AUSTRALIAN

ORTHOPTIC JOURNAL

1978

Volume 16

TRANSACTIONS OF

35th ANNUAL SCIENTIFIC MEETING

CONTENTS

ORTHOPTIC ASSOCIATION OF AUSTRALIA

OFFICE BEARERS, 1978

President:	Miss Patricia Dunlop
Immediate Past President:	Miss Helen Hawkeswood
President Elect:	Miss Mary Carter
Hon. Secretary:	Mrs. A. Hitch
Hon. Treasurer:	Miss J. Stewart

STATE REPRESENTATIVES ON COUNCIL

N.S.W.	Misses S. Brown, S. Gregg, Mrs. E. Cornell.
VIC.	Mesdames D. Craig, P. Wister, Miss E. Richardson.
QLD.	Miss J. Kirby.
S.A.	Mrs. C. Coulson.
W.A.	Mrs. S. Wilders.
TAS.	Miss J. Clerk.

Journal Editor:	Mrs. D. Craig
Registrar:	Mrs. F. Merrick
Editorial Committee:	Mesdames C. Coulson, P. Dunlop, J. Hunter, Macfarlane, A. McIndoe.
Distribution Manager:	Miss H. Hawkeswood 42 Newcastle Street, Rose Bay, N.S.W. 2029

EDITORIAL

Orthoptics is attention to the individual, in testing and treatment. It is to be alert at once to the physiological purpose, and to the patient's need for reassurance, or encouragement. It is empathy in choice of words and manner. It is flexibility in devising ways of circumventing disablements. It is to question tradition unless verified by experience.

Orthoptics is sensitive collaboration with the ophthalmologist, and with colleagues in other disciplines. Going further, it is awareness of the breadth of ophthalmology, from clinical and surgical skills to nationwide planning for blindness prevention.

Most of these facets of our profession are reflected in the contributions to this journal, which support we believe, its essentially educational function. We are happy to include a paper by Helen Hawkeswood, immediate past president of our Association. As often before, she gives us the benefit of her clinical experience, fortified by systematic review of past cases. So do most of our contributors. We are grateful to them because this is the easy way to increase our expertise, picking up tricks of the trade from those who have learned them the hard way. We are grateful too for the concluding papers by ophthalmologists whose breadth of view properly balances our clinical preoccupations.

These papers are the outcome of our May meeting in Singapore, with the theme "The Eyes of Three Cultures". Ahead in 1979 is the Fourth International Orthoptic Congress in Berne from 3rd to 6th September. Theme: Research and Practice. General Secretary: Ursula Altmann, Himmeriweg 8, CH-8052 Zurich, Switzerland. A month later the Australian Orthoptic Association's general and scientific meetings will be held from 8th to 12th October in Sydney. Conference secretary: Ann Hitch, P.O. Box E47, St. James, N.S.W. 2000.

Diana Craig

NOTES FOR CONTRIBUTORS

Papers, case histories and other communications for publication should be sent at any time of year to the Editor, Mrs. D. Craig, 20 Merton St., Ivanhoe, 3079, Victoria. Manuscripts with two high quality copies should be typewritten in double spacing with wide margins, on one side only of quarto paper. Each paper should be preceded by a brief informative summary of not more than 100 words, stating topic, investigation or observations made, and outcome.

Title page: the title should be as brief as possible. The title page should also give the name(s) of the author(s), one or 2 senior degrees (if any) of each author, and relevant hospital or other institutional affiliations. Include name and address for queries or reprints.

Number each page with author's surname, top R.H. corner.

References should be cited in the text by superior numbers which correspond with a detailed list at the end of the article. Punctuation needs special care.

In a reference to an article in a journal the following information should be given in this order: number as in text, surname of author, initials of author, full title of article, appropriately abbreviated name of journal, year of publication, volume number of first page.

In a reference to the book the following information is required: number, surname of author, initials of author, full title of book, editor (if any), publisher, place of publication, date of publication, page number (if relevant).

In a reference to an abstract, the name of the original publication, together with that in which the abstract appeared, should be given with full data of each.

Abbreviations used for titles of journals are those given in the book "World Medical Periodicals", published by the World Medical Association. Guides may be obtained from issues of Aust. J. Ophthal. or Med. J. Aust.

Illustrations: Photographs, line drawings, graphs, charts and tracings should be submitted in their original form or as high quality glossy photographic copies. Redundant material should be trimmed away.

All illustrations should be numbered and cited as "Figure . . ." in the text. Each illustration should be lightly marked on the back with name(s) of author(s), the number used in the text, and an arrow indicating the top.

Legends should be type written, double spacing, on a page similar to the main body of the text and not attached to the illustrations.

Tables should be clearly set out on separate sheets, numbered and cited (as "Table ...") in the text, as for figures.

Please state whether you wish to correct proofs.

MANPOWER SURVEYS

Shayne Brown
Orthoptist in Charge
Sydney Eye Hospital
Sir John Young Cresc.
Potts Point N.S.W. 2011

In 1973 the Department of Labour and Immigration on behalf of the N.S.W. College of Paramedical Studies undertook a manpower study to assist in the planning of the Cumberland College of Health Sciences. During the planning stages of Medibank, the Orthoptic Association of Australia was advised on the importance of current manpower information and in 1976 the association commissioned a manpower study, conducted by Mr. Charles Wulff, a management consultant.

