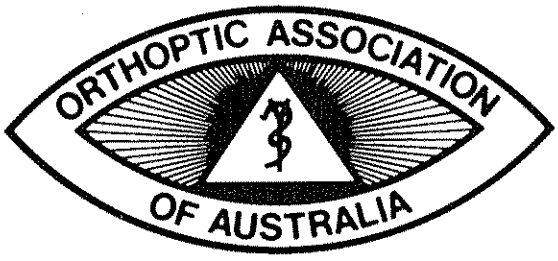
AUSTRALIAN ORTHOPTIC JOURNAL



1995
Volume 31

AUSTRALIAN ORTHOPTIC JOURNAL

Volume 31, 1995
(ISSN 0814-0936)

The Australian Orthoptic Journal is the official journal publication of the Orthoptic Association of Australia Inc. The Australian Orthoptic Journal features full length original research papers, clinical papers, review articles and short communications. Contributions may deal with binocular vision, eye movement disorders, strabismus, ocular motility, vision, or visual and ocular rehabilitation.

Published by the Orthoptic Association of Australia Inc.

Printing and production: Printbound Pty Ltd, 151 Islington Street, Collingwood Vic. Australia 3066

Distribution: Central Secretariat Orthoptic Association of Australia Inc. PO Box 79, Hampton, Victoria 3188.

The Orthoptic Association of Australia wishes to acknowledge the financial assistance of OPSM Pty Ltd with the production of this journal.

Annual subscription: A$45 Australia, A$50 elsewhere

NOTES FOR CONTRIBUTORS

PUBLICATION CRITERIA

It is a condition of acceptance of any article for The Australian Orthoptic Journal that only original material is submitted unless suitable acknowledgement has been made in the references and that such articles have not been previously published nor are under consideration for publication elsewhere. This must be stated in a covering letter. Articles for submission may include original scientific papers, case histories, book reviews or letters. Manuscripts with one high quality copy and three photocopies should be typewritten in double spacing, with wide margins on one side only, on A4 paper. Authors are requested not to underline any part of their work. Place author's names and page numbers in the top right hand corner of each page.

TITLE

Authors are instructed to begin with a **title** page which should be concise. Include the author(s) name(s), qualifications, name of place or institution where work was conducted and an address for communication.

ABSTRACT

The **abstract** should be a succinct representation of the article. The components should relate directly to the format or the body of the article including aim, procedure, results, discussion and conclusion. The abstract should be limited to 150 words and be submitted on a separate sheet. Submit **key words** underneath the abstract. Do not duplicate words in the title and limit these indexing features to five words.

TEXT

Clearly describe the purpose of the study. Include methodological information on procedure and design. Outline statistical methods of analysis and demonstrate results using figures and tables. Avoid duplicating information between text and diagrams. Discuss the relevance and implications of the study and provide a brief conclusion.

ACKNOWLEDGEMENTS

Include professional, methodological, analytical, technical or financial support.

REFERENCES

References should be indicated in the text by superior numbers, in the order that they appear in the text and should correspond with a detailed list at the end of the article. Only references directly referring to the text should be listed. References should follow the format: author(s), title of article, journal name (as abbreviated in Index Medicus), year of publication, volume number and inclusive page numbers. References to books should include author(s), title, editor(s), edition, city of publication, publisher, year of publication and page numbers.

Examples of correct style:

Journals

Young RW. Visual cells and the concept of renewal. Invest Ophthalmol 1976;15 No, 9:700-711.

Books

Cornsweet TN. Visual perception. 2nd ed. New York: Academic Press Inc. 1971:6-26

PHOTOGRAPHS

Should be clear black and white with good contrast.

FIGURES, TABLES AND LEGENDS

Figures and tables should be marked lightly in pencil on the back with an arrow indicating the top, its number (Fig. 1 or Table 1., etc) and the author(s) name. They must be high quality black print on white background.

Legends or captions for illustrations should be typed with arabic numerals corresponding to the illustrations on a separate page. When symbols, arrows, numbers or letters are used to identify parts of the illustrations, identify these clearly in the legend.

CLOSING DATE

Papers for publication in the Australian Orthoptic Journal may be submitted to the Editor at any time. Papers, case histories and other communications for publication should be sent to:

The Editor,
Central Secretariat,
PO Box 79,
Hampton Victoria 3188

These guidelines are in accordance with the Vancouver Agreement[1,2].

1. International Committee of Medical Journal Editors. BMJ 1991; 302:338-341.
2. International Committee of Medical Journal Editors. Ann Int Med 1988; 108:258-265.

AUSTRALIAN ORTHOPTIC JOURNAL

Volume 31, 1995

(ISSN 0814-0936)

CONTENTS

Australian Orthoptic Journal 1995, VOL 31: 1-3

EDITORIAL

SENSORY AND TECHNICAL ASPECTS OF VISION SCREENING

The original articles presented in this edition of the Australian Orthoptic Journal reflect a theme which includes both the sensory and technical aspects of vision screening. Jennings' research demonstrates an integration of an indepth understanding of available perimetric technology and a knowledge of the pathological processes of diabetic disease. These combined facets have produced a clinically orientated research question which has been soundly approached. The Humphrey Field Analyzer Full Field 120 point Threshold Related Screening Test detected visual field deficits in diabetics with and without mild retinopathic signs. The finding that half of the diabetics with no ophthalmoscopic retinopathy had visual field deficits is of major significance for perimetric orthoptists. This original paper supports the recently published diabetic research findings in the Medical Journal of Australia which also demonstrated a statistically significant relationship between the duration of the disease and the level of retinopathy[1]. The author highlights the clinical relevance of these findings and explains the advantages of a perimetric test which is reduced temporally whilst testing a full field. Further research into the differences between patients with mild retinopathy and IDDM versus NIDDM is required. The author emphasizes the potency in screening the preretinopathic status of diabetics by orthoptists.

The basis of the most commonly used optotype for measuring visual acuity is the visual angle subtended through the nodal point and retinal cone size. The assumptions concerning the area of retinal stimulation are challenged in the research article by Duyshart, who was awarded the most recent Emmie Russell Prize. Form vision components range from minimum visible and minimum detectable to resolving power. The author discusses legibility as a factor in optotypes that are constructed using letters and states that this is not constant. Visual acuity testing involves resolution, perception and recognition. Different areas and dimensions of retinal stimulation were compared at reducing levels of visual angle. The author suggests, from the results of this study that the initial visual process is related to the area of the stimulus and that the geometric design occurs secondarily. This two stage process lends itself ideally to further investigative research into the facets involved in children's shape recognition and the visual acuity levels inferred.

Williams and co-workers have published a comparative study of single and linear optotypes, thus continuing the theme of aspects of vision testing and screening. An important clinical addition to the optotype

armory has been made with single letters surrounded by four crowding bars[2]. The advantages of this design are its conceptual simplicity and its approximation to linear visual acuity levels. These authors studied the visual acuity responses of ampblyopes and normals to singles, linear and crowded singles test types. Significantly, the authors confirmed previous findings of differences between singles and crowded singles and linear tests. These differences were apparent in the amblyopic group. No differences in test results were found for the normal group. This equates well as discrimination ability is reduced in amblyopes and forms part of the diagnosis criteria of this condition. The authors rightly state that it is yet to be determined if crowded singles will be appropriate as a vision screening tool.

The theme of vision screening is taken up in the article by Deveraux and colleagues and involves a preventive health project in Nepal. The limitations of the testing protocols highlight the difficulties in maintaining an adequate and constant testing environment in an atmosphere of reduced resources. Priorities become evidently linked to nutrition and basic health requirements. A common reversible ocular problem linked to vitamin deficiency is described in xerophthalmia. Vision and ocular motility referral rates were higher than those recorded for developed countries. The authors suggest that the reasons for the 24% rate may include a lack of public health programs and the relative low socio-economic strata from which the screened orphans had come. Although this was a screening study, immediate remedies for xerophthalmia were instituted by making available adequate doses of Vitamin A. Statistical information from this screening program facilitated the possible institution of public eye health programs by documentation of type and incidence of ocular defects. This is a demonstration of international orthoptic co-operation, public health service and clinical education.

A further aspect of screening is the role that it plays in exploring the aetiological factors in particular disorders. This aspect has begun to be investigated in the original article by Ferguson and co-workers. These authors looked at the efficacy of both the stereoacuity and the fusional vergences of normal siblings of subjects with early onset strabismus. Comparisons were made with normal siblings of subjects with no strabismus. Interestingly, reduced stereopsis was recorded in the sibling group with strabismus family history, but fusional vergences were normal. Given our understanding of the role of vergences in maintaining gross ocular synergism even in the presence of strabismus with central suppression or abnormal retinal correspondence, these findings are consistent with binocular vision premises[3]. Subtle vergence defects may occur with conversion of the initial vergence response into tonic vergence levels. This could be investigated through prism adaptation responses. Further research to pursue this avenue of investigation would be to evaluate the precise levels of reduced stereoacuity with purely random dot stimuli as opposed to the polarized contour stimuli of the Titmus Test. This research provides a valid contribution to the current theories of strabismus aetiology.

A rare acquired eye movement disorder is described in the case presentation by Georgievski and colleagues. This patient underwent interstate evaluations and the clinical picture is fascinating. The description is one of saccadic deficits in downgaze,

post head injury. There are associated pursuit anomalies, which are mild, Parinaud pupil signs, and vergence deficiencies which are symptomatic. The anomalous head posture consists of atypical jerky chin down movements. The lesion site is localized to a small hematoma in the upper midbrain, slightly lateral to the midline, and is demonstrated on radiological imaging. Discussion as to the aetiology of this selective deficit leads the authors to review the neurological pathways for vertical gaze. Controversy still exists concerning the differentiation of up and downgaze, the medial and lateral routes of MLF fibres and discrete or common blood supply to the medial and lateral aspects of this upper midbrain area. This article contributes well to the literature on eye movement disorders and shows how clinical features provide evidence for further investigations. This case highlights the important role that the publication of case reports provides. Individual patient descriptions with specific lesions or disorders contribute along with experimental animal studies to our understanding of ocular pathology. This information can be diagnostic, localizing or have an influence in the management strategies for both common and obscure eye movement disorders.

REFERENCES

1. Couper, JJ et al. The Diabietes Control and Complications Trial. Implications for children and adolescents. Med Jnl Aust 1995; 162(7): 369-372.
2. Parr, JC. Clinical assessment of visual acuity. Trans OSNZ 1981; 33: 157-167.
3. Owens, D & Leibowitz, H. Perceptual and motor consequences of tonic vergence. In C. Schor & K. Ciuffreda (Eds), Vergence eye movements: basic and clinical aspect. Masachusetts: Butterworth's. 1983; 25-74,

Australian Orthoptic Journal 1995, VOL 31: 5-11

ORIGINAL ARTICLE

VISUAL FIELD SCREENING IN DIABETES

BRONWYN JENNINGS BAppSc(Orth) DOBA

Abstract

Regular retinal screening of all diabetic patients is recommended because early detection and treatment of diabetic retinopathy can prevent or reduce visual loss. A screening test which detects changes in retinal sensitivity before retinopathy is detected ophthalmoscopically would be a valuable tool in drawing attention to those patients at risk of developing retinopathy. The aim of this study was to determine if visual field changes in forty six (46) diabetics with little or no diabetic retinopathy could be detected using a Humphrey Field Analyzer full field 120 point (threshold related) screening test. The results show that this test can detect visual field defects both in diabetic subjects with no retinopathy and those with mild retinopathy. Since it is comparatively quick, reasonably sensitive and able to test out to 60 degrees it may be a useful test in preliminary screening for diabetic eye disease.
Key Words: *retinal screening, diabetic retinopathy, Humphrey Field Analyser full field 120 point screening test.*

INTRODUCTION

Diabetes Mellitus (DM) is a metabolic disease characterised by hyperglycaemia (high blood glucose levels) due to a deficiency of insulin. There are two types of Diabetes Mellitus:

Insulin-dependent diabetes mellitus (IDDM or Type 1) involves a true deficiency of insulin. This is due to atrophy of tissues in the pancreas which normally contain insulin producing cells. IDDM occurs predominantly in young people. Symptoms include excessive urine production, excessive thirst and marked weight loss[1].

Non-insulin-dependent diabetes mellitus (NIDDM or Type 2) involves a resistance to the action of insulin rather than a true deficiency, and occurs in middle aged or elderly people who are frequently overweight. The symptoms are often those of the late complications of diabetes[1] including retinal damage, declining renal function, interference with circulation to the legs and peripheral nerve damage.

Diabetic retinopathy (DR) is one of the major complications of diabetes mellitus. It is one of the most important causes of adult blindness, and is said to be the most common single cause of blindness in the under 65 age group[2]. It is, however, a poten-

Address for correspondence: Bronwyn Jennings, 81 Shannon Circuit Kaleen, ACT 2617. Submitted: November 1994. Accepted for publication: April 1995.

tially treatable complication of diabetes mellitus. Loss of retinal pericytes is considered the primary event in DR, while the role of thickening of the endothelial basement membrane is unclear. These combined lead to capillary closure and abnormal vascular permeability, which underline all the ophthalmoscopic lesions of diabetic retinopathy[2].

The longer the duration of the diabetes, the greater the risk of retinopathy. The relationship of metabolic control of the diabetes to the frequency and severity of the retinopathy is not settled but it is generally assumed that good control delays the onset of retinopathy[3].

Regular retinal screening of all diabetic patients has been recommended because early detection and treatment of DR can prevent or reduce visual loss[2].

A screening test which detects changes in retinal sensitivity before retinopathy is detected ophthalmoscopically would be a valuable tool in drawing attention to those patients at risk of developing retinopathy. The importance of screening for diabetic eye disease is outlined by the Retinopathy Subcommittee of Australian Diabetes Society[2] who states that:

> "Other health professionals should also be encouraged to promote screening and carry out preliminary examinations for diabetic eye disease."

There have been several studies which have examined the visual fields of diabetic subjects in order to determine the relationship between field loss and retinopathy[4-9]. Using different types of field test they have found that visual field defects occur in patients with DR, and may also occur in patients with little or no clinically detectable retinopathy[4,8]. However, the field tests used in these studies were either too time consuming for general diabetic retinal screening, not sensitive enough or unable to test out to 60 degrees.

Two studies which have examined the visual fields of diabetics both with and without detectable retinopathy are those of Roth[4] and more recently Trick[8]. Roth[4] tested subjects with no retinopathy as well as subjects with retinopathy using a *central 20 degrees field scotometer*, and found that all patients with ophthalmoscopically visible retinopathy had scotomata as did nearly half those without visible retinopathy. It was postulated from this study that scotomata may represent a form of pre-retinopathy and may be related to defects in the retinal capillary circulation. However, since this visual field test only examines the central 20 degrees, subjects with subclinical microcirculatory changes in the mid periphery would be missed.

Trick et al[8] tested subjects using the *Humphrey* 30-2 automated perimetry, classifying them as either having little to no DR or mild background DR. They further divided them into categories based on whether they had IDDM or NIDDM. Their aim was to determine whether sensitivity in the visual field was reduced in diabetics with little or no DR, as well as to determine if there was an association between visual field loss and insulin dependency of the diabetic subject. They found that visual field defects did sometimes occur in diabetic subjects without detectable retinopathy. They also found that there was a high percentage of field defects in NIDDM subjects with mild background DR. The field test chosen for this study was able to quantify visual field sensitivity but it didn't test beyond 30 degrees and therefore as with the Roth[4] study may have missed some defects in the mid periphery. It was also a time consuming

test and therefore not useful as a routine screening test for all diabetics.

