance Case 3, who had diplopia on right gaze, only had problems with actions involving the right side (checking the blind spot on the right, merging to the right and identifying signs). The other clients experienced problems in all positions of gaze.

DISCUSSION

The results from the study where diplopia was artificially created clearly demonstrated that induced diplopia caused a change in driving skill in two observable areas. It reduced the speed at which they drove and diplopia also caused the subjects to be less accurate in positioning the car. The influence of diplopia on driving speed was not as closely studied in the group with acquired diplopia. However, attention to such areas as monitoring the speed at which subjects with acquired diplopia negotiate corners could be more closely monitored to see if a similar decrease in speed occurs. Alternatively, encouraging subjects with diplopia to maintain a constant speed may highlight the problems that they experience and which may need to be resolved.

In the group with acquired diplopia other problems, such as judgement difficulties, were observed during an on-road test. This highlights the need to study the effects of visual problems on judgement in the driving situation, and under controlled conditions.

Regardless of the origin of the diplopia the subjects in both groups experienced problems when driving. Some of the problems demonstrated by the clients with acquired diplopia were similar to the responses observed in the study group where diplopia was artificially induced. For instance the subjects with acquired diplopia had problems keeping the car to the left of the centre of the road and staying within marked lanes. This was similar to the problems experienced in the group with artificially induced diplopia where the ability to stay within marked lanes decreased in the presence of diplopia. Also the subjects with acquired diplopia had problems accurately placing the car when turning corners. This is similar to the reduced ability of the study group to steer around witches hats in the presence of diplopia.

Although the number with acquired diplopia is small and cannot be accurately compared with the experimental group, the responses suggest that the problems found in the study group are similar to the problems experienced by people with diplopia associated with medical conditions. Thus further studies on the effects of experimentally induced diplopia on driving performance where the variables can be controlled may enhance our knowledge of the effects of diplopia generally.

The combination of, diplopia causing problems in the driving situation and its elimination leading to improved driving performance, emphasises the need of eye care personnel to ensure that patients seen in the clinic who have diplopia and who wish to drive accurately, are assisted to gain a single image.

More extensive research into the effects of diplopia in the driving situation is important.

CONCLUSION

The evaluation of driving skills of subjects with diplopia supports the notion that the presence of diplopia does adversely affect driving ability. This study, although limited, highlights the need to provide methods to overcome the diplopia in drivers as well as the need for further research in the area.

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Case Reports

PARTIAL SIXTH NERVE PALSY RESULTING FROM SPINAL ANAESTHESIA — A CASE STUDY

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Abstract

Sixth Nerve palsies have been reported following spinal anaesthesia these are rare and often incomplete. A 36 year old woman presented with a severe headache and diplopia after delivery of her second child for which she underwent two attempted spinal anaesthesia. A sixth nerve palsy had developed ten days after the delivery and resolved by the fourteenth week.
Mechanical theories present the most plausible explanations for the cause of the sixth nerve palsy. However, none of the theories adequately explain why the palsy is more likely to occur unilaterally than bilaterally.
Key words: *Abducens nerve, epidural anaesthesia, lumbar puncture, esotropia.*

INTRODUCTION

This paper describes a case history of the rare association between spinal anaesthetics and sixth nerve palsy. Sixth nerve palsies have been reported[1-5] following spinal anaesthesia these are rare and often incomplete.[5] Patients usually recover within a matter of weeks.[1-5]

Before discussing this case history, it is useful to review the course of the sixth nerve in order to understand the theories that may explain the cause of this palsy and its association with spinal anaesthesia.

The abducens (sixth) nerves rise from the lower border of the pons and the lateral part of the pyramids. They are about ten millimetres apart with the basilar artery lying between them. The two nerves are then crossed by the anterior inferior cerebellar arteries (Figure 1). The nerves travel superiorly, ventrally and laterally in the posterior cranial fossa for approximately fifteen millimetres before piercing the dura. From here they run superiorly along the back of the petrous temporal bone over its apex (Figure 2) and forward through the cavernous sinuses into each of the orbits via the superior orbital fissure to the lateral rectus.[6]

CASE HISTORY

A 36 year old woman during delivery of her second child underwent two failed attempts of dural taps to administer epidural anaesthetic. This procedure was abandoned when the anaesthetist observed leakage of cerebral spinal fluid. Lack of progress in the labour lead to a general anaesthetic being given for a caesarian section.