Two aspects were highlighted by his study. One was the expansion of orthoptics as a profession, in terms of an actual increase in the number of orthoptists, and the second was the broad spectrum of ocular services which orthoptists deliver to the community.

In the 1973 survey the number of respondents throughout Australia was 116. Of these 62% were practising and 48% were non-practising. In the 1976 survey, with 155 respondents, the percentage of practising orthoptists had risen sharply. 72% were practising and only 28% were non-practising. This is an increase in practising respondents of 38% in 3 years.

	1973	1976	% movement
Respondents	116	155	+ 34%
Practising	52%	72%	+ 38%
Non-practising	48%	28%	- 42%

The reasons for this big increase are twofold. Firstly, orthoptists are staying in the profession longer, which is evident by the upward trend in all age groups of practising orthoptists. The most significant group was the 30-34 year age group where there was a 14% increase in the three years since the 1973 survey. Secondly there has been an increase in the number of orthoptists who have returned to the work force, and this perhaps is a reflection of the general trend of our society.

One other aspect which is apparent when one looks at the non-practising respondents, is that 80% of them would like to return to the work force in a part-time situation. However, a large proportion (or 76%) have not worked for 5 years or more and need refresher courses, which are not available on a regular basis at this time.

The major areas of employment were private practices, hospital out-patient clinics and sponsored practices. Government health departments employ only 5% of practising respondents. 17% of orthoptists work in public hospitals, an increase of 13%. Since the 1976 survey was conducted, the Honorary Medical Staff withdrew their services from some hospital out-patient clinics. Despite this fact, the association is led to believe that the number of orthoptists employed in public hospitals has been maintained, and in some hospitals increased. I feel that this increase must reflect the appreciation of the variety of functions the orthoptist is able to perform in respect to general eye care.

The second significant aspect of the 1976 survey was the expansion of services that orthoptists now perform. The results show orthoptics is a broad industry, as practised by the majority of orthoptists.

This expansion of services means that orthoptists now play a wider, more beneficial and vital role in community eye care.

In the 1976 survey we were asked to identify possible additional services which could or should be offered to the community, and there was a good response. 61% of the numerous services suggested then have already been taken up.

The most significant new area is in the multi-disciplinary approach to patient care. Two good examples are the employment of an orthoptist in the Child Development Unit at the N.S.W. Royal Blind Society, and at the Stroke Clinic at Lidcombe Hospital. At the R.B.S. the orthoptist works closely with the occupational therapists, physiotherapist and social workers to devise suitable vision training programmes. Similarly at Lidcombe, orthoptists work in conjunction with physiotherapists and occupational therapists and assist in the total management of stroke patients. Two papers in this journal highlight the advantage of a multi-disciplinary approach to the patient, and to the members of the team, so that the result is improved patient care.

It is gratifying that so many orthoptists have responded to the surveys, thereby in fact, furthering their own interests. The importance of accurate manpower figures cannot be over emphasized and to date the information from these surveys has been invaluable. It was used in submissions to the Higher Education Board in N.S.W. and the Victorian Institute of Colleges for the application to lengthen the orthoptic courses. The figures were also the basis for a paper on manpower which was presented on behalf of the Orthoptic Association at the Fourth National Health Science Education Conference at Cumberland College in October, 1978.

REFERENCES

LANCE, P.M., 'Orthoptics – Past, Present and Future. 1973-74. Aust. Orthopt. J. 13, 2 - 7.

LANCE, P.M., 'The Education of Orthoptists in N.S.W.' 1975. Aust. Orthopt. J. 14, 17 - 19.

Orthoptic Association of Australia Work Force Study. November, 1976. prepared by Mr. Charles Wulff, management consultant.

Survey of Four Paramedical Professions. Professional and Technical Manpower Study No. 3. Department of Labour and Immigration in association with the N.S.W. College of Paramedical Studies Published by Australian Government Publishing Services, Canberra, 1975, 33 - 40.

ACKNOWLEDGEMENTS

My sincere thanks go to Mrs. Jan Wulff and Mr. Charles Wulff for their help with this paper.

SYMPTOMS AND HETEROPHORIA

Helen Hawkeswood
106/67 Castlereagh Street, Sydney, N.S.W. 2000

A heterophoria when decompensated is characterised by symptoms occurring generally after periods of intense close work or physical fatigue. The effort required in maintaining binocular single vision results in headaches, difficulty in changing focus, loss of accurate stereopsis and photophobia.

Headaches frontal, temporal or occipital, were round in 58% of the 500 cases checked (these cases were taken at random and covered a period of 20 years). The headaches could be severe, in some cases lasting for several days, and most patients associated them with their work. Some were due to sinus infection. Some patients appeared with those frightening large X-ray envelopes.

Difficulty in changing focus was not such a frequent symptom. 26%, mainly students, complained of it.

Loss of accurate stereopsis was noticed by tennis and squash players, golfers and bowlers, and of course teenagers, whose failure to judge the distance of the car ahead has been known to do several hundred dollars worth of damage to a parent's car.