A visual field test which is rapid, sensitive and able to test out to 60 degrees would seem to be the best field test for routinely examining the retinal sensitivity of diabetic patients with little or no clinically detectable DR. If field defects are found in the absence of detectable DR (possibly indicating a state of preretinopathy), these patients could be monitored more closely.

The aim of the present study was to determine if visual field changes in diabetics with little or no DR could be detected using a *Humphrey Field Analyzer* full field threshold related screening test. Any practical screening test must inherently be a compromise between speed, ease of use and sensitivity. This test (taking approximately 5-7 minutes per eye) was comparatively quick, reasonably sensitive and examines 60 degrees of field.

METHOD

Patient Selection

Forty six diabetic subjects (29 males and 17 females) from a private ophthalmic practice were studied over a 5 month period. Permission from subjects was sought by informed consent. The subjects ranged in age from 17 to 80 years, the mean age being 50.6 years.

Each subject was asked to give details of their diabetes type (IDDM or NIDDM) and the duration of the condition. There were 24 IDDM subjects and 22 NIDDM subjects, duration of the condition ranged from 1 month to 35 years, the mean duration being 9.7 years. The level of control as indicated by present average blood glucose levels (BGL's) was also noted, however the subjective nature of the responses were not able to be verified by objective methods, such as blood testing on the day of the assessment of confirmation by the referring practitioner. Control was considered as being good if BGL's averaged 4-8, fair if they averaged 8-12, poor if they averaged more than 12 and unknown if the subject had no knowledge of their BGLs. Twenty one (45.6%) had good control, 16 (34.8%) had fair control, 6 (13.1%) had poor control and 3 (6.5%) had unknown control.

Prior to visual field assessment, each subject's visual acuity (VA) was assessed monocularly at 6 metres using a Snellens chart, and intraocular pressures (IOPs) on those over 40 years of age were assessed using Goldman applanation tonometry.

Criteria for inclusion in the study was a corrected monocular VA of 6/9 or better in at least one eye, IOP < 21mmHg (if over 40 years of age), no history or ocular signs of any disease likely to cause visual field defects, and no previous laser photocoagulation. Thirty six subjects (78.3%) had a visual acuity of 6/5, 8 subjects (17.4%) had acuity of 6/6, and 2 subjects (4.4%) had acuity of 6/9.

The subjects who met the inclusion criteria outlined above, underwent a visual field assessment using the Humphrey Field Analyzer's Full Field 120 point screening test (threshold related).

Humphrey Field Analyzer

The Full Field 120 point screening test pattern was chosen from the Humphrey Field Analyzer's range of screening tests because it tests out to 60 degrees and takes approximately 5-7 minutes to do. The number and location of points tested gives a good compromise between the Full Field 81 point pattern and the Full Field 246 point pattern.

The type of screening strategy chosen was

the threshold related strategy. With this strategy, if the subject sees a point the first or second time it is tested, the area is recorded as normal. When the subject doesn't see the stimulus the point is tested again to make sure the miss wasn't a mistake. If the point is missed a second time, the Analyzer registers a miss and moves on to test other points. Screening is done at an intensity 6 dB brighter than the expected threshold and therefore missed points are known to be at least 6 dB deep.

Testing Procedure

Only one eye of each subject was tested in order to eliminate the possibility of fatigue and possible enhancing effects of the learning curve on cooperation levels. The eye with the best corrected monocular VA was chosen. If the corrected monocular VA for each eye was equal then the right eye was chosen. The near lens correction to be used for the central field testing was calculated automatically by the Analyzer using the subject's distance correction.

Standard parameters were used for each test, namely a size III stimulus white target, a central fixation target and blind spot check size III.

The visual field test procedure was explained to each subject before commencing the test. A black patch was placed on the eye not being tested and then each subject was correctly and comfortably set up at the machine. Prior to the commencement of the Full Field 120 point screening test, a demonstration test (lasting up to 60 seconds) was given to subjects to ensure correct understanding of the test procedure.

After the visual field assessment, each subject's pupils were dilated with mydriacyl 0.5% and they underwent a fundus examination by one of two ophthalmologists for retinopathy assessment. Retinopathy was classified as nil if there was no detectable background diabetic retinopathy at all, mild if there was background diabetic retinopathy and moderate if there were some areas of ischaemia and haemorrhages not requiring laser photocoagulation. Thirty subjects (65.2% had nil retinopathy, 14 (30.4%) had mild retinopathy, and 2 (4.4%) had moderate retinopathy. Table 1 shows the frequencies of diabetes type in relation to retinopathy type.

TABLE 1
Frequencies of diabetes type in relation to retinopathy type.

	IDDM	NIDDM	Totals
Nil	9	21	30
Mild	13	1	14
Moderate	2	0	2
Totals	24	22	46

Statistical Analysis

The mean, maximum and minimum scores, range and standard deviation were calculated for the number of missed points on the field test and for the central and peripheral reference levels used for the field testing of each individual subject. Observed frequency tables were compiled to examine the incidence of field loss in relation to retinopathy type and diabetes type.

Unpaired t-tests were performed to compare diabetes type with age, disease duration, missed points on the field test, and central and peripheral reference levels. One factor analysis of variance (ANOVA) was used to determine the differences between retinopathy and age, disease duration, missed points on field testing, central reference level and peripheral reference level.

Significance levels for all statistical tests

was $p = 0.05$. Subjects with moderate retinopathy were excluded from some of the statistical analyses because of the small sample size involved (n=2). After consultation with colleagues it was decided that 5 or more missed points would be considered clinically significant. Given that the Field Analyzer always rechecks missed points to make sure the miss wasn't a mistake, it was decided that allowing more than 4 missed points to be ignored could result in retinal pathology being missed. Although the Analyzer does check for patient reliability during testing it was decided that up to 4 points missed could be due to patient fatigue or misunderstanding and therefore should not be considered as significant.

RESULTS

Table 2 shows the means, maximum and minimum scores, range and standard deviation for the points missed on field testing, and for the central and peripheral reference levels.

TABLE 2
Means, maximum and minimum scores, range and standard deviation for the points missed on field testing.

Summary statistics	Missed points	Central reference point (in dB)	Periperal reference point (in dB)
Mean	9.7	36.7	33.7
Minimum score	0	32	26
Maximum score	59	40	42
Range	59	8	16
Standard deviation	11.1	2	4.4

Table 3 shows the frequency of retinopathy type in relation to whether fields were normal or abnormal.

TABLE 3
Frequency of retinopathy type in relation to whether fields were normal or abnormal.

	Nil retinopathy	Mild retinopathy	Totals
Normal	13 (43.3%)	5 (35.7%)	18 (40.9%)
Abnormal	17 (56.7%)	9 (64.3%)	26 (59.1%)
Totals	30	14	44

Table 4 shows the frequency of diabetes type in relation to whether fields were normal or abnormal.

TABLE 4
Frequencies of diabetes type in relation to whether fields were normal or abnormal.

	IDDM	NIDDM	Totals
Normal	9 (40.9%)	9 (40.9%)	18 (40.9%)
Abnormal	13 (59.1%)	13 (59.1%)	26 (59.1%)
Totals	22	22	44

Tables 5 and 6 show the breakdown of IDDM and NIDDM subjects into subgroups based retinopathy type and field type.

TABLE 5
IDDM
Breakdown of subjects into subgroups based on retinopathy type and field type

Retinopathy	Fields	Number	Percentage
Nil	Normal	4	18.2
Mild	Normal	5	22.7
Nil	Abnormal	5	22.7
Mild	Abnormal	8	36.4

Unpaired t-tests showed that there was *a significant difference* between diabetes type and age ($t = -3.53$, $df = 44$, $p = 0.001$)

and duration of the condition (t = 6.47, df = 44, p = 0.0001). However there was *no significant difference* between diabetes type and points missed on field testing (t = -0.8, df = 44, p = 0.4276), central reference level (t = 1.36, df = 44, p = 0.1793) and peripheral reference level (t = 1.3, df = 44, p = 0.2004).

TABLE 6
NIDDM
Breakdown of subjects into subgroups based on retinopathy type and field type

Retinopathy	Fields	Number	Percentage
Nil	Normal	9	40.9
Mild	Normal	0	0
Nil	Abnormal	12	54.5
Mild	Abnormal	1	4.5

One factor ANOVA tests showed that there was *a significant difference* between retinopathy and diabetes duration (F = 51.75, p = 0.0001). However there was *no significant difference* between retinopathy and age (F = 0.03, p = 0.8611), points missed on field testing (F = 0.52, p = 0.476), central reference level (F = 0.05, p = 0.8163), and peripheral reference level (F = 0.01, p = 0.9343).

DISCUSSION

This study demonstrated that visual field defects in diabetics can be detected using the Humphrey Field Analyzer's Full Field 120 point screening test (threshold related). More importantly, this study found that there was no statistical difference between retinopathy type and the number of points missed on field testing. This indicates that the screening test can detect field defects in both subjects with no retinopathy and subjects with mild retinopathy.

Roth[4] and Trick et al[8] have shown that field defects can occur in subjects without detectable retinopathy. The Roth study[4] found that 48.5% of the subjects without retinopathy had scotomata. Trick et al[8] found that 26.3% of their total diabetic subjects had fields which were "probably abnormal". Of those without detectable retinopathy, 17.7% of the NIDDM group and 14.3% of the IDDM group had field defects.

The present study revealed that of those with no detectable retinopathy, 56.7% had abnormal fields (using 5 or more missed points as the criteria for classifying fields as abnormal). These results are more consistent with those of Roth[4] than those of Trick et al[8]. The classification of fields as being normal or abnormal was determined subjectively by clinicians in this study, whereas in the study by Trick et al[8] field classification was determined statistically by the STATPAC analytical program contained in the Humphrey Field Analyzer threshold strategies (not contained in the Humphrey screening programs).

In this study there was a statistically significant difference between diabetes type and age, and between diabetes type and duration of the condition. The average age of diabetic subjects was less for IDDM subjects than NIDDM subjects, and diabetics with a longer duration of the condition were more likely to have IDDM than NIDDM. These findings are consistent with the literature, which indicates that IDDM occurs predominantly in young people while NIDDM occurs predominantly in older people[1].

There was also a statistically significant difference between retinopathy type and duration of the condition. This is consistent with the findings of the Retinopathy Subcommittee of Australian Diabetes Soci-

ety[2], which has found that the longer the duration of diabetes, the greater the risk of retinopathy.

Trick et al[8] found that visual field defects occurred most frequently in NIDDM subjects with mild background retinopathy (72.3%). The present study found that there was no statistically significant difference between diabetes type and points missed on field testing. However the sample size for NIDDM subjects with mild retinopathy in this study was so small (n=1) that no comparisons can be made for this subgroup. For NIDDM subjects with no retinopathy (n=21), 57.1% had abnormal fields, compared with 55.6% of IDDM subjects with no retinopathy (n=9).

Diabetic subjects without retinopathy who demonstrate field defects may in fact be exhibiting a type of preretinopathy, indicating alteration in retinal function[4]. If this is the case then follow up studies of subjects without retinopathy (both with and without field defects), could be carried out over a specified time period in order to determine whether in fact those with field defects develop retinopathy more frequently than those without field defects. It could also be determined whether there is an increase in field defects prior to the onset of detectable retinopathy, as suggested by Roth[4] in his discussion of a possible follow up study.

CONCLUSION

The present study has shown that the Humphrey Field Analyzer Full Field 120 point screening test (threshold related) is able to detect changes in the retinal sensitivity of both IDDM and NIDDM subjects. This test is comparatively quick, reasonably sensitive and able to test 60 degrees, and therefore may be useful in preliminary screening for diabetic eye disease. Since orthoptists have expertise in field testing procedures, they would be in a position to undertake retinal screening of diabetics, and therefore to open up a new avenue of orthoptic care in the eye health team.

ACKNOWLEDGEMENTS

This project was conducted under the supervision of the School of Orthoptics, Faculty of Health Sciences, La Trobe University. The author wishes to thank Alison Pitt and Ian Story for their help and support.

REFERENCES

1. Trounce J. Clinical pharmacology for nurses 13th ed, Edinburgh, Churchill Livingstone, 1990.
2. Retinopathy Subcommittee of Australian Diabetes Society, Diabetic Retinopathy in Australia 1988.
3. Pavan-Langston D. (ed.) Manual of ocular diagnosis and therapy Boston/Toronto, Little, Brown and Co. 1985.
4. Roth J. Central visual fields in diabetes, Br J Opthalmol 1969: 53, 16-25.
5. Wisznia K., Lieberman T. & Leopold I. Visual fields in diabetic retinopathy, Br J Ophthalmol 1971: 55, 183-188.
6. Federman J. & Lloyd J. Automated static perimetry to evaluate diabetic retinopathy, Trans Am Ophthalmol Soc 1984: 82, 358-368.
7. Bell J. & Feldon S. Retinal microangiopathy. Correlation of OCTOPUS perimetry with fluorescein angiography, Arch Ophthalmol 1984: 102, 1294-1298.
8. Trick G., Trick L. & Kilo C. Visual field defects in patients with insulin-dependent and non insulin-dependent diabetes, Ophthalmol 1990: 97, 475-482.
9. Wolfe K. & Sadun A. Threshold Amsler grid testing in diabetic retinopathy, Graefe's Arch of Clin Exp Ophthalmol 1991: 229, 219-223.

Australian Orthoptic Journal 1995, VOL 31: 13-19

ORIGINAL ARTICLE

VISUAL ACUITY: AREA OF RETINAL STIMULATION

REBECCA DUYSHART BOrth

(Awarded the Emmie Russell Prize for 1994)

Abstract

This paper presents a new concept in design specifications of optotypes used and questions whether the difference in area between optotypes at the same level of threshold acuity influences legibility. Forty subjects were examined with two series of test cards - Equal Area (EA) and Equal Dimensions (ED) - that consisted of single, solid, geometric shapes. The results suggest that on first examination the area of an optotype is significant in the perception process of identifying the optotype perceived.

Keywords: *design specifications, legibility, optotype, resolution, vision.*

INTRODUCTION

The evolution of optotype design used to assess visual acuity has in principle remained unchanged since 1862 when the Dutch ophthalmologist Snellen devised an eye test chart and Donders in 1866 advocated its use[1,2,3]. Letters were used as optotypes and its specifications were based on the mathematical principle that optical infinity occurs at 6m and the minimum angle of resolution is at one minute of arc[4]. Every Snellen letter subtends five minutes of arc and the line width corresponds to 1 minute of arc for each visual acuity level[2,4-11] (Figure 1).

The two fundamental shortcomings of Snellen Letter optotypes are that they do not provide equal legibility and that they represent a measurement of form perception about which very little is known[4,11,12]. These design anomalies emphasise that the assessment of vision with Snellen optotypes incorporates not only the processes of resolution but perception an recognition[13-16].

Ffooks[13] suggests that visual acuity tests depend on the quantity of information presented per unit area. All optotypes are designed within two dimensions; length times width within the five by five minutes of arc grid. Yet, optotype calculations are founded on a linear measurement, that is the diameter of a retinal cell at the fovea. Thus, despite all current optotypes complying with the Snellen principle, each

Address for correspondence: R. Duyshart, School of Orthoptics, Faculty of Health Sciences, La Trobe University, Bundoora, Victoria, 3083. Submitted: November 1994 Accepted for publication: April 1995. This paper was presented in part at the 51st Annual Scientific Conference, Orthoptic Association of Australia, NSW 1994.

provide a different area of retinal stimulation than that of another.

Vitz and Todd[17] suggested that perception of geometric shapes is dependent on progressive analytical levels of line, angle and area; that is, the longer the line, the wider the angle and the larger the area, the greater the chance of a geometric shape being perceived. Could the difference in area between optotypes per level of visual acuity possibly be one of the factors that contribute to the differences associated with legibility?