After delivery, the patient developed severe generalised headaches, neck pain and photophobia. Ten days post partum she developed horizontal diplopia, initially intermittent, and then constant for distant fixation and on left gaze.

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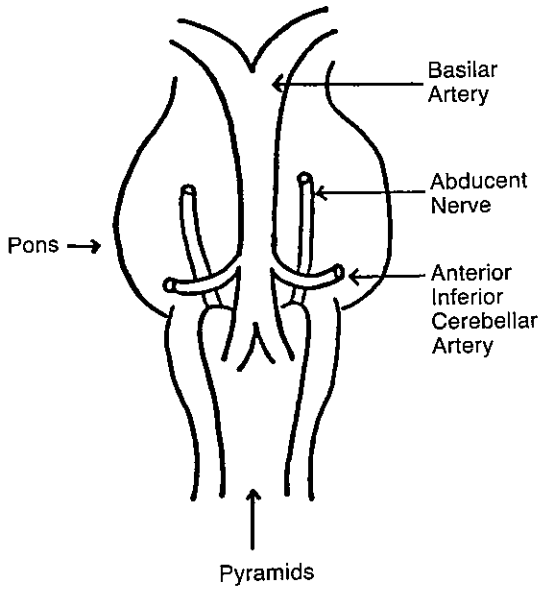

Figure 1: Schematic diagram illustrating the relationship between the abducens nerve, basilar artery and the anterior inferior cerebellar artery — ventral view.

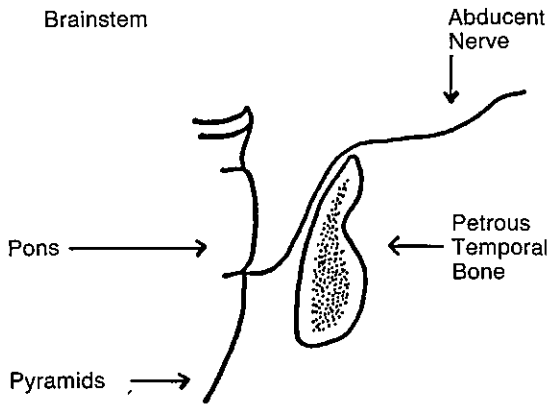

Figure 2: Schematic diagram illustrating the relationship between the petrous temporal bone and the abducens nerve — lateral view.

METHODS

The following are details of ocular examinations conducted over fourteen weeks and demonstrate the rate of recovery (Figure 3).

Two Weeks Post Partum

The patient presented with a severe constant headache which was relieved by lying down.

General health was good, and in particular there was no diabetes or other conditions to account for the palsy.

Visual acuity without glasses was 6/12 right eye and 6/9 left eye. Visual acuity with pin hole was 6/9 right and left eyes. Pupils were equal and reactive. Cover test distance demonstrated a large left alternating esotropia 30$^{\Delta}$ base out. Cover test near indicated a small esophoria 4$^{\Delta}$ base out. Extraocular movements indicated under action of the left lateral rectus. Examination showed that the Fundus was normal.

Three Weeks Post Partum

The headache had improved although there was no change in the patient's strabismus. At this stage the patient was referred to a neurologist.

Full neurological examination, including a Computed Tomography Scan (C.T. scan), revealed no abnormal neurological signs apart from a sixth nerve palsy. There was no neurological evidence to explain the presence of a sixth nerve palsy (e.g. tumour, lesion, etc.).

The neurologist's report also noted that the patient had experienced some initial neck pain and photophobia.

Five Weeks Post Partum

The patient's headache, neck pain and photophobia resolved. The esotropia showed improvement.

Prism cover test distance demonstrated a large left alternating esotropia 20$^{\Delta}$ base out. The prism cover test near remained unchanged with a small esophoria 4$^{\Delta}$ base out.