Photophobia, our textbooks tell us, is experienced by exophores, but many esophores complain to me of the same discomfort and, unlike exophores, they seldom get relief from shutting one eye.

When binocular single vision becomes too difficult to maintain, the symptoms change, and the patient complains of diplopia or blurred vision. These two are linked together. If the patient has little or no supression he may notice the print going blurred immediately prior to it slipping in two. Blurred vision could also be due to relaxation of accommodation, in which case the patient would show an accommodation defect on the R.A.F. near point rule.

The orthoptist's job is initiated by routine testing, visual acuity, Worth lights, Maddox rod and wing, cover tests, and finally the test for binocular vision. The results of these tests in conjunction with an accurate history lead one to know what exercises are necessary to get the patient symptom-free.

The general pattern found after testing is a varying amount of suppression and, almost without exception, poor or inadequate convergence.

Routine orthoptic treatment is commenced by eliminating suppression. This is done by two methods, anti-suppression treatment on the synoptophore using graded slides with fine controls, and physiological diplopia taught as a home exercise. The elimination of suppression is the main reason why patients need to be seen regularly and why handout home exercises will generally fail. Once suppression has been eliminated so that diplopia is recognised when convergence fails, convergence training can be commenced.

Convergence training is carried out on a synoptophore and as a home exercise. It can be fascinating and frustrating. Fascinating when you watch the sheer concentrated struggle taking place, just to converge, until the later visit when the idea suddenly appears; and fascinating to see the patient's difficulty in accepting the fact that he must accommodate in order to go on converging. Frustrating when improvement is literally measured a degree at a time. One sees these esophorias with such stiff convergence that sometimes a form of jump convergence is the best treatment.

Those cases which show a marked accommodation defect deserve some mention, because they require a slightly different approach. Emphasis is placed at first on monocular and binocular accommodation using small print from a postcode book pasted on a tongue depressor. Once accommodation improves, so does convergence.

It is the orthoptist's job to realise that full convergence (one pen touching the tip of the nose) cannot be achieved by all patients and one must alter one's standards to suit the individual, his symptoms and his work. For example, accountants, students, draftsmen all need better and easier convergence than a housewife.

Although these cases present perhaps the easiest form of orthoptic treatment, they are a constant challenge because the symptoms are real and often severe. There is the battle of personalities, of the lazy person who feels her ophthalmologist has let her down ("after all a simple pair of glasses would be so much easier"). On the other hand one gets the refreshingly self disciplined person, who, providing she has little suppression, becomes symptom free and orthoptically satisfactory in two visits. Perhaps the most difficult patients are those elderly people who have had a symptom-producing heterophoria for years. They appear at a late age for treatment, at a time perhaps when their active life is slowing down and the need to read or watch television occupies more of each day; because of vast deep suppression and hopeless convergence little can be done to help them.

The treatment of symptoms associated with heterophoria is rewarding, The average length of treatment of the 500 cases reviewed was 6 treatments and 90% were symptom free following orthoptic treatment.

This paper was read at a combined meeting of ophthalmologists and orthoptists arranged by our NSW branch in 1977. I wish to express my thanks to my partner Pat Lance for looking after the percentages, to Maree Brown for listening to varied versions of this paper, and to Shayne Brown for asking me to read it.

MEDICAL THERAPY FOR AMBLYOPIA AFTER OCCLUSION FAILURE

Hector Maclean and Mary Carter
Melbourne University Department of Ophthalmology, and Orthoptic Department,
The Royal Victorian Eye and Ear Hospital.

INTRODUCTION

This paper is an interim report on a prospective trial where ecothiophate is used to stimulate an amblyopic eye and atropine is used to blur the good eye. The treatment has been used where conventional occlusion therapy has failed to give a satisfactory result, or where amblyopia has recurred after such therapy.

PATIENTS AND METHODS

Patients in the trial are either attending the Professorial Unit Out Patient Clinics at the Royal Victorian Eye and Ear Hospital or are private patients of one of us (H.M.). The two criteria for entry into the trials are: (1) failure of previous occlusion therapy (2) return of amblyopia after previous therapy. Visual acuity of 6/9 or less for distance, and any level of near acuity are acceptable. No restrictions are placed on the type of squint, the type of fixation, the age of the child, or the degree of ametropia provided it is fully corrected. Nystagmus, whether latent or congenital, is also acceptable. From previous experience with the method, six months seemed a reasonable time limit for the therapy. If improvement is still continuing at six months, treatment is continued until there is no more change in vision.

The treatment regime used is one drop of 1% atropine eye drops to the good eye at bedtime, followed by one drop of 0.125% Phospholine Iodide (ecothiophate) to the amblyopic eye. To minimise possible pupil cyst formation, 10% phenylephrine HCl was given to the amblyopic eye in addition.

For analysis of the results, criteria of improvement were defined as –

Success : Improved at least two Snellen lines.

Doubtful : Improved one or one and a half Snellen lines or 2 N grades only.

Unchanged : Less than one Snellen line or 2 N grades improvement.

Worse : Any degree of worsening.

The youngest child in this series was aged 2¼ and the eldest 12¼. Length of treatment varied from 2 months to 11 months. Results have been analysed on 34 patients; 11 others are continuing treatment.