Apparatus

Two series of **test cards** were produced; Equal Area (EA) and Equal Dimension (ED). In both series the solid single optotypes which the subjects had to discriminate were the circle, triangle, square and diamond (a square rotated forty five degrees). The diamond was included as an internal control within the two series; being of the same dimension and area as the square, per level of visual acuity. The basis of calculation at each level of visual acuity in both series, was on the circle - which subtended five

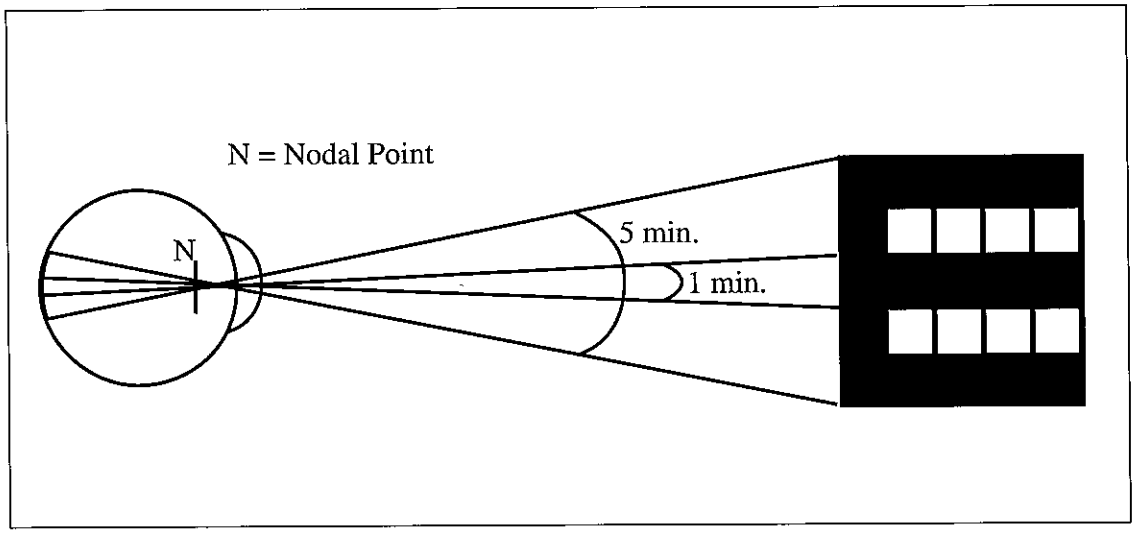


Figure 1. Angles subtended by Snellen optotype in minutes of arc.

METHOD

Subjects

Forty subjects, 13 males and 27 females, aged between four and six years of age were recruited from Theodore Preschool in Canberra. Selection was dependent on parental consent, the child's willingness to participate, the ability to concentrate throughout the tests and availability for retesting. Subjects were randomly allocated into groups A and B by order of presentation irrespective of age and gender.

minutes of are. Calculations were based on the circle in order to comply with the retinal organisation of the receptive fields, which are essentially circular in shape[13]. Thus, the circle in either series was of the same dimension and area at each level of visual acuity. In the ED series, at each level of Visual Acuity (VA), the geometric shapes have the same dimensions as the circle. That is for example, at the theoretical distance of 6000mm, the circle has a diameter of 8.73mm, the square and diamond

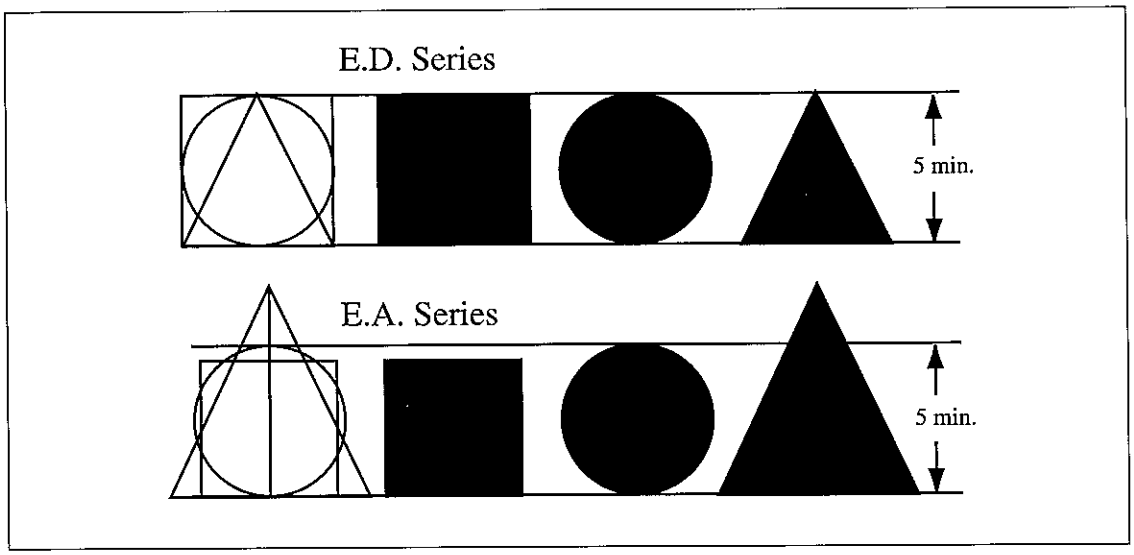


Figure 2. Comparative size difference of shapes in each series of test cards

have 8.73 x 8.73mm dimensions and the triangle's base and height is also equal to 8.73mm. The geometric shapes in the EA series, at each level of visual acuity, covered the same area as the circle. From the area of the circle, per level of visual acuity, the dimensions of each shape were calculated. For example, at the theoretical distance of 6000mm, the circle had a diameter of 8.73mm and it's area equal to 59.82mm squared. The dimensions of the square and diamond, were then calculated to be 7.74 x 7.74mm and, the triangle's base and height measuring 11.75mm (Figure 2).

The EA and ED series of test cards had the geometric shapes displayed as single black optotypes that were laser printed (nine hundred dots per square inch), centrally on a white sheet of A4 paper; width and length 21 and 29 centimetres respectively. The sequence of shapes at each level of visual acuity varied from the preceding and proceeding sequence and where possible, avoided the diamond/square and square/diamond succession. The sequence of optotypes was identical in each series of test cards.

The answer card consisted of a laminated sheet of white A4 paper, which displayed a laser printed black circle, triangle, square and diamond. Cut outs of the geometric shapes were of the same dimensions as those printed on the answer card and were constructed from green cardboard. These separate cut outs were termed teaching shapes (Figure 3).

Procedure

An initial pre-examination training session was performed with the examiner sitting beside the subject for a maximum of five minutes. Each shape was isolated by a single green teaching shape, placed over the same black shape on the answer card, while a verbal description of that shape - from a point form dissertation - was given.

After each shape was identified and described the subject's comprehension of the shapes was tested. The examiner sat beside the subject while a single optotype was displayed from either the EA or ED

series. The subject was asked to indicate, by pointing to a shape on the answer card, which was (thought to be) the same as the shape that had just been presented. The procedure was repeated until all four geometric shapes were correctly identified. The training session was repeated if any of the shapes were mismatched. If the subject failed the second comprehension test, that subject was not included into the sample.

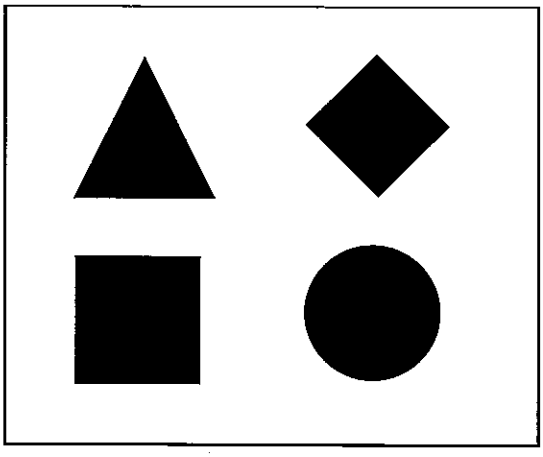


Figure 3. Answer Card

Group A subjects were examined initially with the EA series first, followed by the ED series. Group A subjects were then re-examined two days later with the ED series first, followed by the EA series of single optotypes. Group B subjects were first tested with the ED series, followed by the EA series. Group B were re-examined two weeks later with the EA optotypes first, followed by ED optotypes. The allocation of subjects into Groups A and B was to control for the possibility of a learning effect, which may have influenced the re-examination results. It was hypothesised that Group A would have a greater chance of remembering how the test was performed and thus, fewer errors than Group B. The right and left eyes were examined alternately with each series.

The test was conducted at six meters. This distance was initially measured and marked on the floor. If the subject was unable to identify the majority of shapes within the first three visual acuity levels with either eye at the six meter distance then that subject was not included in the sample.

The single geometric shapes were all displayed at each level of visual acuity; the display started at 6/60, progressing towards 6/5. The criterion for establishing the visual acuity level per shape was that the subject must be able to see the shape at the previous level to that level where the shape could not be seen. Thus, when an incorrect response was given for a shape, that shape was not displayed again. The criteria for an incomplete test was determined by the child's poor concentration/co-operation, illhealth, and/or an indication by the child of not wanting to proceed with the examination.

Re-testing occurred at the same time of day. All tests were conducted in the same room in order to keep the examination environment constant. The examination room was separate from the main pre-school room, all external lighting extinguished. The artificial light was recorded 1.2 meters off the ground (where the vision test was held by the examiner) to be that of 350 Lux. The light intensity was measured by a cosin and colour corrected Topcon IM-2D light meter (serial no. 81572374). It was internally calibrated and had a Lux range from 0.9 to 19,000 units.

RESULTS

The following data was evaluated in terms of percentage error; calculated by counting the number of eyes which did not have

the same visual acuity level in the alternate series, divided by the total number of eyes in the sample. The results were evaluated with Groups A and B combined as no learning effect was indicated.

TABLE 1
Percentage errors for the EA series

	Optotype			
	Circle	Triangle	Square	Diamond
1st	17.5	17.5	35.0	32.5
2nd	21.3	6.3	18.8	17.5
Total	38.8	23.8	53.8	50.0

In either series the circle had approximately the same percentage error (Tables 1 & 2). For all geometric optotypes apart from the circle, the percentage error on second examination decreased (Tables 1 & 2). The triangle had the least percentage error on second examination and the greatest difference in percentage error between first and second examination (Tables 1 & 2).

TABLE 2
Percentage errors for the ED series

	Optotype			
	Circle	Triangle	Square	Diamond
1st	18.8	26.3	27.5	22.5
2nd	21.2	8.8	13.7	13.8
Total	39.4	35.1	41.3	36.6

In the EA series, on first examination the triangle has the same percentage error as that of the circle which was also the lowest (Table 1). However, in the ED series on first examination the triangle's percentage error differed marginally from that of the square, which had the highest percentage error in the ED series (Table 2).

DISCUSSION

The circle was of the same area and dimension in each series and thus, it would be expected that the percentage error would be the same in either series. However, it is interesting to note that the circle was the only geometric shape which had an increase in percentage error on second examination in either series. On first examination, the percentage error for the EA square and diamond was approximately twice that of the EA circle and, less than the circle on second examination in either series. The EA square and diamond have dimensions that are less than that of the circle and triangle in the EA series and thus appear smaller in size (Figure 2). However, the small difference in size would not account for the elevated percentage errors. It may be postulated that the high percentage errors associated with the EA square and diamond were due to the difficulty in determining whether the shape seen was either a square or a diamond - possibly associated with the anomaly of spatial orientation, which is inherent within the pre-school population[18]. Furthermore, once the subject had learnt to differentiate between a square and a diamond there may have been confusion between these shapes and that of the circle, which would account for the circle's increase in percentage error on second examination in either series. Eskridge[2] suggests that letters which have a similar appearance are often more complex to distinguish than those with distinctive features. Area may be a key component to Eskridge's[2] hypothesis and the reason why the LH test is a successful pictorial visual acuity test

as each contoured optotype has a similar overall shape and thus, legibility[19].

The greatest difference in area of the same shape in each series and in comparison with other geometric shapes was the triangle. The difference in area between the EA and ED triangle is approximately fifty percent - the ED triangle being the smaller of the two (Figure2). The percentage error was greater for the ED than the EA triangle (Table 1 & 2) and thus, it may be inferred that because the ED triangle was the smaller of the two it was more difficult to identify. To suggest a relationship between decreased optotype area and increased difficulty in optotype identification would imply that the ED triangle should have had the greatest percentage error than any other optotype. However on first examination in the ED series the triangle was almost as difficult to identify as the square. Yet, on second examination, the ED triangle had a significantly lower percentage error associated with the EA triangle on second examination. It therefore may be inferred that area was initially an influential factor in shape perception and once the distinctive features of the triangle were identified, the triangle was then recognised.

Visual acuity incorporates the processes of resolution, recognition and perception[13-16]. The traditional interpretation of visual acuity measurements has been on the basis of resolution as only the line width of the optotype is considered to be of clinical importance due to its correlation with the functional integrity of the retina. while perception and recognition are thought to be of psychological origin their role in visual assessment has not been fully explored and in particular correlated to stimulus specifications within the preschool population. Where vision plays a key component in the assessment and management of orthoptic and ophthalmic cases it is important to remember that normal acuity of vision does not ultimately lead to correct shape discrimination[20]. It has been suggested that pictures and letters may be distinguished by overall shape, rather than resolving the line width[19,21,22].

Although the single solid geometric optotypes used in this study were not a resolution task, the results imply that within the pre-school population perception and recognition are influential factors in visual functions and should be considered when a child's vision is assessed for the first time in a clinical setting or screening programme. Further research needs to be conducted with a larger sample size of normal and amblyopic pre-school children on the subject of visual acuity and area of retinal stimulation.

CONCLUSION

The importance of discovering in detail what factors, apart from resolution, influence the assessment of visual acuity has been highlighted as the results imply (for a normal pre-school population) that optotype area on first examination was an influential factor in shape perception until the distinctive features were identified then recognised.

ACKNOWLEDGEMENTS

The author would like to thank Ms Shayne Brown and Dr Ian Story for their support during 1993, Mr Y Chen who checked all mathematical calculations, the ACT Department of Education and Training, the staff at Theodore Preschool and, Mr Steven Altree-Williams (Occupational Health & Safety Officer) from the Australian National University who assessed the lighting levels within the examination environment.