Eight Weeks Post Partum

Diplopia was not evident in the primary position. However, there was still some diplopia in left gaze.

Prism cover test distance disclosed a small esophoria 6$^{\Delta}$ base out. Prism cover test near was stable. Extra-ocular movements revealed slight under action of the left lateral rectus.

Fourteen Weeks Post Partum

The patient was now fully recovered.

Uncorrected visual acuity was 6/6 right and left eyes. Cover test distance was noted to

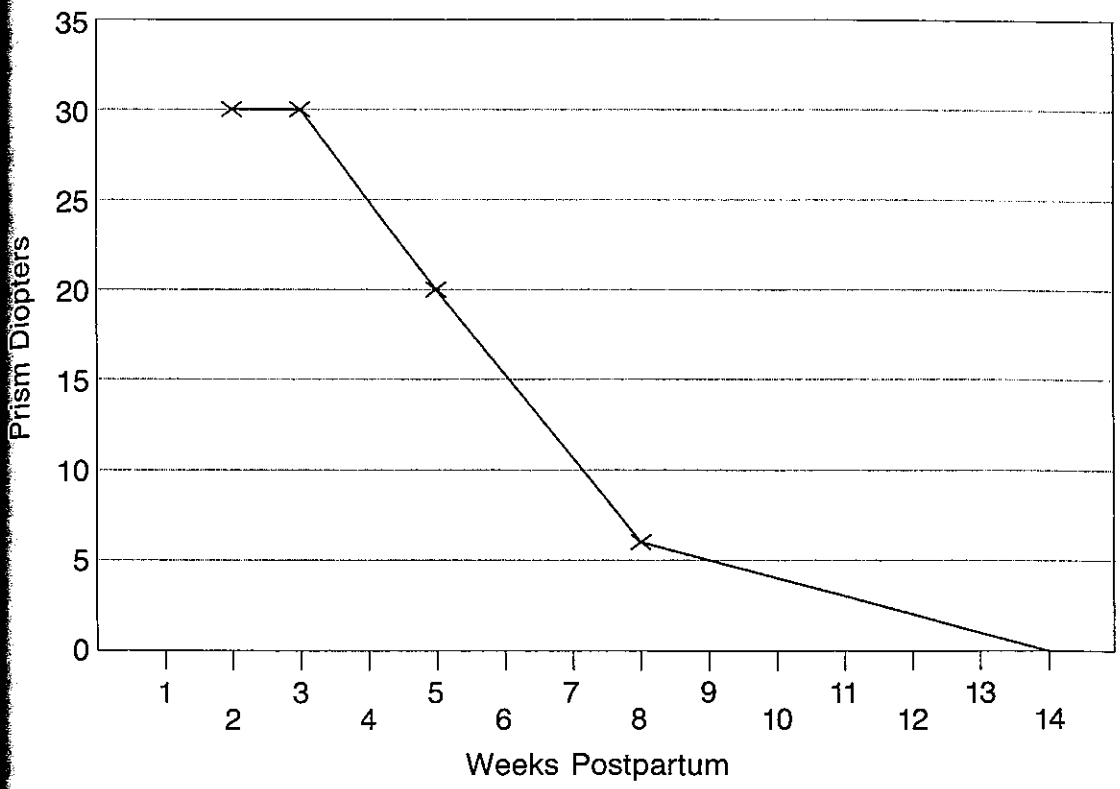

Figure 3: Recovery rate of esotropia.

be orthophoric. Cover test near remained unchanged. Extraocular movements were normal (there was no diplopia evident on left gaze).

MANAGEMENT

Management of this condition is conservative. On the second visit, however, a 25$^{\Delta}$ base out fresnel prism was fitted to a pair of sunglasses to relieve the diplopia at distant fixation. The prism proved of little assistance to the patient due to induced blurred vision by the prism, diplopia on left gaze, and incomitance of the deviation.

Lee and Atkinson[5] have suggested occluding the lateral third of the patient's glasses to avoid diplopia in lateral gaze as well as the wearing of sunglasses to relieve the photophobia.