Each child was seen by both orthoptist and ophthalmologist at approximately 6 week intervals. A full orthoptic examination was done. The ophthalmic examination included slitlamp checking for possible cyst formation and check on the level of induced myopia. Any other side effects were recorded.

RESULTS

SUCCESS	16
DOUBTFUL	4
UNCHANGED	9
WORSE	0
ABANDONED TRIAL	5
	34
CURRENT	11
	45

Sixteen children were classified as being improved. In some, the maximum improvement was not obvious until they had stopped their drops. This was not related to miotic-induced myopia. Their average age was 8½. This group includes a child of 2¼ who showed reversal of her squint at 6 weeks, and a boy of 11¼ whose vision had remained at an obstinate 6/18 through four years of conventional occlusion therapy, and who achieved 6/5 and N 5 vision after nine months of the drop therapy. One patient was allergic to atropine, and hyoscine ¼% had to be substituted. One child hated the drops much more than wearing a patch. All the others said they preferred the drops to wearing a patch.

FINAL VISUAL ACUITY

	6/9+	6/12^{pt} – 6/9	6/24^{pt} – 6/18	6/36 or less
SUCCESS	11	4	1	0
DOUBTFUL	1	3	0	0
UNCHANGED	2	4	2	1
ABANDONED TRIAL	0	1	3	1

The four children in the "doubtful" group are too few for worthwhile analysis.

The nine failures are a slightly younger group with an average age of 7¼. The range of ages was as for the successful group and the durations of treatment were similar.

Five children stopped too early for any assessment to be made.

One child went overseas. Another's drops were stopped by his parents because he had a sore throat and running nose five days after starting. A previous non-wearer of occlusion, he then decided to tolerate a patch and eventually improved to 6/6. Two girls of 9 declined to allow their parents to instil drops. The fifth child was a boy of 5 who had had a Gunderson flap pulled across his right cornea in infancy because of imminent perforation from an abscess. The miotics closed his pupil in behind the remaining area of flap.

Factors which it was felt might influence results have been analysed in relation to the treatment results, and these are tabulated (Tables 2-6).

ATROPINE VA SUPERIOR TO AMBLYOPIC VA

SUCCESS	10 of 16
DOUBTFUL	1 of 4
UNCHANGED	6 of 9

NEAR VA – ATROPINISED EYE

	N48 – N24	N18 – N10	N8 – N5	?
SUCCESS	3	6	4	3
DOUBTFUL	3	1	0	0
UNCHANGED	4	1	2	2

DISCUSSION

The outcome of the trial so far is regarded as exciting, both in terms of the number of results where other treatments of amblyopia have failed, and also the older than average age of the children in the series. The reason for the method working is not understood – it certainly does not relate to the use of atropine alone. Half the children in each results group had previously failed to respond to the use of atropine alone. It does not relate to the degree of blurring produced by atropine. Tables 3 and 4 show there are no differences in either near or distance visual acuity of the atropinised eyes in any of the results groups. A vision of 6/4 and N 5 in an atropinised eye proved no bar to a 9 year old improving from 6/18 and N 18 to 6/5 and N 5 in his amblyopic eye after six months on the drops.

PREVIOUS FAILED ATROPINE OCCLUSION

SUCCESS	8 of 16
DOUBTFUL	1 of 4
UNCHANGED	5 of 9
DROP OUT	1 of 5

We found the use of single letter optotypes useful in the older children for forecasting how far vision could be expected to come on a conventional Snellen chart, and thereby how long drop therapy should be continued. The appearance of pupil cysts was occasionally worrying in this regard, but in no child did they progress to the point where therapy had to be stopped.

INITIAL FIXATION

	SUCCESS	DOUBTFUL	UNCHANGED
FOVEAL STEADY	0	3	4
FOVEAL UNSTEADY	12 (2 NYSTAGMUS)	0	1
JUXTA FOVEAL	1	1	1
ECCENTRIC	1	0	2
NOT RECORDED	2	0	1

ECCENTRIC FIXATION

Table 6 suggests that an abnormal fixation pattern may be a prime indication for this type of therapy.

SIDE EFFECTS

	SUCCESS	DOUBTFUL	UNCHANGED
NONE	9	2	5
STINGING	1	2	2
IRIS CYSTS	6	1	1
BEHAVIOUR PROBLEM	1	0	2
ATROPINE ALLERGY	1	0	0

SIDE EFFECTS (Table 7)

For a three month period in the middle of the trial no supplies of phenylephrine eye drops were available. Three children developed cysts during this period but these regressed when the phenylephrine was restarted. We have found it important to specify the use of Isopto Phenylephrine eye drops. Only two children have reported any stinging from them, whereas we found the use of other varieties of phenylephrine hydrochloride to be universally associated with complaints of stinging. It is noteworthy that two children found that atropine stung, and tw others found that Phospholine Iodide stung.

The three children listed as having behaviour disorders, included one hyperactive child who became even more hyperactive while on the drops, and two – one a success and one a failure – who became very morose and withdrawn at school because of teasing about unequal pupil sizes.