REFERENCES

1. Duke-Elder S. The physiology of the eye and of vision. System of Ophthalmology; vol (4). London: Henry Kimpton, 1968:665-667.
2. Eskridge JB, Amos JF, Bartlett JD. Clinical procedures in optometry. New York: Lippincott, 1991:1-761.
3. Frisen L, Frisen M. How good is normal visual acuity? - a study of letter acuity thresholds as a function of age. Graefes Arch Ophthal 1981; 215:149-157.
4. Tasman W, Jaeger E. Duane's Clinical ophthalmology. (Revised ed.) Philadelphia; JB Lippincott, 1992: Vol(1) 30-50 & (2)5-20.
5. Abrams D. Duke-Elder's practice of refraction (9th ed.) Edinburgh: Churchill Livingston, 1978:104-108.
6. Elliot R. A new linear picture vision test. British Orth J 1985;42:54-57.
7. Kay H. New method of assessing visual acuity with pictures. Brit J Ophthal 1983;67:131-133.
8. Kay H. A new picture visual acuity test. Brit Orth J 1984;41:77-80.
9. Peyman GA, Saunders DR, Goldberg MF. Principles and practice of ophthalmology. In: Philadelphia: WB Saunders, 1980:195-196.
10. Sheridan M. Diagnosis of visual defect in early childhood. Brit Orth J 1963;20:29-36.
11. Westheimer G. Visual acuity. In: Hart WM, ed. Adler's Physiology of the Eye. 9th ed. St. Louis: Mosby, 1993:531-547.
12. Tomlinson E, Martinez D. The measurement of visual acuity - comparison of Teller acuity cards with Snellen and MBL results. Am Orth J 1988;38:130-134.
13. Ffooks. Neurophysiology and assessment of visual function in children. Brit Orth J 1969;26:10-14.
14. Harrison A. Methods of assessing visual acuity in young children. Am Orth J 1975;25:109-114.
15. Hedin A, Olsson K. Legibility and the construction of a new visual acuity chart. Ophthalmologica 1984;189:147-156.
16. Woodruff M. Observations on the visual acuity of children during the first five years of life. Am J of Optom Arch Am Optom 1972;49:205-215.
17. Reed S. Psychological processes in pattern recognition. New York: Academic Press, 1973:11-86.
18. Sonsken P, Macrae A. Vision for coloured pictures at different acuities - the Sonsken picture guide to visual function. Dev Med Child Neurol 1987;29:337-347.
19. Mayer D, Gross R. Modified Allen pictures to assess amblyopia in young children. Ophthalmol 1990;97(6):827-832.
20. Davidoff J, Warrington E. A dissociation of shape discrimination and figure-ground perception in a patient with normal acuity. Neuropsychologia 1993;31(1):83-93.
21. Enns J, Girgus J. Developmental changes in selective and integrative visual attention. J Exp Child Psych 1985;40:319-337.
22. Wong D, Kay S. Chart for visual acuity screening. Brit J Ophthalmol 1989;73:457-460.

Australian Orthoptic Journal 1995, VOL 31: 21-27

ORIGINAL ARTICLE

COMPARISON OF CROWDED SINGLE OPTOTYPES WITH LINEAR ACUITIES IN AMBLYOPES

MEGAN WILLIAMS BAppSc(Syd)
TIFFANY WONG BAppSc(Syd)
HELEN GOODACRE MHlthScEd(Syd), DipAppSc(Orth)

Abstract

In order to evaluate the effectiveness of the letter matching test (LM test) in the detection of the crowding phenomena, the authors compared it to two conventional tests, the linear Snellens/Sheridan Gardiner test and the Sheridan Gardiner Singles test. The LM test differs from conventional tests in that the less complex singles test has been enhanced by the addition of four 'crowding bars' to increase sensitivity to the crowding phenomena. To find out if the LM test provides an accurate equivalent to the linear charts, visual acuities were compared in amblyopic patients (n = 15) and a control group of normals with no history of amblyopia (n = 30). The amblyopic group was found to have significantly different acuities when comparing results in the singles tests with the results in both the linear and LM test. In addition there was no significant difference found between the linear and LM test acuities. These findings are consistent with the hypothesis that the crowding bars contained in the LM test provide contour interactions which are similar to the linear chart, therefore providing a reliable alternative method to single optotypes in the measurement of vision in the young.
Key Words: *crowding phenomena, contour interactions, singles acuity, crowding acuity, pre-school children.*

INTRODUCTION

It is widely recognised that amblyopia is a major problem in the development of normal (equal) vision in children. The earlier it is detected, and treatment initiated, the greater the chance for the best possible outcome for vision. Early detection of amblyopia therefore is of major clinical importance.

Previous research by Stager[1], states "most amblyopia develops before age four when children are less verbal and less cooperative with complex visual tasks". A test therefore, is required, that is accurate in detecting amblyopia and is not too complicated for a child to comprehend.

Conventionally tests involving single optotypes have been used, such as the Sheridan Gardiner singles test. However, these tests have been found to be less effective

Address for correspondence: Helen Goodacre, School of Orthoptics, Faculty of Health Sciences, University of Sydney, P.O. Box 170, Lidcombe 2141. Submitted: January 1995. Accepted for publication: April 1995.

in detecting amblyopia than lines of letters "because single symbols do not present contour interactions that exploit the crowding phenomena"[2]. This phenomena has been described as when "neighbouring contours impair the resolution of a centrally fixed letter"[3].

Therefore to increase testing simplicity but still maintain sensitivity to amblyopia several 'crowded' acuity tests have been developed. These tests include optotypes flanked by bars on each side to introduce contour interactions without introducing a second test type. Rodier[4] studied crowding by using modified Allen pictures, and Stager[1] used letters with confusion bars on the BVAT. Stager[1] showed that the visual acuity recorded for normal and amblyopic eyes was lower when crowded optotypes were used than with isolated optotypes. Problems with these tests have been that Allen pictures are generally unknown and do not adhere to the Snellen's principle of its components subtending 1 minute of arc. The BVAT is currently scarcely used due to its expense and because it is not portable enough for vision screening in schools.

A test developed in the University of Otago in New Zealand[5] called the letter matching test (LM Test) has been devised that claims to be sensitive to the crowding phenomena yet is simple to comprehend. The aim of this study is to determine whether the LM Test is as sensitive to the crowding phenomena as the Snellens/Sheridan Gardiner linear test. If this is so it may then be useful in detecting amblyopia in young children.

METHOD

This study compares the LM Test it to the conventional Snellens/Sheridan Gardiner linear test and the Sheridan Gardiner singles test in amblyopic and normal subjects.

Acuity Tests

a) LM test: The LM Test uses 'crowded' optotypes to assess vision and is held at four metres (or two if necessary). It is a high contrast test that consists of black letters on white card (see Figure 1). The test is much the same as the Sheridan Gardiner test, as single letters, a matching card and the same letters , that is, A, H, O, U, T and V are used. The crucial difference of the LM test is the four black bars that surround the letters called 'crowding' bars. The crowding bars are positioned 1/2 width of the letter separation from the letters being viewed. Researchers found in 1990 that this separation is the best approximation of amblyopic linear vision in the BVAT testing context[1]. This test assesses vision from 6/60 to 6/6. All children were first introduced to the test at near and the 'matching' procedure explained.

b) Linear acuity: Linear acuity was

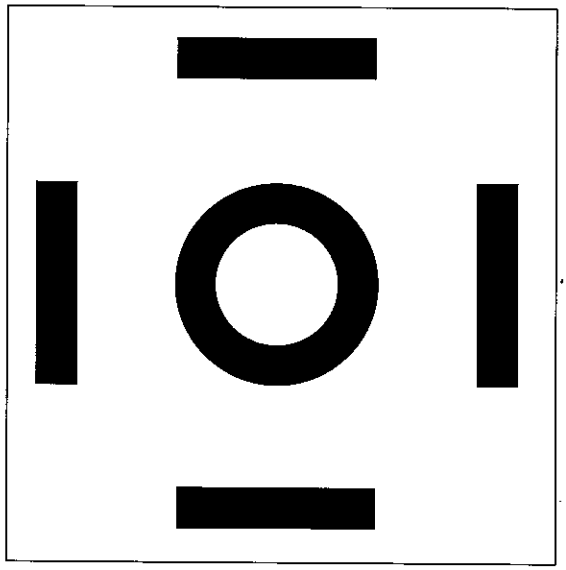


Figure 1: LM test optotype showing crowding bars surrounding the single letter.

assessed using the either the Snellens chart or the Sheridan Gardiner linear chart measuring the acuity from 6/60 to 6/5 at 6m. Amblyopes with acuity of less than 6/24 could not be included in the study as a line of less than three letters does not provide contour interactions which exploit the crowding phenomena[3].

c) Singles acuity: Isolated optotype visual acuity was tested with the Sheridan Gardiner singles test at 6m measuring acuity from 6/60 to 6/3.

Subjects

Subjects had to be old enough and of sufficient concentration to perform all three tests without a great loss of concentration. All subjects were examined by one of the three examiners using all three tests, except five amblyopes who were only tested with the linear and LM tests. Those with organic or neurological causes of decreased vision such as nystagmus or cataracts were not included in this study. Specially designed recording forms were used to ensure a random ordering of tests for each patient. For each separate test the eye assessed first was also randomly selected. These measures were undertaken to avoid the confounding effects of fatigue, loss of concentration and the learning curve. Responses were recorded when gained on the first and second attempt with no prompting. Testing was performed at primary schools and a public hospital.

a) Amblyopic subjects: Subjects were included as amblyopes if their linear acuity differed by one line or more between the two eyes. There were 15 subjects consisting of 13 children and 2 adults, whose ages ranged from 4 to 33 years (mean = 9.6 yrs). They were classified into anisometropic (40%), strabismic (20%) and a combination (40%) of them both.

b) Normal subjects: There were 30 normal subjects, 2 adults and 28 children who had less than a lines difference in linear visual acuity between the two eyes and a visual standard of greater than or equal to 6/6. Their ages ranged from 4 to 30 years with a mean of 7.4 years.

Scoring Procedure

In order to compare the three visual acuity tests a system of scoring had to be developed. This was because the linear chart contains different numbers of letters on each line, while the singles and LM test have only three letters per level of acuity. For the linear test a maximum score of three was attainable for each line with the overall minimum score for a complete line being 3 (6/60) and the maximum score being 21 (6/6). For incomplete lines each letter within that line was ascribed a fraction of three. This was calculated by dividing the number of letter gained by the number of letters in the line and multiplying this fraction by three. The converted scores were termed 'acuity units'.

As the LM test only measures visual acuity to 6/6, the linear test measures to a maximum of 6/5 and the singles measure to a maximum of 6/3, all results were truncated at 6/6 so comparisons could be made.

Statistical Analysis

Comparisons between tests within a subject type (amblyopic, preferred and normal eyes) were analysed using Friedman two way ANOVA. The comparison of each test between subject types was achieved through the use of a one way ANOVA.

To reduce the probability of a type one error, (a difference in performance between isolated/linear and surrounded optotypes

when there is actually no difference), the p for each individual test was set at 0.0056.

RESULTS

Mean visual acuity results (expressed in acuity units and the standard VA level) for normal and amblyopic subjects (amblyopic and preferred eyes) are summarised in Table 1 and Figures 2 & 3.

Amblyopic Subjects

The mean acuity of the amblyopic eyes when tested with the linear method was approximately a line and a half difference than when tested with the singles test ($p = 0.0019$). When tested with the LM test there was also found to be approximately one line and a half difference than when tested with the singles test (Figures 2 & 3). As expected both these differences were statistically significant ($p = 0.0019$). There was no significant difference in mean acuity of the amblyopic eye when tested with the linear chart and with the LM test.

When comparing mean acuities of the preferred eyes of the amblyopes between the three tests, there was no statistically significant difference between the linear and singles test, the singles and LM Test, or the linear and LM test (Figures 2 & 3).

Normal Subjects

When comparing mean acuities of normal subjects there was no statistically signif-

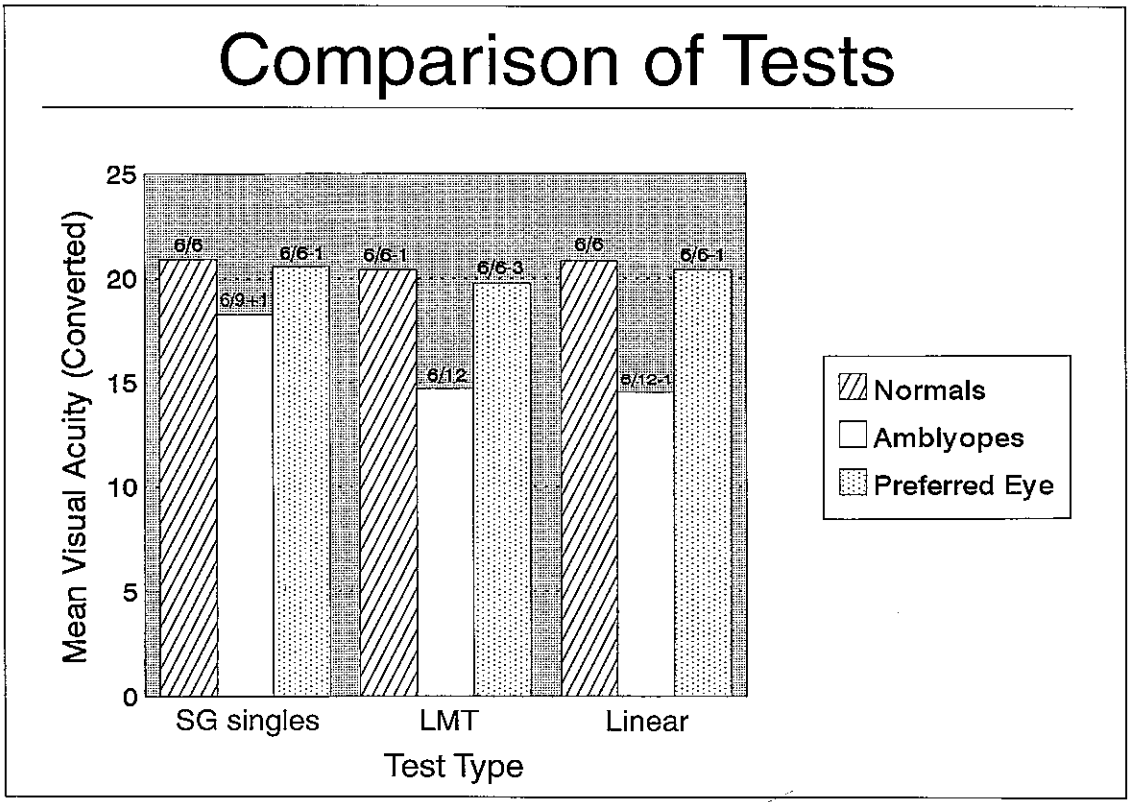


Figure 2: Comparison of mean visual acuity between the three vision tests for normal subjects and amblyopic subjects with amblyopic and preferred eyes.

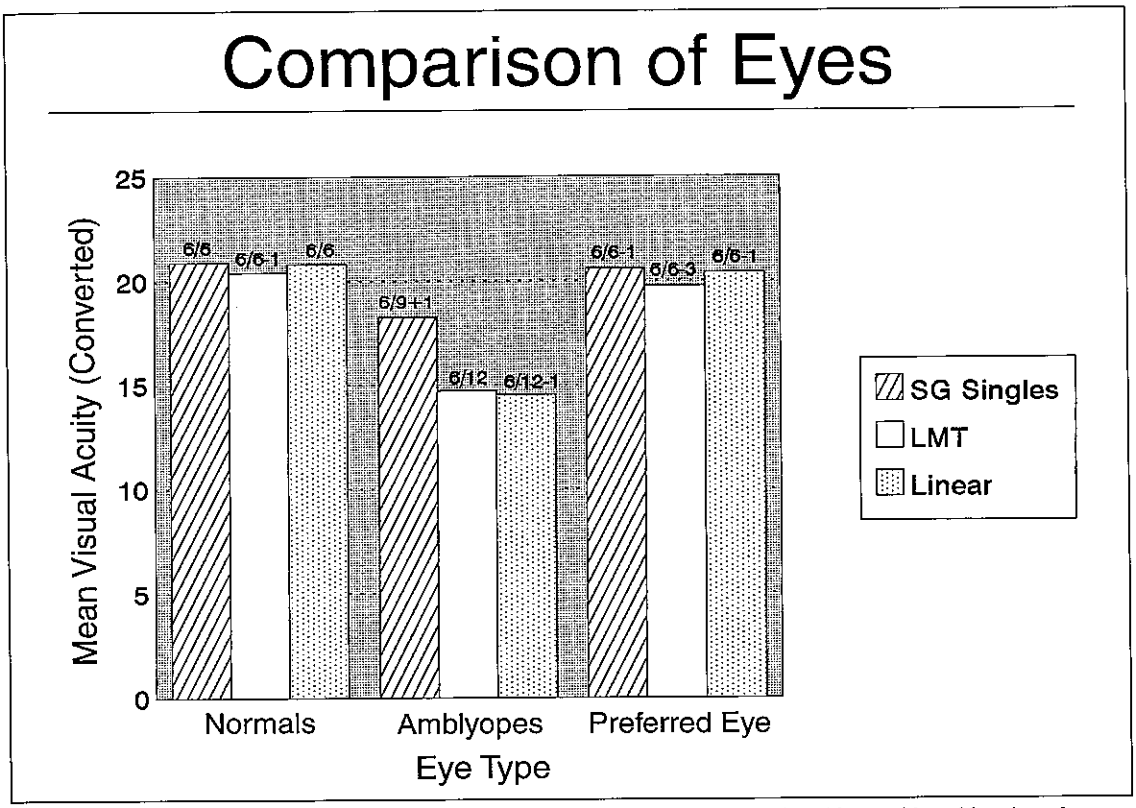


Figure 3: Comparison of mean visual acuity between normal subjects and amblyopic subjects with amblyopic and preferred eyes for each of the three vision tests.

icant difference found between the linear and singles test, the singles and LM test, or the linear and LM test (Figure 2).