DISCUSSION

Spinal anaesthetics have given rise to palsies of all the cranial nerves except the olfactory (first), glossopharyngeal (ninth) and vagus (tenth) cranial nerves.[1,2,4,5] It appears that the sixth nerve is the most commonly affected, either unilaterally or bilaterally. Although it is rare, occurring approximately one in every two hundred to three hundred cases, unilaterally more than bilaterally.[1,4,5]

Symptoms which appear to precede this type of sixth nerve palsy are headaches, neck pains, dizziness, nausea and photophobia.[1] Diplopia occurs from the second day to the third week after the anaesthetic, most commonly occurring on or about the seventh day. Full recovery occurs anywhere from the fourth week to the fourth month,[1,2,4,5] but may be delayed for up to twelve months.[1]

Various theories have been postulated as to the cause of the sixth nerve palsy. These are: toxicity theory;[1,4,5] inflammatory theory;[1,4,5] and mechanical theory.[2,4,5]

Toxicity Theory

The toxicity theory suggests that a specific action of the drugs used in anaesthesia may lead to a degeneration of the cells[1] along the exposed course of the sixth nerve which is why it is most commonly affected.[1,4]

The alternative view is that a general cerebral toxaemia may lead to a breakdown in binocular coordination thus causing diplopia.[4,5]

Inflammatory/Infective Theory

The inflammatory theory suggests that the procedure of spinal anaesthesia leads to low grade meningitis[1,5] or aseptic meningitis.[1]

The toxicity and inflammatory theories may be connected because the anaesthetic solution may alter the chemical balance in the cerebral spinal fluid (CSF) and lead to irritation of the meninges.[4]

Mechanical Theory

There appear to be two views regarding the possible mechanical causes that lead to a sixth nerve palsy.

(a) Spinal anaesthesia alters the hydrodynamics of the CSF pressure leading to a displacement of the cerebellum. This causes stretching of the anterior inferior cerebellar artery which is attached to the basilar artery — thus possibly compressing the nerve[5] (Figure 1).

(b) The spinal anaesthesia causes a downward displacement of the brain due to change in the CSF pressure. This displacement may lead to a stretching of the nerve over the petrous temporal bone[2,4] (Figure 2).

The mechanical theories present the most plausible explanations for the cause of the sixth nerve palsy. This is because decompression of the subarachnoid space is likely to be common to all such spinal procedures,[2] as may be a change in the hydrodynamics of the CSF.[5]

CONCLUSION

There was no anatomical evidence in the C.T. scan of this patient to offer any additional evidence to explain why the palsy was unilateral.

In addition, none of the theories adequately explain why the palsy is more likely to occur unilaterally than bilaterally.

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VISUAL THERAPY IN HYSTERICAL BLINDNESS — A CASE STUDY

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Abstract

A general overview of some of the features and methods of investigating and managing patients with presumed functional visual disorders is presented, together with a case study of such a patient. Following numerous investigations, including psychiatric evaluation, the patient was referred for visual therapy in an attempt to improve his left visual acuity. With much encouragement and intensive orthoptic exercises, over a period of four weeks, the recorded vision in the left eye improved from no perception of light to 6/6.

Key words: *Functional visual loss, hysterical blindness, malingering.*

INTRODUCTION

The orthoptist may at times be confronted by a patient claiming poor vision but subjective ocular assessment suggests that visual function would appear much better than the patient volunteers. In particular, there may be no clinical aetiology apparent. In such cases, suspicions may be aroused that visual loss is of a functional nature.

In the literature,[3,5,6] functional visual defects are usually divided into two categories: hysteria and malingering.

Hysteria is a form of neurosis. There is an underlying psychological problem that manifests itself as a physical symptom, in this situation a visual defect. It is a conversion reaction used to escape some intolerable situation. It is a subconscious process and even though there may be some form of gain involved, eg. attention, this is not a conscious thought of the patient.[3,5]

Malingering on the other hand, is the deliberate feigning or exaggeration of illness and is frequently done for the purpose of a consciously desired end.[3,6] One particular example frequently cited in the literature is of those wishing to avoid hazardous duties in the military service.[3,5,7]

However, the dividing line as to where neurosis ends and malingering begins is by no means clear cut.