Reservations have to be expressed on the grounds of the cost. The drops themselves are only a small part of this, but the frequent visits necessary for accurate checking represent a high investment in health care. This must however, be set against the risks of leaving a growing child with an amblyopic eye. It has to be remembered that the use of Phospholine Iodide drops can depress the levels of the enzyme pseudocholinesterase in the blood, and a child who requires an operation up to six months after last having the drops may have a prolonged period of apnoea (failure to resume spontaneous breathing) if the anaesthetist is not aware of this possible complication.

Six children had a second course of drops. Four had done well on the first course but vision had dropped back again – all have picked up, and only their greater improvement is included in the analysis figures. One had been inadvertently stopped too quickly and the sixth was an exotrope who improved two lines on his first course of three months but managed only a further line when it was decided to improve him further – his second course lasted six months at the age of 10. Only his first course is analysed.

Like others who have reported the use of this treatment, we have been impressed by the early change in fixation pattern before visual acuity changes are evident, by its ready acceptance by the children, by its usefulness in the older children and, by the lack of any correlation of length of therapy for success or failure with the age of the child, or with any other parameter we have analysed.

CONCLUSION

An interim report on a prospective trial on the use of Phospholine Iodide to an amblyopic eye and atropine to the dominant eye shows that of 29 children successfully completing a course of therapy, 16 have been significantly improved, four doubtfully improved, and nine unimproved. Treatment periods ranged from two to eleven months and patient ages from two years to twelve years. No factors have been found which influence the outcome of a course of this therapy.

REFERENCES

1. *JOHNSON, D.S., and ANTUNA, J. (1965). Atropine and Miotics for the Treatment of Amblyopia. American Journal of Ophthalmology, 60, 889-891.*

2. *JOHNSON, D.S., LIMPAECHER, E., POLLOCK, H.M., CRECO, J.J. and RODIER, D. (1976). Pharmacologic Penalization of Pharmacologic Orthoptics – Acta 22nd Concilium Ophthalmologicum, Paris 1974, 2, 892-895.*

3. *POPE, L.J. (1971). Treatment of eccentric fixation without occlusion. British Orthoptic Journal, 28, 77.*

LEGENDS

TABLE 1. Results

TABLE 2. Final Visual Acuity

TABLE 3. Atropine VA Superior to Amblyopic VA

TABLE 4. Near VA – Atopinised eye

TABLE 5. Previous failed Atropine Occlusion

TABLE 6. Initial Fixation

TABLE 7. Side Effects

THE VALUE OF TESTING BINOCULAR VISUAL ACUITY IN INTERMITTENT EXOTROPIA

Ann Hitch,
Submitted May 1976

Exotropia, constant or intermittent, is a frequent occurrence in Australia, and seems more common in the nothern areas. Fusional control of such deviations may produce symptoms which cannot always be relieved by conventional orthoptics. It is important to investigate the role of accommodation in this respect. If excessive accommodation is involved in controlling the deviation, the symptoms become dramatic; there may be a risk of ciliary spasm developing, and orthoptic treatment becomes more difficult.

In my experience this accommodation feature is not a common problem with exotropic deviations. It should be treated as a separate entity if it occurs. Assessment of binocular vision acuity at 6 metres is therefore essential prior to any treatment. The following case study illustrates the problem.

Mr. P., aged 29 years, presented with self-diagnosed "tension headaches", and concern about the cosmesis of a large intermittent divergent squint. He was anxious to undergo surgery as soon as possible.

Refraction	:	R $\frac{-0.50}{+0.50 \times 180^\circ}$, L +0.50.	Nil ordered
Visual acuity	:	R 6/6, L 6/6	Both eyes fixing 6/60
Cover tests	:	I.D.S. 40$^\Delta$. No diplopia, but heteronymous diplopia under stimulation.	
Convergence	:	held to 8 cm with much effort.	

There was a small fusional range from the angle of deviation. The patient was advised to practice full versions in 8 positions of gaze, and to stimulate convergence on a detailed target, pre-operatively.

Surgery (1): bilateral lateral rectus recession 7 mm. Post-operative findings:

Cover test at 6 metres	:	I.D.S. 22$^\Delta$ This later regressed, to 40$^\Delta$
Binocular visual acuity		unchanged
AC/A ratio	:	attempted measurement unsuccessful

As observed during casual discussion, the intermittent exotropia persisted, and the intermittent recovery of binocular fixation seemed a habitual, mechanical action, unrelated to any visual stimulus such as diplopia or clear vision. The patient had no awareness of these motor adjustments.

In preparation for further surgery, orthoptics seemed necessary to eliminate deep central suppression and to re-establish a stable state of binocular single vision with a normal accommodation and convergence relationship.

To treat the patient conventionally by stimulating recognition of diplopia might well increase the excess accommodation "spasm". To use concave lenses as described by Merrick offered too great a risk of creating a true ciliary spasm. It was decided therefore to neutralise the deviation, or a part thereof, with Fresnel prisms, and gradually to reduce the strength, while maintaining single binocular vision with acuity of 6/6.

The minimum prism strength which clinically achieved this end was 10$^\Delta$ base in, divided between the lenses which were now ordered for the small refractive error. The patient was seen at 3-weekly intervals, and on each occasion it was found that he could accept a 2$^\Delta$ reduction of prism without losing 6/6 bifoveal acuity. At the 5th visit the patient, who had now worn a 4$^\Delta$ prism for three weeks, could control the deviation and maintain bifoveal visual acuity of 6/6, but only with great effort. Convergence and accommodation were just within normal limits. The patient asked for more surgery.