Normal and Amblyopic Eyes

When comparing the mean visual acuity of the normal eyes and amblyopic eyes a significant difference was found on all three tests. The linear test showed a difference of over two lines (6.31 acuity units) between the amblyopic and normal eyes ($p<0.0001$); the Sheridan Gardiner singles test showed a difference of less than a line (2.64 acuity units $p<0.050$); the LM test showed a difference of approximately two lines (5.71 acuity units $p<0.0001$) (Figure 3).

When the mean visual acuity of normal eyes is compared with that of preferred eyes, there was no significant difference between them in all three tests.

DISCUSSION

In this study the amblyopic eyes showed a significant difference in mean acuities between the linear and singles, and LM test and singles tests, while the preferred eyes of the amblyopes and the normal eyes showed no significant difference between tests. The LM test and linear test show approximately equal acuities for amblyopic eyes and both are more sensitive to a reduction in vision than the singles test. Previous research has shown that single letters do not contain the means for detecting the crowding effect due to their lack of contour interactions[6]. The present study has shown that the addition

TABLE 1
Mean Visual Acuity for normal and amblyopic subjects.

	NORMAL SUBJECTS		AMBLYOPIC SUBJECTS			
	NORMAL EYES		AMBLYOPIC EYES		PREFERRED EYES	
	VA: Acuity Units	VA: Standard Format	VA: Acuity Units	VA: Standard Format	VA: Acuity Units	VA: Standard Format
Linear Chart	20.85	~6/6	14.54	~6/12-1	20.4	~6/6-1
LM Test	20.42	~6/6-1	14.71	~6/12	19.74	~6/6-3
Single Letters	20.92	~6/6	18.38	~6/9-1	20.6	~6/6-3

of contour interaction bars to single letters (the LM test) provides a test which is sensitive to the crowding phenomena and obtains results which are similar to the conventional linear test.

The results gained from comparing the mean acuities of the normal and amblyopic subjects in each of the three tests, were shown to be significantly different in all cases. Both the linear and LM test showed a difference of approximately two lines while the singles test showed a difference of less than a line. Although a significant difference was found between normal and amblyopic subjects in the singles test, it fails to demonstrate an obvious clinical difference between the two groups, as a discrepancy of a few letters may be attributed to other factors such as concentration. The LM test and the linear chart, however shows a recognisable clinical difference.

Research has noted that preferred eyes of amblyopes[7] and to a lesser extent normal eyes[3] are also sensitive to the crowding phenomena. One may then have expected to find a difference in mean acuities of normal eyes and preferred eyes between the tests with crowding (SG linear and LM test) and the single letter test in this study. Although the preferred eye acuities were less than the acuities of normal eyes in our study, they were not found to be significantly different. This may well be due to the truncation of the linear and singles results to the level of 6/6 where subtle differences were negated.

There are two main problems with this study that would need to be overcome in future research. Firstly is the method of scoring each of the three tests. If one letter in a line on the linear chart is missed the total score decreases only by a small fraction of three. Moving down the linear chart the numbers of letters per line increases, and the fraction becomes smaller. When using the LM test or singles test there is a maximum of three letters per level of acuity. If one letter is missed the total score is decreased by a whole unit. This may have increased the probability of gaining a type one error in this research (finding a significant difference when there is not one actually present).

Secondly the truncation of the results to

6/6 to aid comparison between tests, reduced the variability contained in our results on the higher acuity levels. Since statistical tests analyse variability, the presence of many similar scores (that is, 21 acuity units) may have hidden any interesting subtle findings.

When analysing the normal subjects' individual acuity levels the majority of subjects gained a visual acuity of 6/4 or 6/3 on the singles test if they were able to gain a visual acuity of 6/6 to 6/5 on the linear chart. This tends to agree with previous observations[8] that the singles acuity of normal eyes is approximately 6/4 to 6/3. It is suggested that in order for any decrease in vision to be detected when testing with Sheridan Gardiner singles, testing must be carried out to the level of 6/3.

It was also observed that a score of 6/5(-) or 6/5 on the linear chart approximated a score on the LM test of 4/4 (6/6). Further a score 6/6 (-) or 6/6 on the linear chart approximated a score on the LM test of 4/6 (+) or 4/4 (-) that is, 6/9 (+) or 6/6 (-). When testing linear acuity it was noticed that responses were slow for the 6/6 line subjects tended to gain a score of less than 4.4 (6/6) on the LM test. This tends to further suggest that the LM test is indeed able to detect crowding, though further investigation is warranted. Modification of the LM test to include letter sizes smaller than 4/4 is suggested.

CONCLUSION

This study showed that the LM test provides a good approximation of visual acuity to that obtained with a Sheridan Gardiner/Snellen's linear chart. It does indeed appear to be sensitive to the crowding phenomena occurring in amblyopes. What remains to be established is whether the LM test as simple as the Sheridan Gardiner singles test to comprehend in the population where it is most needed-: screening a large number of preverbal children who cannot perform linear tests.

ACKNOWLEDGEMENTS

The authors wish to thank Dr Rob Heard for his advice and assistance in the statistical analysis of the results.

REFERENCES

1. Stager, D. Everett, M. Birch, Ell Comparison of Crowding Bar and Linear Optotypes acuity in amblyopia. Am Orth Jnl. 1990, 40, 51-55.
2. Muller, P.. Uber das Sehen der Amblyopen. Ophthalmologica, 1951, 121, 143-149. Cited in Rodier, D.W. Mayer, D.L. Fulton, A. b.. Assessment of young amblyopes; array v's single picture acuities. Ophthalmology1985, 92, 1197-1202.
3. Ciuffreda, K.J. Levi, D.M. Sclenois, A.. Amblyopia basic and clinical aspects 1991. Boston, U.S.A.: Butterworth Heinemann.
4. Rodier, D.W. Mayer, D.L. Fulton, A.B.. Assessment of young amblyopes; array v's single picture acuities. Ophthalmology 1985, 92, 1197-1202.
5. Parr, J.C. Clinical assessment of visual acuity. Transactions of the OSNZ 1981: 33, 157-167.
6. Flom, M.C. Weymouth, F.W. Kahneman, D.. Visual resolution and contour interaction. J Opt Soc Am 1963, 53, 1026-1032 Cited in Rodier, D.W. Mayer, D.L. Fulton, A.B.. Assessment of young amblyopes; array v's single picture acuities. Ophthalmology 1985, 92, 1197-1202.
7. Sparks, J.A. Johnson, J.. The significance of crowding. Transaction of the VIIth International Orthoptic Congress 1991, Nürnberg, Germany: Fahner Verlag.
8. Fitzgerald, A.. The incidence of reduced visual acuity and squint in pre-school children aged three in Australia. Aust Orth Jn 1994, 30: 17-25.

Australian Orthoptic Journal 1995, VOL 31: 29-33

ORIGINAL ARTICLE

VISUAL SCREENING IN A NEPALESE COMMUNITY

CATHERINE DEVEREUX, DipAppSci(Orth), BEd, MAppSci(Orth)
LUCINA BENICH, DipAppSci(Orth)
ANDREA DRUM Orthoptic student

Abstract

Health and vision screening is not routinely undertaken in Nepal. The control of severe ocular dysfunction such as vitamin A deficiency and cataract blindness is understandably of great concern to both local health authorities and non government organisations. In January 1994, a screening program was conducted in two orphanages in the Kathmandu district. A total of 220 children were examined who ranged in age from 5-17 years. The failure rate on tests of vision and ocular motility was 23.6% as defined by the examiners' criteria for this project. A variety of reasons including the lack of public health programs, low socio-economic status, and the influences of adolescent myopia and inadequately trained convergence are discussed as possible contributors to this high referral rate.
Keywords: *screening, vision, convergence insufficiency, exotropia, xerophthalmia.*

INTRODUCTION

Nepal is considered to be an economically depressed country with few natural resources. Forty percent of government health expenditure is externally financed from overseas aid and annual per capita expenditure is less than A $2.00[1]. An estimated 2.8% of the Nepalese population are partially blind[2]. Xerophthalmia, the ocular manifestation of Vitamin A deficiency is measured to be responsible for 18-43% of cases of childhood blindness[3,4]. Chronic under nutrition affects up to 80% of children and life expectancy is very low (52 years)[2]. Health screening is not conducted in this country. It is vital that vision screening programs be undertaken as extensively as possible to curtail preventable vision disorders. Blindness and vision defects, associated diet problems, and myopia and amblyopia are preventable, but at present are not screened for.

While in Nepal to undertake clinical placement and conduct ophthalmic assistant training our group from La Trobe University in Melbourne, had the opportunity to under-

Address for correspondence: School of Orthoptics, La Trobe University, Bundoora, 3083. Victoria. This paper was presented in part at the 51st Annual Orthoptic Association of Australia Scientific Conference, NSW 1994. Submitted: January 1995. Accepted for publication: April 1995.

take a vision screening program. With the assistance of the orthoptists and administrators of the Kathmandu Eye Hospital screening for vision and ocular motility disorders was conducted in 2 separate orphanages in the Kathmandu district. The main aim of this project was to gain some insight into the incidence of visual problems in the child/adolescent community and allow those who failed the screening the opportunity to have further ocular assessment.

METHOD

Subjects and Location

Two hundred and twenty Nepalese children were involved in 2 screening programs, one of which was conducted at a city orphanage (Balmundir) where 136 male and female children ranged in age from 5-17 yrs, and the other at a rural boys orphanage (Panchkhal) where 84 boys ranged from 11-17 years. The largest number of children possible were examined during the allocated time with language and administrative difficulties arising at times. Balmundir orphanage was supported by Nepalese Royalty and the Children's Foundation and located in central Kathmandu while Panchkhal was in a small town 2 $^{1}/_{2}$ hours by road north of Kathmandu. Here funding was provided by the National Foundation of Boys Orphanages. The poor facilities indicated a shortage of funding in comparison to Balmundir.

Procedure

The examiners involved in the screenings included 2 Australian orthoptists, 2 La Trobe University final year orthoptic students, 2 Nepalese orthoptists and 1 Nepalese ophthalmic assistant.

Testing was conducted in the open air at Panchkhal as suitable indoor facilities with adequate illumination did not exist. Testing at Balmundir was conducted in a hall with vision charts placed next to open windows for maximum natural illumination.

A criteria for pass or fail was designed prior to the screening and was used at both orphanages. This criteria was:

- Vision of 6/12 or worse in either eye or a difference of more than one line between each eye;
- Convergence insufficiency of 15cm or greater associated with exophoria and/or symptoms;
- Manifest or significant intermittent strabismus;
- Mechanical defect;
- Other significant ocular conditions such as iris coloboma, entropian, conjunctivitis.

All children being screened completed a form with their name, age and sex prior to examination. The tests completed in the screening examination were as follows:

- Visual acuity of the right and left eye using linear chart where possible;
- Cover test at 1/3 and 6 metre distances;
- Extra ocular muscle excursions;
- Convergence near point;
- Stereoacuity if the examiner needed to confirm the presence of binocular single vision;
- Examination of the conjunctiva for signs of xerophthalmia.

Any children with evidence of xerophthalmia in the form of Bitots spots (stage 1B) were given oral Vitamin A tablets[1-4].

The supervisors at the orphanage were given a list of those children who failed the screening examination.

RESULTS

Table 1 gives an overview of the overall

failure rate at the 2 orphanages. A larger number of the children at the city orphanages failed the screening test.

TABLE 1
Overall failure rate.

	Fails	Total	%	Sex
Balmundir	37	136	27.2	24F 13M
Panchkhal	15	84	17.8	15M
Total	52	220	23.6	

Specific ocular defects have been categorised in Table 2 with the incidence rates of each defect. It should be noted that 4 children who had a vision defect also had convergence insufficiency, but reduced vision was considered to be the more significant problem.

TABLE 2
Incidence of ocular defects.

Defect	No	% of Fails	% of Total
Vision	30	57.7	13.6
Convergence Insufficiency	13	25.0	5.9
Mechanical	2	3.8	0.9
Inter/Manifest Squint	3	5.8	1.4
Other ocular	4	7.7	7.7
Totals	52	100%	23.6

Table 3 outlines the number of children with ocular defects in particular age groups. There is a high number (11 of 12) failing the vision test at age 8 with 3 of 7 failing at age 12 and 4 of 7 failing at age 14 years.

TABLE 3
Ocular defects related to age.

Age	No. with defect	% of total defects
5	1	1.9
6	1	1.9
7	1	1.9
8	12	23.1
9	2	3.8
10	6	11.5
11	4	7.7
12	7	13.5
13	0	0
14	7	13.5
15	5	9.6
16	3	5.8
17	3	5.8
	52	100%

DISCUSSION

The referral rate obtained from screening 220 Nepalese children was 23.6% as defined by the criteria for this screening project. This criteria for fail would be considered to be less strict than in other screening programs. The examiners justification for this was to encourage those with significate ocular defects to be referred for further assessment. If more rigid criteria were applied, an even higher referral rate in this population would be expected.

In comparison, the combined prevalence of strabismus and amblyopia has been reported to be significantly lower, around 5%[5]. Other studies have reported the incidence of pre school amblyopia as between 1.2% and 5.6% as documented by De Becker et al[6].

It is important to hypothesise regarding the reasons for such a high referral rate in the Nepalese orphanages. One of the probable reasons for the large difference in

referral rates would be the lack of visual assessment of young Nepalese children at an age where the visual system is still immature and visual improvement would be possible with appropriate intervention. This is particularly appropriate in cases of refractive error and amblyopia. The importance of detection of visual problems in pre-school children was evident from a Lancet study which found 50% fewer visual problems in children who had undergone screening[7]. In the Western world a generally lower referral rate has been attributed to public health and screening programs.

Other contributing factors may include malnourishment (stunted growth observed in some of the children), poor socio-economic status and inadequate facilities in comparison to Western conditions. Even in Australia, recent studies indicate that the state of a person's health is influenced by the socio-economic group to which an individual belongs[5-8]. Xerophthalmia with resultant visual disturbance is directly related to inadequate nutrition. A population of orphans may also have an increased number of disabilities which may in turn be associated with a higher incidence of ocular disorders as has been documented by Pitt and Jesse[8].