Kramer et al[3] noted various features of the malingerer. They usually tend to be young, in their 20s to 30s, under some pressure especially associated with their employment and there is frequently a preceding trivial ocular incident. They note however, that patients with ocular hysteria "frequently display 'la belle difference' " and the attention that is sought is often of a more emotional nature. The latter patients may show a wider age spectrum, from school age children[5] to the fourth decade.[3]

Functional visual defects may occur in one or both eyes and may range from moderate to

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complete visual loss. In the case of complete blindness in one eye, in the presence of a normal fundus and normal vision in the fellow eye, there can be only two causes: a unilateral retrobulbar lesion or a functional visual disturbance.[6]

Investigation

In the investigation of such patients, much information may be gained by observation, for example, the patient's mobility and general manner. There are also various tests that may be of specific value. The choice of tests will vary according to the degree of apparent visual loss. There are numerous orthoptic tests that may be used. A positive response on such tests requires reasonably good vision in both eyes, with or without binocularity, and would thus be incompatible with a significant reduction of vision in one or both eyes.

Stereopsis Tests

Positive recognition of stereopsis on tests such as the Titmus and TNO stereo tests, especially for the finer stereoacuity levels, requires reasonably good vision in both eyes (6/12-6/18).[3,5,6]

Bar Reading

Fluent bar reading with both eyes requires binocularity and reasonably good vision in each eye. In the case of true unilateral visual loss, there would be hesitancy and head movement required to read along a line, as the bar, appearing solid, would partially obscure the print.

Lenses

These are placed in front of the good eye to induce fogging, and vision is then assessed with both eyes open. There are various methods that may be used.

Plus lenses may be used for distance testing with lower lens powers (eg +3.00DS), or they may be used for near with high lens powers (eg +10.00DS). In the former case, normal vision would not be possible at distance for the fogged eye. In the latter case, reading with the fogged eye would be possible only at very close range (<10cm). If reading continues normally as the print is gradually moved further away, it must be with the affected eye.

Cylindrical lenses may also be used. These are used in pairs of opposite powers eg + and −4.00DC, placed initially at parallel axes and then one cylinder is rotated slightly by 10-15 degrees.

In each of the above situations normal reading at the standard test distances of six metres and one third of a metre respectively, would therefore have to be with the alleged affected eye.

Another method is to place paired lower power (+/−1.00) spherical or cylindrical lenses in front of the affected eye, and then to test the vision monocularly. No actual refractive change occurs in this situation, but the patient may believe that the lenses are improving the vision.

Prism Testing

Prisms may be used horizontally or vertically. A positive prism vergence response with a 4-8 dioptre prism, placed base out in front of the affected eye, would indicate relatively good central vision in that eye. A 4 dioptre prism placed vertically in front of the one eye should induce diplopia and if both eyes are seeing normally, would cause reading to be hesitant.

Synoptophore Response

Appreciation of the smallest, bifoveal slides require vision of approximately 6/12-6/18. The patient may then be questioned about the details on such slides.

Visual Fields

Fields are typically concentrically constricted with all sized targets and do not vary appropriately when assessed at different distances. For example, the field may show no change at all or decrease in size, instead of becoming larger with increasing distance. Patients with ocular hysteria may also show spiralling of the field.

Other particularly useful objective tests include:

Pupillary Reactions

Normal, symmetrical, direct and consensual pupil reactions, especially when there is an

absence of a relative afferent pupil defect (RAPD), would indicate integrity of the pupillary portion of the visual pathway, together with functional retinae. A normal response to this test is particularly significant in the case of claimed unilateral visual loss.

Also, shining a very bright light into the affected eye will elicit a blink response when there is some visual function present. Such a reaction may also be induced in response to some form of visual threat.