Surgery (2): Resection left medial rectus 5 mm. Bilateral free tenotomy of inferior oblique muscles. (Insertions of both lateral recti were investigated, and found to be at equator).

Postoperative findings:

Cover test	:	8$^\Delta$ exophoria/tropia
Binocular visual acuity	:	6/6. The patient complained that this was blurred vision, and preferred to let eye deviate. Full binocular function at zero angle, with a small fusion amplitude.

He was instructed to wear glasses constantly (without prisms) and to carry out vigorous exercises as before. One week later, there was a marked improvement in clinical control of the latent deviation, and associated accommodation difficulty. The patient reported comfortable, clear vision at all distances. All measurements remained unchanged. This single case study is presented as an example of a rare but considerable problem. With prism therapy the patient learnt to dissociate the accommodation-convergence pattern, thereby producing a more stable condition for surgical correction.

Investigation of binocular visual acuity is a valuable test and should be routinely carried out at the initial examination of all patients with intermittent divergent squint.

My thanks go to Dr. Marlene Strathdee for permission to discuss this case history.

REFERENCES

MERRICK, F. (1974), Aust.Orthopt.J., 13, 17.

FRESNEL PRISMS IN THE MANAGEMENT OF SUNLIGHT DEVIATIONS

Keren Edwards

In Queensland, with constant bright sunlight the year through, intermittent exotropia is particularly likely to become manifest out of doors in spite of surgery and orthoptic treatment. Common features of sunlight deviations are –

- eye rubbing
- marked photophobia
- face pulling to bring down the upper lid
- hand over one eye.

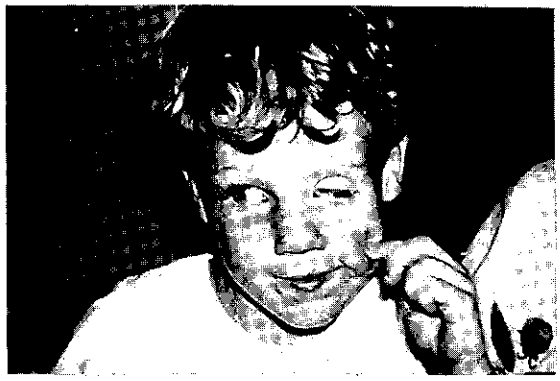


Fig. 1. Face Pulling in sunlight.

Parents and friends show concern about these habits. Adults recall difficulty with ball games, especially tennis. One remembered the sun filtering through a paling fence on the way to school as painful and embarrassing. With hand covering the deviating eye he would pass as quickly as possible to avoid comment from his friends.

Initial examination usually reveals the following:

- poor or no fusional amplitude
- dense supression of the divergent angle
- no diplopia awareness
- on average, a maximum of 20 prism diopters.

The Ravault paper [1] was a welcome answer to my dissatisfaction with our usual methods. In 1974 I treated one patient (who could not have surgery because of anaesthetic risk) with Fresnel prisms along the exact lines described, very successfully. Currently I am treating all suitable patients with Fresnel prisms. Prism correction makes the patient temporarily orthophoric for all normal viewing conditions. Constant binocular stimulation at the divergent angle provides an anti-supression treatment enabling full fusional amplitude to develop.

Method. The maximum outdoors distance prism is placed on the optical correction or plano lenses. The prism can be divided evenly or with the greater power over the fixing eye to aid the amblyopic or suppressing eye. As the larger powers can blur vision to 6/12 or 6/18 a minimal or no correction on one side is more easily tolerated. The division of power between the two eyes may also be assessed with a view to possible future prism reduction (20^{Δ} divided 15/5 allows initial 5^{Δ} reduction). A vertical element can be corrected by using prisms obliquely.

Fig. 2. Right Divergent Squint in Sunlight

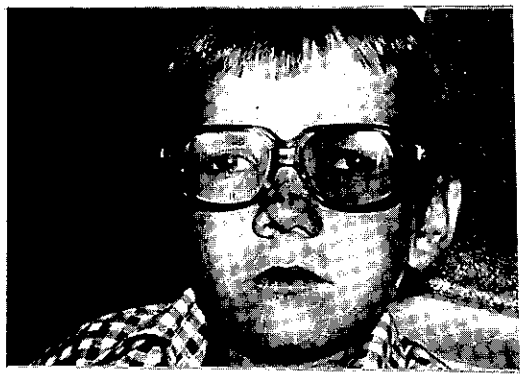


Fig. 3. Prism Orthophoria; 20→ base in divided on plano lenses,

The strength of the prism is reduced at intervals according to control, so that a state of orthophoria is maintained for near and distance while glasses are worn. Intervals of four to six weeks, especially in the early stages are the general pattern. Widely spaced visits are an advantage to families living a long way from the treatment centre. Six months seems to be the average treatment period. Reductions should not be hurried, especially in the early stages, and reversion to a higher power may be necessary at times. Binocular fixation without prisms for all distances is the ideal.

It is important that the lens be cleaned regularly. Contact lens cleaning solutions are effective. Cutting the lens within 1 mm of the border and positioning it under water prevents bubbles.