It is interesting to observe that all of the children with squint were exotropic, either intermittent or manifest. Jenkins[9] reported in an IOA study that Nepal was found to have the highest incidence of exotropia (76%) as compared to esotropia. A significant relationship was found to exist between the prevalence of exotropia and light intensity and Asian subjects were found to have higher prevalence of exotropia than Caucasians or Africans[9]. All children with significant squint in this study were found to be exotropic with many of those with convergence weakness possessing large exophorias. It has been postulated by Donders[10] that Asian countries have a higher incidence of myopia, exotropia and decompensating exophorias. In a country such as Nepal where schooling is non-intensive the accommodation/convergence relationship would not be maximally stimulated from an early age. These factors may impact on each other increasing the incidence of convergence weakness.

This screening also failed 30 (57.7%) children for reduced vision in one or both eyes. Some of this is definitely attributable to uncorrected refractive error, possibly myopia in the older age groups although this is not apparent from the dispersion of age related defects in Table 3.

There are a number of limitations to this project. Testing conditions at both of the orphanages were far from ideal. There was no direct lighting for vision charts and children were cramped and could easily have been distracted by others excitedly waiting their turn for examination. Several different examiners with varied levels of training conducted the testing simultaneously which may induce some inter-examiner error. Not every child underwent every test, some examiners failed children as soon as they did not pass one test giving no indication of whether the child possessed multiple visual problems. Language may have been a barrier with some of the children, slowing the examination and making it more difficult. The small sample size can also be criticised as not being a representative sample of Nepalese children and adolescents. However the study was not designed to be methodologically rigorous but was to be a benefit to participants and to provide information regarding the incidence of visual defects in this community. When

this program was designed, time constraints, limited resources and facilities were taken into account.

CONCLUSION

This screening project examined 220 Nepalese orphans aged from 5-17 years in the Kathmandu district in 1994. These results gave Nepalese authorities information regarding the incidence of vision and ocular motility disorders in this community. As discussed, a variety of factors could contribute to the very high referral rate of 23.6% observed in this population. Many of the defects found would be considered preventable and screening would be of great value in detecting these problems. Local health authorities should be encouraged in future to undertake preventative screening rather than allocate all resources to the less cost efficient treatment of vision and health problems.

ACKNOWLEDGEMENTS

The authors would like to thank the othoptists of the Kathmandu Eye Hospital for the initiation of, and assistance with, this screening program.

REFERENCES

1. World Bank. Nepal social sector strategy review. Report No. 7498-NEP. New York: World Bank, 1989.
2. World Bank. Nepal policies for improving growth and alleviating poverty. Washington D.C.: World Bank, 1989; 53-55.
3. Upadyhay, MP. et al. Xerophthalmia among Nepalese children. Amer J. of Epidemiol. 1985; 121: 71-76.
4. Brilliant GE. The epidemiology of blindness in Nepal. Chelsea, MI. SEVA Foundation, 1988, 331-75.
5. Ehrlich M, Reinecke R, and Simons K. Pre-school vision screening for amblyopia and strabismus. Surv. Ophthalmol, 1983; 28 (3): 145-163.
6. DeBecker I, MacPherson H, LaRoche R, Braunstein J, Cottle R, McIntyre L and Kozousek V. Negative predictive value of population - based pre-school vision screening programme. Ophthalmol, 1992; 99(6): 998-1003.
7. Feldman W, Milner R, Sackett B and Gilbert S. Screening - Effects of pre-school screening for vision and hearing on prevalence of vision and hearing problems 6-12 months later. The Lancet. Nov 8, 1980; 1014-1016.
8. Pitt, A and Jesse, K. The incidence of visual dysfunction and effectiveness of orthoptic intervention in intellectually impaired children. A report of a study in the North Eastern Region. Melbourne. 1987.
9. Jenkins R. Demographics: Geographic variations in the prevalence and management of exotropia. Amer. Orth. J. 1992; 42: 82-87.
10. Donders FC. An essay on the nature and consequences of anomalies of refraction. Oliver Ca, Ledl Blaiston and Son, Philadelphia 1899.

Australian Orthoptic Journal 1995, VOL 31: 35-40

ORIGINAL ARTICLE

STEREOACUITY AND FUSIONAL VERGENCE RANGES OF SIBLINGS OF CHILDREN WITH FAMILIAL STRABISMUS

DONNA FERGUSON BAppSc (Hons)
ALISON MAYO BAppSc (Hons)
ELAINE CORNELL DipAppSc MA DOBA
ROBERT HEARD PhD

Abstract

Genetic factors play an integral role in the cause of many cases of strabismus and it is currently believed that the mode of transmission is multifactorial with a threshold effect, that is, that specific genes must be present above a certain threshold before a strabismus is produced. If this is the case, then it is likely that close relatives of those with strabismus may also possess some of these 'abnormal' genes, but to a lesser extent, and therefore have subtler abnormalities of binocular vision without eliciting a manifest strabismus.
This assumption was tested by measuring the stereoacuity and fusional vergence ranges of 16 non-squinting siblings of children with early onset strabismus. Analysis of the data showed that compared with a control group of children with no family history of strabismus, there was a significant decrease in the stereoacuity of the experimental group (p=0.0245) but no significant difference in the fusional vergence ranges.
It is possible that defects of the vergence response may be too subtle to be elicited by this method and more information may be gained by studying the dynamics of the response. Nevertheless, the reduced stereoacuity supports the multifactorial theory for the inheritance of strabismus.
Keywords: *inheritance, genetics, strabismus, stereoacuity, fusional vergence.*

INTRODUCTION

Observations on familial strabismus have been documented since the origins of the medical profession. Hippocrates is said to have stated that:

> "the children of parents having distorted eyes, squint also for the most part"[1]

The relative failure of the Mendelian model to explain the inheritance of strabismus, along with the observation that, in strabismus, a continuum exists among individuals (from orthophoria to heterotropia) gives credence to the current belief that 'familial' strabismus is most probably

Address for correspondence: School of Orthoptics, Faculty of Health Sciences, The University of Sydney, PO Box 170, Lidcombe, NSW, 2141. Ph. (02) 412 3004 Fax. (02) 415 2314. Submitted: December 1994. Accepted for publication: April 1995.

due to multifactorial inheritance with a threshold effect[1,2a,3]. Basically it is a condition which is expressed when independently inherited genes work additively to produce the specific strabismus genotype, but only when a distinct threshold is exceeded[1].

In multifactorial inheritance it is recognised that the parents are unlikely to be genetically uniform in regard to the presence of 'abnormal' genes, therefore, each different parental gene combination will produce a different frequency of affected offspring, as affectation is dependent on how many abnormal genes are present in the combined parental gene pool. Due to this complexity, it is usually impossible to predict the likelihood that an offspring will be affected. However, it is known that with the birth of each affected child the likelihood of future offspring being similarly affected increases[1].

Binocular Single Vision is dependent upon the eyes' anatomy, sensory aspects and refractive state. These factors are thought to be determined polygenically, that is, numerous genes act to encode the development of the eyes and orbits, neurological connections, the grades of binocular single vision, the AC/A ratio and the refractive error[4].

Currently it is not known precisely what genes are involved and what quantities of abnormal genes are required for an individual to have strabismus. "However, whatever factors do exist, it is likely that they will have differing effects depending on their combinations"[1].

According to Spivey[4], the co-occurrence of poor vergence ability and hypermetropia mediate against the full development of binocular vision. Shclossman and Priestly[3] postulated that at least two genes (one affecting the ectoderm and the other affecting the mesoderm in the developing embryo) caused strabismus and that both genes probably had different patterns of inheritance. Richter[2b] also felt that two or more genes were responsible in the expression of strabismus. She held that these genes were independent and dominant; one determining a phoria (motor anomaly) and the other determining an anomaly of binocular vision (sensory anomaly.

It has been determined that phoria[1], sensory anomalies and refractive errors can all be inherited independently but if members of pedigrees with different types of anomalies marry, some of the offspring are more likely to have strabismus[1]. It has been shown[5,6] that many relatives of affected individuals demonstrate various slight abnormalities of binocular function without actually having strabismus, for example, non-squinting parents of children with early onset esotropia tend to have lower fusion ranges[5] and stereopsis[6] that those of the general population.

Although it is impossible to precisely predict the probability of offspring being affected, three conditions have been defined which significantly increase the likelihood of offspring inheriting esotropia[3]:

1. When a parent has an esotropia.
2. When parents are unaffected but there is a strong family history of the condition.
3. When parents are unaffected but have a low fusional ability and/or a significant refractive error.

So if it is possible for parents to pass the genes responsible for strabismus to their offspring, it is feasible to assume that non-squinting siblings of affected children may have inherited some aspects of abnormal binocular function from their parents without manifesting strabismus itself; that is, "there could be subnormal (or submodal)

functioning of one or more of the contributing components of binocular single vision"[7], namely simultaneous perception, fusion and stereoscopic vision.

The research question therefore addressed in this study was:

Do the non-squinting siblings of children with early onset esotropia of presumed genetic origin have reduced stereoacuity and/or reduced fusional vergence amplitudes?

METHOD

Subjects

Two groups of subjects were involved in the study. The control group consisted of 55 subjects between 4 and 13 years of age with no family history of strabismus. They were pupils from primary schools in the Sydney metropolitan region. The experimental group was obtained from orthoptic clinics and consisted of 17 subjects of the same age range, all of whom were non-squinting siblings of strabismic patients. (For the fusional vergence amplitude analysis, only 13 of these subjects were tested).

In order to be included in the study, control group subjects had to meet the following selection criteria:

- Written parental consent.
- No family history of strabismus (no affected siblings, parents, grandparents, cousins, paternal/maternal uncles or aunts.
- Non-corrected visual acuity of 6/6 or better with either eye (this was established during testing).

Stereoacuity

This was conducted using the Titmus Stereotest under good background illumination and held 40 centimetres from the subject's face. Firstly, gross responses, that is, the house fly, were checked. This was then followed by testing the animals, followed by the circles. Stereoacuities were recorded as the level before two consecutive mistakes were made. Any suspicious points were rechecked once. The test plate was always geld by the researches as suggested by the instruction guide accompanying the Titmus Stereotest.

Horizontal fusional vergence amplitudes

Horizontal fusional vergence amplitudes were then measured at ⅓m and 6m using a prism bar. One recording at each distance was taken due to time restrictions imposed by schools and clinics. An average of three measurements would have been preferred. However, all subjects were equally disadvantaged and therefore results would not have been influenced. Divergence range was measured first, followed by convergence range which was measured using a non-accommodative target, to remove as much as possible any effects of accommodation influencing convergence. The fusional vergence amplitude was taken as the power of prism prior to that which caused fusion to break. Subjects were not encouraged to maintain a single image as such fusional exertion does not occur in everyday situations.

RESULTS

Preliminary analyses showed that both the size of the latent deviation, the age of the subjects and the vision of both groups were equivalent.

Stereoacuity

The stereoacuity levels of the control group ranged between 40 and 80 seconds of arc with a mean score of 43.09 and a mode of 40 seconds of arc. The sibling group's stereoacuity had a range of 40 to

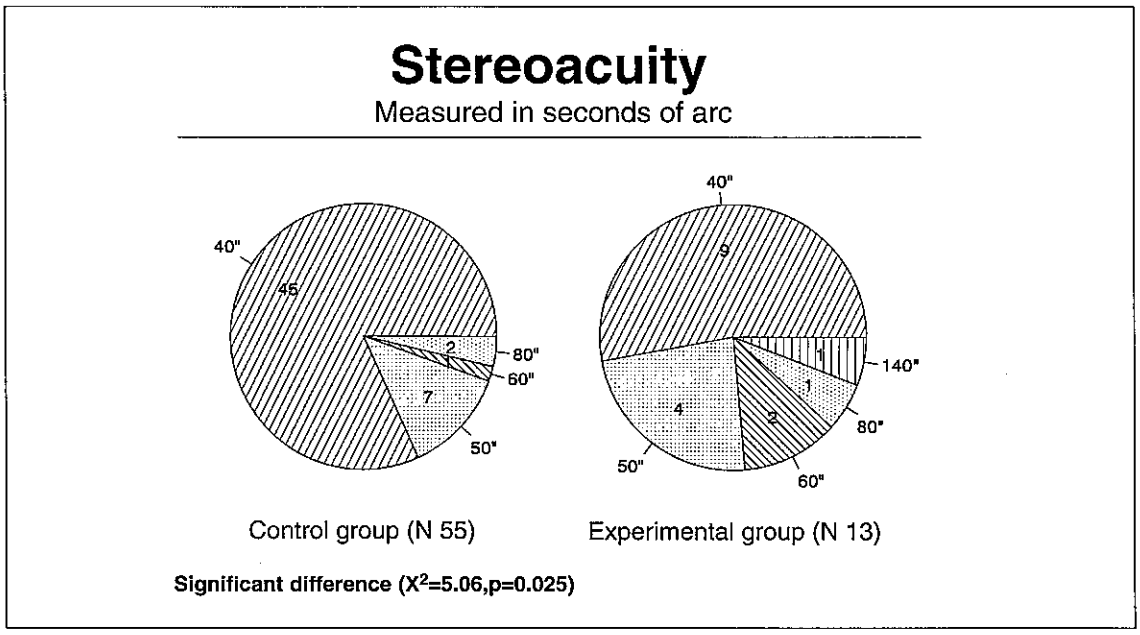


Figure 1. Stereoacuity

140 with a mean of 53.13 and a mode of 40 seconds of arc.

The two groups' stereoacuities were compared using a Kruskal-Wallis one-way non-parametric ANOVA test and were found to be significantly different at the 0.05 level ($X^2 = 5.06$ and $p = 0.025$). (See Figure 1). The two groups appear to have

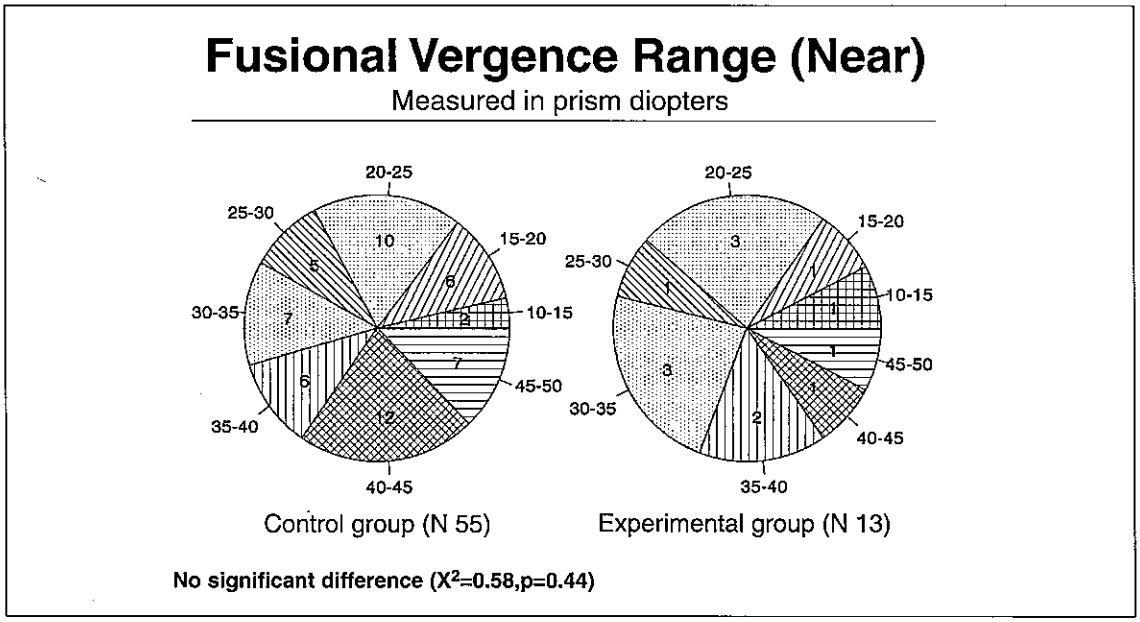


Figure 2. Fusional vergence range (near)