Optokinetic Nystagmus

A positive response on OKN testing indicates that vision must be present. This test may be performed with an OKN drum or tape. However, the malingerer may be able to suppress the response. A better method of testing is to use a fairly large swinging mirror, half a metre to two thirds of a metre in diameter, held at near in front of the affected eye. This is a more fixation-provoking test and thus more difficult to consciously suppress.

Visual Direction

Here the patient is asked to look towards their own finger or hand. A truly blind or hysterically blind person will do this with ease. The malingerer will tend to move their eyes around and look elsewhere.

Visual Evoked Potential (VEP)

This test provides a very helpful method of objectively assessing visual function, particularly when patterned stimuli are used. The pattern VEP is primarily a response of the central visual field, particularly the central four degrees.[4] Thus, the presence of a normal VEP indicates integrity of central retinal/macular function.

The amplitude of the VEP varies with the check size used, being maximal with check sizes of 10-20 seconds of arc[1,2,4] and decreases with larger and smaller check sizes. Thus, assessing the VEP with a variety of check sizes may be helpful to correlate with the patient's volunteered acuity.

However, it is possible for people to voluntarily alter their VEP and so "fool" the test.[2] This may be achieved by fixating off-centre, so that the small sized check pattern is projected onto the peripheral retina where the resolving power is insufficient to produce a normal waveform. It is therefore important to carefully monitor fixation while the test is being performed on this type of patient.

In general, the presence of large amplitude, normal latency responses, especially for small (less than 20 seconds of arc) checks, indicates intact visual pathways. It is important though to remember that even a 10 seconds of arc check is only comparable to 6/60 Snellen vision equivalent, and that the presence of a normal VEP response does not necessarily guarantee normal acuity (i.e. 6/6). However, this test is particularly useful in the case of feigned unilateral visual loss. If the VEP responses are not only normal, but importantly are symmetrical, the visual acuity in the eye in question is probably comparable to the good eye. Responses such as this may be sufficient to end the search for pathology of the visual system.

Discussion

With much time and patience, it may be possible to coax some individuals to a point where the vision chart is read quite normally. At times the malingerer, when faced with the fact that vision must be better than claimed, may "come around" with a noticeable improvement in function.

Having determined that vision is better than the patient will admit, there remains the problem of management. It is important to try and establish what the underlying or triggering mechanism could be. It is usually advisable to have the patient undergo a psychiatric consultation, especially in the case of ocular hysteria where there is usually some underlying emotional problem that may be treated successfully with psychotherapy. The results found at the psychiatric evaluation may well direct the entire course of management.[3]

As orthoptists, we are called upon to investigate these time consuming patients with a view to establishing more definitely that there is indeed a case of functional visual loss. Commonly however, these patients are referred back to the ophthalmologist and possibly on to a