The following case is an example of management with Fresnel prisms. It is that of a five year old boy (see figures 2 and 3) with right to alternating divergent squint, particularly noticeable when outside in sunlight. There was right photophobia. Clinical examinations:

November 1977

Intermittent RDS manifest at 6 metres and beyond
Prism cover test at 1/3m. : 12^{Δ} exo
6m. : 16^{Δ} exo
over 6m. : 20^{Δ} exo
Fusional amplitude: -4° to $+5^{\circ}$
Prisms 20^{Δ} base in divided ordered on plano lenses.

2 Month Review

Without prisms, intermittent ADS at 6m and beyond, with good control by diplopia recognition.
Fusional amplitude -8° to $+30^{\circ}$
Prisms not altered

4 Month Review

Without prisms, intermittent ADS outside in sunlight, diplopia recognised.
Fusional amplitude: -8° to $+20^{\circ}$
Prisms reduced to 15^{Δ} left eye only,

5 Month Review

Without prism, exophoria to ADS on dissociation at 6 metres and beyond.
Fusional amplitude: -8° to $+25^{\circ}$
Prism reduced to 10 dioptres left eye.

6 Month Review

Exophoria at all distances without glasses
Fusional amplitude: -8° to $+25^{\circ}$
To wear glasses with 10 dioptres left eye when outside only.

8 Month Review

Exophoria at all distances.
To leave glasses off altogether and review in 4 months.

This patient wore the glasses very well throughout the 8 months. He was particularly pleased when the right eye prism was removed.

To date I have thirteen children aged 4 to 10 years wearing Fresnel prisms. The smallest initial correction was 8^{Δ} and the largest 25^{Δ}. The children have worn the prisms quite willingly; only one objected. Three with deviations of 10,20 and 25 prism dioptres have completed treatment, and now have full control of the deviation without surgery. Time will tell whether their gains are permanent. All those under treatment are developing larger fusion amplitudes with less suppression and better subjective control. Photophobia is presenting less of a problem, and the need to shut one eye is lessening.

Adults with firmly established sensorial anomalies are more difficult to treat. They are more disturbed by the reduced visual acuity caused by the higher prism powers, although some after a few weeks of perseverance have suddenly adjusted well.

It appears that prisms may prove successful in overcoming obstacles to binocular vision for which orthoptic therapy and surgical intervention are inadequate. They certainly go far in eliminating suppression and developing a large fusional reserve in these sunlight deviation cases.

There is much more to be learnt about the possibilities of these prisms and the extent to which anatomical features, the convergence/accommodation ratio, density of suppression, and retinal correspondence, affect their efficacy. Collation of our individual experiences could well contribute to a higher success rate all round.

I wish to thank Drs. Fraser, Kelly and Crawford for allowing me to present data from their cases.

REFERENCE:

RAVAULT, A.P., Bongrand, M., & Bonamour, G., The Utilization of Prisms in the Treatment of Divergent Strabismus in Orthoptics. Eds. Mein, Bierlaagh & Brummelkamp-Dons. Excerpta Medica. Amsterdam 1972.

ABSTRACT: The Utilization of Prisms in the Treatment of Divergent Strabismus. Ravault, A. P., Bongrand, M., & Bonamour, G., in Orthoptics. Eds Mein, Bierlaagh, & Brummelkamp-Dons. Excerpta Medica. Amsterdam 1972

Results are reviewed of prismatic treatment of 20 cases of primary divergent strabismus or of divergent strabismus secondary to esotropia or convergence insufficiency. The treatment was used alone, or with surgery or orthoptics. Four illustrative case histories are given.

Points emphasised in the preliminary investigation are refraction under prolonged atropinisation, and measurement of the AC/A ratio (a high ratio is said to indicate good prognosis), estimation of the maximum deviation by prism cover test, and functional examination. Prisms were prescribed for the exact angle of deviation unless there were abnormal binocular tendencies, in which case over-correcting prisms and alternate occlusion were ordered as the first step.

Regular assessment of the patient was made with a gradual reduction of prismatic correction at each visit. It is hoped the patient will ultimately maintain orthophoria without prismatic correction for all distances of fixation. Failing this, surgery is undertaken. Orthoptic therapy, where necessary, is only given after surgery, to avoid risk of convergence spasm. Binocular visual acuity is noted in each case history.

The study shows that a deviation greater than 30^{Δ} does not respond successfully to prism therapy. Abnormal retinal correspondence presents a problem in adults. The authors conclude that prismatic treatment is valuable not only for its ease of use, but also for its efficiency, because "it is a permanent anti-suppression treatment, in physiological conditions of use".

Jenny Hunter

ABSTRACT: Prisms as an Orthoptic Tool in the Management of Primary Exotropia. S. Veronneau-Troutman, S. Shippman, & A. C. Clahane, in Orthoptics, Past, Present and Future, Stratton, New York. 1976.

In this article, thirty seven cases of primary exotropia seen between 1970 and 1973 are reviewed. All had follow-up for at least six months if surgery was involved, and for up to one year if orthoptics only was used.