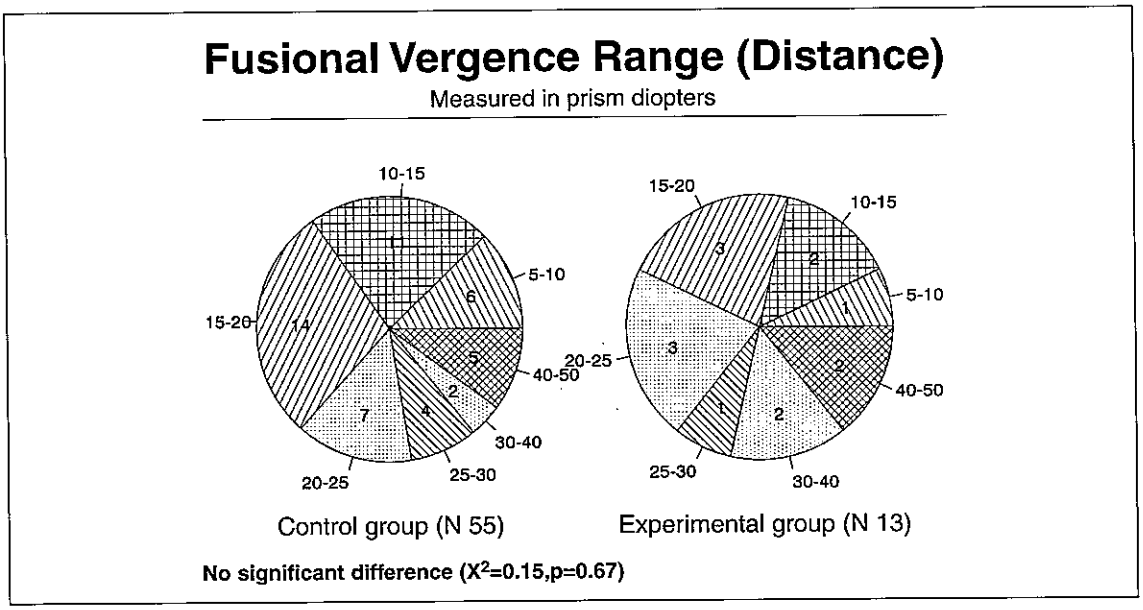


Figure 3. Fusional vergence range (distance)

come from different populations. This analysis indicates that there is a significant difference between the stereoacuity of siblings of children with a strabismus, that is, those children with a suspected affected gene pool, and the general population.

It can therefore be summarised that children who have a genetic predisposition to strabismus are more likely to have a reduced level of stereoacuity compared to a group of children for whom there is no evidence of a genetic predisposition.

Fusional vergence amplitudes

The base in and base out amplitudes of each group were added to give the total range for both near and distance. (See Figures 2 & 3).

A Kruskal-Wallis one-way ANOVA showed that no significant difference existed between the control group and the experimental group for near fusional vergence range ($X^2 = 0.58$ $p = 0.44$) and distance fusional vergence range ($X^2 = 0.0.15$ $p = 0.67$).

DISCUSSION

Stereoacuity

Although the sample size of the experimental group for the research was fairly small, it can be considered that the difference of stereoacuity levels between the two groups is indeed a clinically significant difference. This is due to the power of the experimental design. As the sample consisted of only 16 subjects in the experimental group, it would be expected that a significant difference would be found only if the effect size in the population was larger. If the effect size was only subtle then differences between the two groups would be likely to be found only if the sample sizes were much large, that is, approximately 200 subjects or more[8]. Therefore, for this difference between the two groups to be found with only 16 subjects it must be quite significant.

Fusional vergence amplitudes

The results for this variable would also have been affected by the low number of subjects in the experimental sample. The power of statistical tests to detect population differences depends on the size of the samples taken from that population and the magnitude of the effect within the population. Had there been a large effect within the population, a sample size of 13 should have been adequate to detect it. The fact that the probability levels in this study did not even approach significance suggests that, if the effect does occur in the population, it is unlikely to be a very large effect[8].

It may be that relatives of strabismic individuals demonstrated slight defects of fusional vergence amplitude. Perhaps these abnormalities are too slight to be detected by the methods used in this study. A sample size of approximately 200 would be needed to give a reasonable chance of detecting any subtle effect which may exist in this case. It was not feasible to collect a sample of this size for this study. However, further research should involve much larger samples than those used here.

Perhaps more refined methods of investigating the quality of the fusion reflex may be needed, for example, Schor[9] stated that "fusional vergences are controlled by a fast acting (transient) mechanism that aligns the eyes in response to retinal image disparity and a slow (sustained) mechanism that sustains binocular alignment". Studies such as this could be extended by the use of precise methods of analysing the vergence response to investigate this aspect of the vergence response, or possibly the latency and accuracy of the response.

SUMMARY

Reduced stereoacuity levels have been demonstrated in the siblings of children with early onset esotropia. This has shown support for the theory that several gene pairs are required to produce strabismus in a quantity large enough to reach a threshold level, and that without this threshold level being reached, the result may be a reduced level of binocular single vision.

That the experimental group did not demonstrate significant differences in fusional vergence amplitudes suggests either that this component of binocular single vision is not affected by inherited factors, or, possibly, that the effect is slight and requires a more detailed analysis to reveal any difference in this function.

REFERENCES

1. Grutzner P, Yazawa K, Spivey BE. Heredity and strabismus - Review. Sur. Ophth. 1970;14;No.6: 441-456.
2a. Francois J. Multifactorial or polygenic inheritance in ophthalmology. Dev. Ophth. 1985;10:26-28.
2b. Richter S. as cited by Francois.
3. Schlossman A, Priestly BS. Role of heredity in etiology and treatment of strabismus. AMA Arch. Ophth. 1952;47;No. 1:1-20.
4. Spivey BE. Strabismus: factors in anticipating its occurrence. Aust.J.Ophth. 1980;8;No.1:5-9.
5. Tydeman WL, Spaan JM, Polman L, van Balen A Th M, Abbink A. A viewpoint on the heredity of congenital strabismus. Year and source unknown.
6. Helveston EM. An atlas of strabismus surgery. IVth Ed Mosby 1993:410-414.
7. Mash AJ, Grutzner P, Hegmana JP, Spivey BE. Genetic determination of clinical eye disease. Year and publisher unknown: 261-275.
8. Welkowitz J, Ewen RB, Cohen J. Introductory statistics for the behavioural sciences. 2nd ed. Academic Press 1976.
9. Schor C. Fixation disparity: A steady state error of disparity induced vergence. Am. J. Optom & Phys Optics 1980:57;No. 9:618-631.

Australian Orthoptic Journal 1995, VOL 31: 41-44

CASE PRESENTATIONS

SUPRANUCLEAR DOWNGAZE SACCADIC PALSY PERSISTING AFTER TOTAL OPHTHALMOPLEGIA FOLLOWING SEVERE CLOSED-HEAD TRAUMA

ZORAN GEORGIEVSKI BAppSc(Orth)Hons [1,2]
LIONEL KOWAL FRACO FRACS [2]
ROBIN WILKINSON BAppSc(Orth)DOBA(D) [3]
PAUL J McCARTNEY FRACO FRACS [3]

[1] *School of Orthoptics, La Trobe University, Melbourne*
[2] *Ocular Motility Clinic, Royal Victorian Eye & Ear Hospital, Melbourne*
[3] *Eye Clinic, Royal Hobart Hospital, Hobart*

INTRODUCTION

Head injury has been seen to cause various visual disturbances and ocular motility defects, including supranuclear gaze palsy[1-4]. After sustaining considerable and diffuse brain damage, the patient presented in this case study has an unusual constellation of midbrain signs, most fascinating of which is a downward gaze saccadic palsy.

CASE STUDY

Mr C.B., aged 29 years, sustained a severe closed head injury following a logging accident in which a tree branch fell on his head, breaking his helmet. At the scene he was conscious, able to talk and move all limbs. He was airlifted to hospital where X-ray and CT scanning revealed a right temporal extradural haematoma and base of skull fracture, extending into the temporal and parietal regions and up to the vertex on the right side.

C.B. underwent right parieto-temporal craniotomy and evacuation of the extradural haematoma. A repeat CT scan showed right frontal lobe oedema with effacement of the ventricles. Post-operatively he was transferred to intensive care, gradually improving over the following weeks, and being discharged to a rehabilitation unit.

Initial eye examination was carried out during his hospitalisation. There was total ophthalmoplegia, his eyes fixed in the primary position. Best visual acuity that could be obtained was 3/60 and N48 OU.

Examination 10 weeks after the accident showed his uncorrected acuity to be 6/24 and N14 OD, 6/36 and N14 OS. Confrontation visual field testing appeared normal. There was no strabismus, but constant and involuntary horizontal scanning movement of

Address for correspondence: Georgierski, School of Orthoptics, Faculty of Health Sciences, La Trobe University, Bundoora, VIC 3083. E-mail ortzg@lure.latrobe.edu.au.

the eyes. Ocular motility examination showed defective horizontal and vertical gaze. Horizontally, saccadic movements were intact to the right and left, but smooth pursuit movements were absent with a series of cog-wheeling saccades to either side. Vertically, saccades were absent and some pursuit could be elicited into up-and downgaze, although this was limited. Bell's phenomenon was was present. Binocular vision testing showed central fusion disruption with vertical bobbing of the diplopic image as described by Pratt-Johnson and Tillson[5].

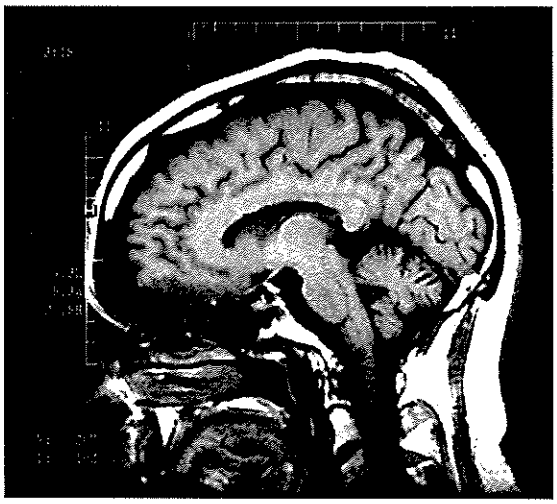


Figure 1. Sagittal section of MRI scan taken 12 months post-trauma.

Presently (28 months after the accident), this central fusion disruption has remained and C.B. complains of intermittent mostly horizontal diplopia, especially with smaller objects. He can superimpose in free-space but has poor motor fusion or fine stereopsis. All attempts to improve the situation with exercises or prisms have failed, and occlusion of any form has not been tolerated. He is also aware of a problem with looking into downgaze. Further, he has reported a few episodes of transient amaurosis fugax in the right eye, for which an extensive work-up has failed to find a cause.

C.B. wears glasses for pseudomyopia (-1.50DS OU) - uncorrected acuity improves from 6/12 to 6/5 OU with tropicamide (1%). He has orthophoria for distance and a 16$^{\Delta}$ exophoria with slow recovery for near fixation. Convergence is an effort, the near point being reduced to 15cm and his ability to jump converge from 6m to 20cm is poor. Vertical muscle balance testing with Maddox rod reveals a concomitant left hyper of 2-3$^{\Delta}$ in all testable positions. There is no apparent EOM dysfunction.

Horizontal smooth pursuit and saccades are intact, as are these movements into upgaze. Into downgaze, the vertical pursuit movements are slow and defective, although with effort his eyes can follow into extreme downgaze. On vertical saccadic testing, there is a complete inability to execute downward saccades. The eyes can be rotated downwards with a doll's head manoeuvre. However, despite this and the downgaze palsy, C.B. demonstrates chin down head jerks, which effectively reposition his eyes in a relatively elevated position. The vestibulo-ocular reflex is intact. On pupil testing, light-near dissociation remains a persistent sign.

Of particular interest is the downward gaze saccadic palsy, which is consistent with the MRI scan (12 months post-trauma) as showing residual changes of a small parenchymal hematoma in the lower thalamus/upper midbrain, just to the right of the midline at, below and posterior to the red nucleus (Figure1). This midbrain lesion is clearly evident in and correlates with an early CT scan (2 weeks post-trauma) (Figure 2).

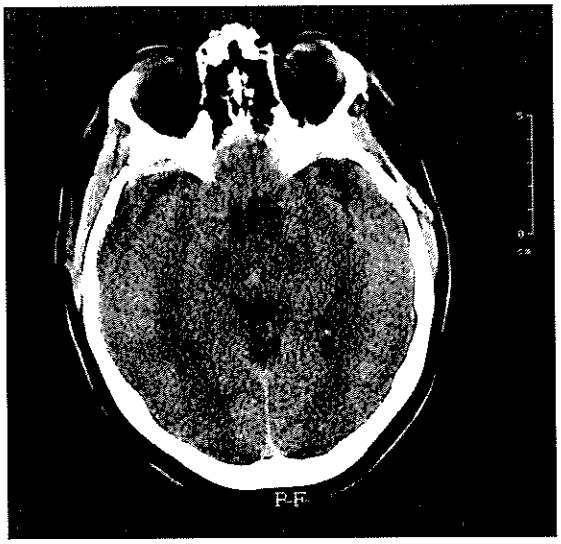


Figure 2. Transverse section of CT scan taken 2 weeks post-trauma

DISCUSSION

Preferential loss of downgaze eye movement is recognised to be an exceptionally rare finding[6-10]. Upgaze defects, on the other hand, are more common and typically feature in dorsal midbrain lesions, resulting in Parinaud's syndrome[6-8]. The patient presented in this case study initially sustained a total ophthalmoplegia, which mostly resolved, leaving a selective deficit of downward saccadic eye movement. This was obviously a result of diffuse brain damage, but the recovery of the ophthalmoplegia has unmasked a defect which can be attributed to discrete lesions in the prerubral fields of the rostral mesencephalon.

Firstly it is appropriate to briefly consider the vertical eye movement system pathway (Figure 3). Vertical gaze is brought about by bilateral stimulation from the frontal eye fields and superior colliculi for saccades, and the parieto-occipital areas for smooth pursuit, via the fronto- and occipito-mesencephalic pathways. The paramedian pontine reticular formation (PPRF) is also involved in the pre-programming of vertical eye movements[11]. Receiving strong input from the PPRF, the rostral interstitial nucleus of the medial longitudinal fascisulus (riMLF) is the principle premotor relay for vertical saccades[7], while the more caudally located interstitial nucleus of Cajal (INC) plays an important role in generating smooth pursuit and vertical gaze-holding[11]. These structures are thought to project fibres responsible for downward gaze ventral to the cerebral aqueduct, whereas those for upward gaze are projected dorsally (via the posterior commissure), to the oculomotor nuclei[11].

By studying the distribution of lesions leading to paralysis of vertical gaze in several cases, Büttner-Ennever et al[6] have proposed that the lateral part of the riMLF mediates downward saccades, while the medial part mediates upward saccades. Lesions involving the medial portion of the riMLF, however, would affect both upward and downward gaze since the lateral portion's efferent fibres and afferent blood supply would also be affected. It is believed that this may explain why isolated upward gaze palsy has not resulted from bilateral lesions in medial portion of the riMLF[6]. On the other hand, Pierrot-Deseilligny et al[7] believe that there is no such topographical separation for upward or downward gaze burst neurones, and that downgaze saccadic palsy results when the tracts efferent from the riMLF are damaged.