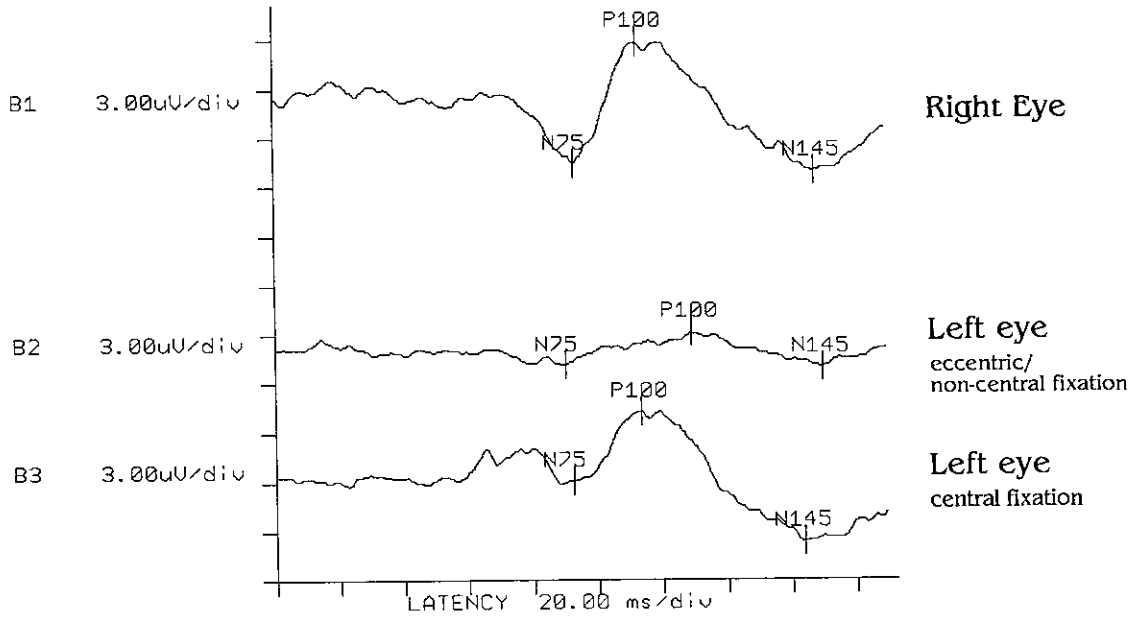

Figure 1: Visual evoked potential

psychiatrist and are lost to follow up. The following case was of interest not only from the investigation point of view, but more importantly, because he was actually referred back to the orthoptists for treatment.

CASE STUDY

An 18 year old Army private, C.G., was referred with a history of loss of vision in his left eye. He reported that during army exercises, following a minor injury to the left eye, he was required to sight his gun and noticed that the vision in his left eye was blurred. Within four days, he stated that he had complete loss of vision. He noted an occasional stinging sensation in the eye but no pain was present on movement of the left eye.

Over the next six weeks, C.G. was seen by two ophthalmologists, a neurologist, two psychiatrists and an orthoptist. Numerous testing procedures were performed, including CT scan and blood tests, with unremarkable results.

Ophthalmological examination revealed an uncorrected vision of 6/4 for distance and N5 for near in the right eye and no perception of light in the left. Slit lamp and fundus examination were normal. Pupillary reactions were normal, both direct and consensual to light and accommodation. There was no evidence of relative afferent pupil defect. OKN responses were normal binocularly and when fixing with the right eye. Testing of the left eye initially revealed a relatively normal OKN response but it then appeared that this was being subjectively

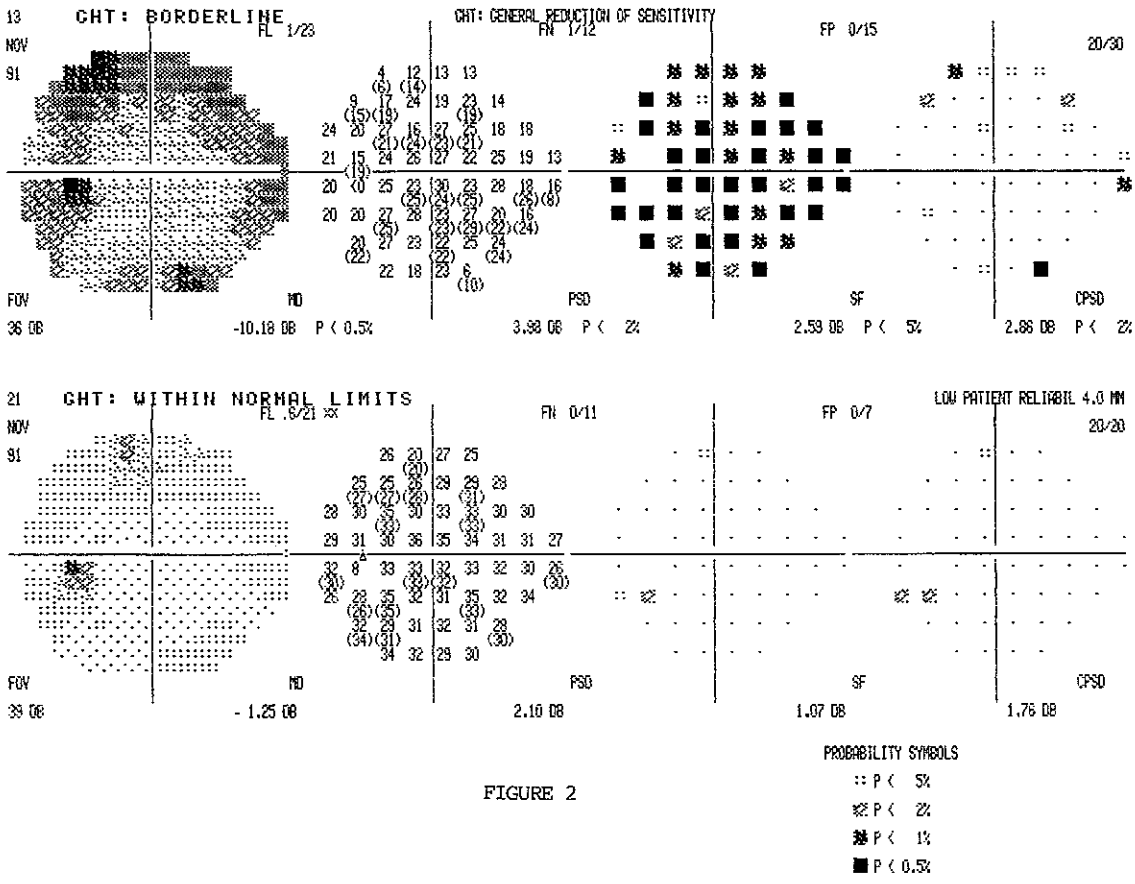

FIGURE 2

Figure 2: Humphrey Field Tests

suppressed. Thus the general ophthalmic examination revealed no ocular aetiology for a complete unilateral loss of vision.

Visual evoked potential (VEP) testing was performed using a variety of check sizes, the smallest of which was equivalent to 6/36 vision (6.5' of arc). The right eye showed normal responses with all checks. The left eye initially showed a barely recognisable VEP waveform, but investigation revealed fixation was off-centre. With encouraged central fixation, normal VEP responses were also elicited with the "blind" left eye with all check sizes, therefore indicating at least 6/36 acuity. It was also apparent that the VEP responses were symmetrical for each eye, therefore indicating that the left eye probably could see as well as the 6/4 right eye. (See Figure 1)

Reports from several of the people investigating C.G. made comment of his dissatisfaction with army life, and the psychiatrist felt his apparent loss of vision may be part of a conversion disorder.

He was subsequently admitted to hospital for further psychiatric evaluation. It was felt that C.G. had "trapped himself into a corner" and that his only escape was to admit that he could indeed see normally. The psychiatrist felt that visual therapy could provide an opportunity for

his vision to improve. He was subsequently referred for orthoptic treatment.

Being an inpatient, he was seen on a very frequent basis (almost daily). Visual therapy commenced with OKN stimulation and progressed through a series of conventional orthoptic exercises. The latter included convergence exercises, red filter work, stereograms and bar reading. During treatment sessions, he was encouraged to tell of the improvement he was noticing. For example, he reported "tingling sensations" with OKN and as the vision progressively improved that things were "looking brighter now", that he could "see figures now" and that he was "starting to see details". Along with this, the orthoptists made many positive suggestions as to how the vision may be being helped, such as "this exercise should help improve your vision", "colour vision should be coming back now", "can you feel that making a difference?"

In view of the fact that the volunteered left vision was NPL at the beginning of treatment, fields were not initially assessed. Humphrey fields were performed midway through treatment and at the end of treatment. As can be seen in Figure 2 apparent improvement occurred, with a normal field response at his last visit.

From apparent blindness in the left eye, gradual improvement in vision was made over a four week period. When last seen the left acuity was 6/6, and there was normal stereopsis and colour vision. He was then transferred from hospital to an Army base with the view to a medical discharge.

CONCLUSION

Within our role as orthoptists, our association with functional visual loss patients usually extends only to the investigation and diagnosis stage. A question frequently raised is to distinguish between hysterical blindness and malingering, however, in this patient's psychiatric evaluation, it was interesting that no definite line was drawn between the two. Rarely do we become involved in the subsequent follow-up and treatment of such cases, especially with such notable results.

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