Whatever the exact physiology, to produce a deficit of downward saccadic eye movement there would have to be bilateral involvement, although this is not obvious on viewing C.B.'s scans. Certainly, it can be deduced from the clinico-pathologic studies[6-7] that the only way such a

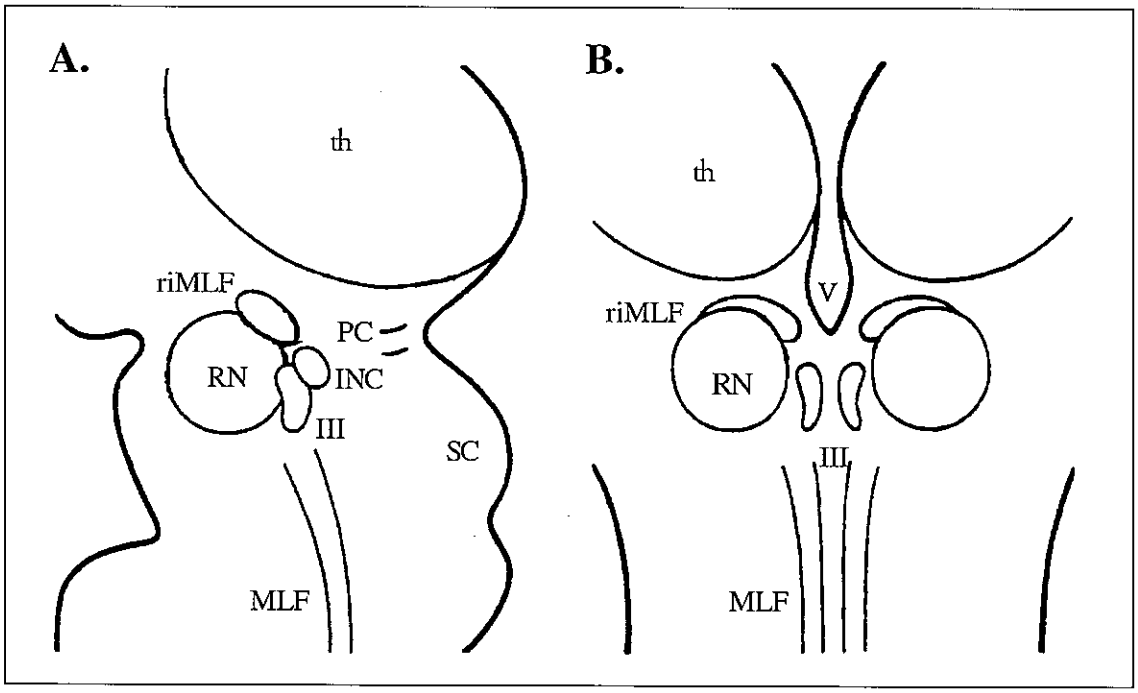


Figure 3. Sagittal (A) and coronal (B) views of structures involved with vertical gaze in the midbrain. th = thalamus: riMLF = rostral interstitial nucleus of the medial longitudinal fasciculus; INC = interstitial nucleus of Cajal: PC = posterior commissure; III = oculomotor nucleus; RN = red nucleus; MLF = medial longitudinal fasciculus; V= third ventricle.

defect can be produced is for the riMLF on each side to be affected, most likely due to interruption of the vascular supply to this area from the single unpaired posterior thalamo-subthalamic paramedian artery[6].

ACKNOWLEDGMENTS

The authors wish to acknowledge the support of the Sir Edmund Herring Memorial Scholarship, awarded by the Royal Automobile Club of Victoria in 1994, to Zoran Georgievski.

REFERENCES

1. Kowal L Ophthalmic manifestations of head injury. AustNZJ Ophthalmol 1992: 20(1): 35-40.
2. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. Surv Ophthalmol 1991: 35(4): 245-267.
3. Sabates NR, Gonce NA, Farris BK. Neuro-opthalmological findings in closed head trauma. J Clin Neuro-ophthalmol 1991: 11(4): 273-277.
4. Apostolou N. Ocular disorders in head trauma: A review. In: Advancing Rehabilitation. Proceedings of the Inaugural Conference of the Faculty of Health Sciences, La Trobe University 1994: 376-387.
5. Pratt-Johnson JA, Tillson G. Management of strabimus and amblyopia. A Practical Guide. New York: Thieme, 1994.
6. Büttner-Ennever JA, Büttner U, Cohen B, Baumgartner G. Vertical gaze paralysis and the rostral interstitial nucleus of the medial longitudinal fasciculus. Brain 1982: 105: 125-129.
7. Pierrot-Deseilligny C, Chain F, Gray F, Serdaru M,Escourolle R, Lhermitte F. Parinaud's syndrome. Electro-oculographic and anatomical analyses of six vascular cases with deductions about vertical gaze organization in the premotor structures. Brain 1982: 105: 667-696.
8. Glaser JS. Neuro-ophthalmology. 2nd ed.Philadelphia: Lippincott, 1990.
9. Halmagyi GM, Evans WA, Hallinan JM. Failure of dowward gaze. Arch Neurol 1978: 35: 22-26.
10. Spalton DJ. Vertical gaze, its control and disorders. Brit Orthopt J 1982: 39: 22-28.
11. Leigh RJ, Zee DS. The Neurology of Eye Movements. 2nd ed. Philadelphia: FA Davis, 1991.

BOOK REVIEWS

DIAGNOSIS OF DEFECTIVE COLOUR VISION

Jennifer Birch
Oxford Medical Publications.
Oxford University Press, Oxford 1993.
ISBN 0-19-261870-9 (hbk)
0-19-262388-5 (pbk)

This text provides invaluable information for practitioners involved in the examination of colour vision. The information is largely practical with sufficient theoretical background for explanation and credence. The style is fluid and the organisation is clear and logical. As a clinical reference tool, the practitioner can directly access information for patients with colour deficiencies on the occupational consequences of colour vision anomalies. These have been graded into careers requiring normal colour vision, and careers where colour deficiency is a disadvantage. The disadvantages can range from exclusion from matching tasks of small and large colour differences to exclusion from all colour recognition tasks. Problems with colour codes and driving are discussed.

Recommended colour vision tests for colour vision quality assurance assessments are outlined fully and are most useful in current industrial climates. Significant clinical decisions are required when assessing colour vision and Birch elaborates on this difficult area by dividing the approach to colour vision between congenital and acquired defects. The monitoring of retinal disease states by colour changes is fully described in the chapter on acquired colour vision defects. Explanations are given of pathological signs. The large range of test for patients under seven years of age shows the current varieties available and how they differ.

Importantly, Birch emphasises the manipulation of raw colour vision data in order to gain accurate interpretation of test results. There is a particularly clinically useful segment - tests in current use- that describes the appropriate analyses for test of hue discrimination such as the F-M 100 Hue Test. The errors in raw scores are well described.

Exposure is also given to the reader of the range of internationally available pseudoisochromatic, matching and discrimination tests. A sample case report for colour vision reporting is an invaluable inclusion in this monograph.

A review and update on colour deficiency aetiologies precedes the tests and clinical components of this book. Some references to the very earliest theories of colour vision mechanisms provide an historical perspective on this currently useful but somewhat under utilised clinical indicator which is colour assessment. The approach by Birch to this topic is effective in delivering the topic of colour vision to both theorist and the clinician.

BOOK REVIEWS

CONTACT LENS PRACTICE: A CLINICAL GUIDE

Robert Fletcher, Luigi Lupelli and Angela L. Rossi.
Blackwell Scientific Publications 1994.
ISBN 0-632-03287-1

The aim of the authors as stated was "to offer a concise account of contemporary contact lens practice". The authors have considered the history and development of both hard and hydrogel lenses in detail.

Part 2 concentrates on the hard lenses covering their chemical composition in detail, although this maybe irrelevant to the practitioner. Comprehensive information is provided for the fitting of the appropriate lens for the appropriate eye. Many worthwhile colour plates illustrate the points made in the text. Keratoconus fitting, the use of lenses to hold the progress of the cone, is discussed, as is also the use of the pick a back (piggy back) lens. Other areas of important information that the reader can gain from the authors include:

Assessment of corneal performance.

The aim of best fit and fluorescene pattern assessment.

Hard lens (gas permeable) care.

The use of sufacants and enzyme agents to combat the problem of denatured protein together with the complication of not addressing these problems.

Details of hyrdogel lenses, their types and uses are thoroughly looked at, examining factors such as mode of wear and water content with regard to oxygen demand being a consideration in the dry eye syndrome.

The text is well supported by relevant illustrations and attention is given to the assessment of fit and lens care and the complications caused by non compliance.

Overall the text provides detailed data on all aspects of contact lenses, from the history of the development of the first lenses to the fitting and handling and the use of sufacants.

Modification of contact lenses, review of the optical features, presbyopia and the use of contact lenses in infancy are included in the Appendix.

The orthoptic practitioner will find this text a useful detailed presentation to compare with their own clinical approaches and the statements of techniques put forward by the authors.

Geraldine McConaghy

NAMED LECTURES, PRIZES AND AWARDS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA INC.

THE PATRICIA LANCE LECTURE

1988 (Inaugural)	Elaine Cornell
1989	Alison Pitt
1990	Anne Fitzgerald
1992	Carolyn Calcutt
1993	Assoc. Professor Judy Seaber

THE EMMIE RUSSELL PRIZE

1957	Margaret Kirkland	Aspects of vertical deviation
1959	Marion Carroll	Monocular stimulation in the treatment of amblyopia exanopsia
1960	Ann Macfarlane	A study of patients at The Children's Hospital
1961	Ann Macfarlane	Case history: "V" Syndrome
1961	Margaret Kirkland	Post operative diplopia in an adult
1962	Adrienne Rona	A survey of patients at the Far West Children's Health Scheme, Manly
1963	Madeleine McNess	Case History: right convergent strabismus
1965	Margaret Doyle	Diagnostic pleoptic methods and problems encountered
1966	Gwen Wood	Miotics in practice
1967	Sandra Hudson Shaw	Orthoptics in Genoa
1968	Leslie Stock	Divergent squints with abnormal retinal correspondence
1969	Sandra Kelly	The prognosis in the treatment of eccentric fixation
1970	Barbara Dennison	A summary of pleoptic treatment and results
1971	Elaine Cornell	Paradoxical innervation
1972	Neryla Jolly	Reading difficulties
1973	Shayne Brown	Uses of Fresnel prisms

NAMED LECTURES, PRIZES AND AWARDS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA INC.

THE EMMIE RUSSELL PRIZE

1974	Francis Merrick	The use of concave lenses in the management of int. divergent squint
1975	Vicki Elliot	Orthoptics and cerebral palsy
1976	Shayne Brown	The challenge of the present
1977	Melinda Binovec	Orthoptic management of the cerebral palsied child
1978	Anne Pettigrew	
1979	Susan Cort	Nystagmus blocking syndrome
1980	Sandra Tait	Foveal abnormalities in ametropic amblyopia
1981	Anne Fitzgerald	Assessment of visual field anomalies using the visually evoked response
1982	Anne Fitzgerald	Evidence of abnormal optic nerve fibre projection in patients with Dissociated Vertical Deviation - a preliminary report
1983	Cathie Searle	Acquired Brown's syndrome: a case report
1983	Susan Horne	Acquired Brown's syndrome: a case report
1984	Helen Goodacre	Minus overcorrection: Conservative treatment of intermittent exotropia in the young child
1985	Cathie Searle	The newborn follow up clinic: A preliminary report of ocular anomalies
1988	Katrina Bourne	Current concepts in restrictive eye movements. Duane's retraction syndrome and Brown's syndrome
1989	Lee Adams	An update in genetics for the orthoptist, a brief review of gene mapping
1990	Michelle Gallaher	Dynamic Visual Acuity versus Static Visual Acuity. Compensatory effects of the VOR
1991	Robert Sparkes	Retinal Photographic Grading: the orthoptic picture
1992	Rosa Cingiloglu	Visual Agnosia: An update on disorders of visual recognition
1993	Zoran Georgievski	The effects of central and peripheral binocualar visual field masking on fusional-disparity vergence
1994	Rebecca Duyshart	Visual acuity: Area of retinal stimulation

NAMED LECTURES, PRIZES AND AWARDS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA INC.

THE MARY WESSON AWARD

1983 (Inaugural)	Diana Craig
1986	Neryla Jolly
1989	Not awarded
1992	Kerry Fitzmaurice

PAST PRESIDENTS OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA

1945-46	Emmie Russell	1970-71	Neryla Heard
1946-47	Emmie Russell	1971-72	Jill Taylor
1947-48	Lucy Willoughby	1972-73	Patricia Lance
1948-49	Diana Mann	1973-74	Jill Taylor
1949-50	E D'Ombrain	1974-75	Patricia Lance
1950-51	Emmie Russell	1975-76	Megan Lewis
1951-52	R Gluckman	1976-77	Vivienne Gordon
1952-53	Patricia Lance	1977-78	Helen Hawkeswood
1953-54	Patricia Lance	1978-79	Patricia Dunlop
1954-55	Diana Mann	1979-80	Mary Carter
1955-56	Jess Kirby	1980-81	Keren Edwards
1956-57	Mary Carter	1981-82	Marion Rivers
1957-58	Lucy Retalic	1982-83	J Stewart
1958-59	Mary Peoples	1983-84	Neryla Jolly
1959-60	Patricia Lance	1984-85	Neryla Jolly
1960-61	Helen Hawkeswood	1985-86	Geraldine McConaghy
1961-62	Jess Kirby	1986-87	Alison Terrell
1962-63	Patricia Lance	1987-88	Margaret Doyle
1963-64	Leonie Collins	1988-89	Margaret Doyle
1964-65	Lucy Retalic	1989-90	Leonie Collins
1965-66	Beverley Balfour	1990-91	Leonie Collins
1966-67	Helen Hawkeswood	1991-92	Anne Fitzgerald
1967-68	Patricia Dunlop	1992-93	Anne Fitzgerald
1968-69	Diana Craig	1993-94	Barbara Walsh
1969-70	Jess Kirby		

EDUCATIONAL FACILITIES FOR UNDERGRADUATE AND POST-GRADUATE ORTHOPTIC PROGRAMMES RECOGNISED BY THE ORTHOPTIC ASSOCIATION OF AUSTRALIA

NEW SOUTH WALES

School of Orthoptics
Faculty of Health Sciences
The University of Sydney
East Street, Lidcombe NSW 2141
Telephone: (02) 646 6250 Facsimile: (02) 646 6359
Head of School: Neryla Jolly

VICTORIA

School of Orthoptics
Faculty of Health Sciences
La Trobe University
Bundoora VIC 3083
Telephone: (03) 9479 1920 Facsimile: (03) 9479 3692
Head of School: Associate Professor Alison Pitt

STATE BRANCHES OF THE ORTHOPTIC ASSOCIATION OF AUSTRALIA

NEW SOUTH WALES

President:	J Kelly
Hon Secretary:	E. Cornell, PO Box 282, Lidcombe 2141
	Pager: 016 28 1059

VICTORIA

President:	B Devidas
Hon Secretary:	R Duyshart, PO Box 487, Carlton South 3053

QUEENSLAND

President:	J Hall
Hon Secretary:	D Gilmour, 22 Lonecroft Street, Brighton 4017

SOUTH AUSTRALIA

President:	A Burr
Hon Secretary:	A Burr, 27 Tennyson Drive, Beaumont 5066

WESTERN AUSTRALIA

President:	D Gilchrist
Hon Secretary:	M Tangney, WA Eye Clinic, Royal Perth Hospital, PO Box X2213, Perth 6001

AUSTRALIAN CAPITAL TERRITORY

Contact:	B Jennings, 81 Shannon Circuit, Kaleen 2617

TASMANIA

Contact:	J Barbour, "Ericvale" Leighlands Road, Evandale 7212