She describes the prism correction divided equally between the two eyes or placed with the lesser power over the amblyopic or suppressing eye. The average age of the patients treated was between 8 and 9 years and the angle of deviation in all cases was approximately the same. The average duration of treatment was 3.9 months.

The authors found that the prisms improved the convergence amplitude and the type of retinal correspondence and in general they offer more improvement than exercises alone can achieve. They concluded: "Finally, we consider prisms a useful, and even an essential tool in the management of primary exotropia and emphasize the fact that the use of prisms as well as orthoptics in general requires a thorough knowledge of the sensory and motor aspects of the binocular act."

Anne McIndoe

A COMPARISON OF THE TITMUS AND T.N.O. STEREOACUITY TESTS

Neryla Jolly DBO (T)

The purpose of this paper is to compare the Titmus stereoacuity test with the T.N.O. random dot test, and to compare the extent to which scores on each are affected by ocular deviation, visual acuity, and age.

Both tests measure stereoacuity and utilise a book, held at 40 cm from the eyes, combined with special spectacles. Both require co-operation from the patient in indicating the stereoscopically displaced element(s) on each plate.

The tests differ in their methods of differentiating the right and left eye patterns, Titmus using polarisation, and TNO using complementary colours. Another major difference is that Titmus plates have bold contour lines defining each item which is to be compared with another for depth difference, while in TNO the borders of the perceived item are defined by the depth difference itself. The only contours in TNO are the short low contrast ones of individual dots, so similar to surrounding dots that there is little to guide heterophoric eyes into position for fusion.

Additionally, the range of disparities, and the levels tested within the range, differ for the two tests. For the purpose of this study, the stereoacuity required for the Titmus "fly" was estimated at 2400 seconds of arc, and that for the TNO screening pages as approximately 2000 seconds. These being included, the total range for Titmus is from 2400 to 40 seconds, while for TNO it is from 2000 to 15 seconds. Within those ranges, Titmus tests at 10 levels, TNO at 7 levels. The only level common to both is at 60 seconds.

SUBJECTS AND PROCEDURE

Fifty-four orthoptic patients were randomly selected. The age, sensory state, type of deviation, and angle of deviation were then noted.

Each patient was investigated using the commercially supplied Titmus and TNO stereoscopic tests. The order of presentation of the tests varied. Each was carried out as follows:– the patient's most recent optical correction was worn and good illumination was placed above the test book, which was held 40 cm from the eyes and parallel to the patient's face and glasses. The patient was allowed time to adapt to the test, and given encouragement by gentle sideways movement of the test book, and progression to one or two items beyond that on which he first faltered. Results were recorded in seconds of arc, according to the last correct answer.

RESULTS

Overall, of the 54 patients investigated, 45 showed some evidence of stereopsis on the Titmus test, and 37 on the TNO test. Nine showed no perception of depth on either test.

Further breakdown of the figures is shown in the following tables, which examine the relationships of stereoacuity to other variables.

STEREOACUITY AND TYPE OF DEVIATION

Stereoacuity scores on both tests are grouped according to the binocular disorders present, set out in order of increasing severity, namely Convergence Insufficiency, Heterophoria, Intermittent Squint, and Constant Squint. All patients in the first three groups used bifoveal fixation. No bifoveal fixation was recorded for those in the constant squint group, which included some small angle squints, or microsquints.

It will be seen that all convergence insufficinecy patients had fine stereoacuity, appreciating disparities of 60" or less. The proportion of patients with fine stereoacuity is progressively less in the following groups. One exceptional patient in the intermittent squint group had 15" stereoacuity; she had already undergone orthoptic treatment, and gained good control.

Seven patients in the constant group, and two with intermittent squint, showed no stereopsis on either test. The latter two had manifest deviations in the near testing position, due respectively to A exotropia and convergence excess.

These results support the belief that the less the stress or disruption to bifoveal single vision, the better the chance of good stereoacuity, on either test.

DEVIATION ANGLE AND STEREOACUITY

The measurements of deviation (see figure 2) in prism dioptres were made with correcting spectacles worn, and for fixation at 1/3 metre. It can be seen that most acuity scores of 60" and better are associated with exodeviations, and most of the zero scores with esodeviations. Nearly all patients with 40" acuity or better have deviation angles less than five dioptres.

Five patients had a hypertropia or hyperphoria; in four of these stereoacuity was defective.

VISUAL ACUITY AND STEREOACUITY

Stereoacuity is compared with the visual acuity of the worse eye (optical corrections being worn) in figure 3. It can be seen that visual acuity below 6/9 in one eye is associated with stereoacuity below 140 seconds, and that fine stereoacuity is linked with visual acuity of 6/6 or better.

AGE AND STEREOACUITY (Figure 4)

The possibility of reliable responses from patients under the age of 5 years often determines the popularity of a test to be used by orthoptists. Of the patients aged 5 years and younger who indicated any awareness of binocular depth perception, all but one did so on both tests, the youngest being an exceptional 2 year old.

DISTRIBUTION OF SCORE PAIRS

Figure 5 shows the frequency of the score pairs obtained from each patient. The figure in any cell tells how many people gained the particular scores indicated by the line (Titmus) and column (TNO) which cross at this point. The heavy black lines enclose all score pairs which may be accepted as equivalent, in view of the different disparity levels used in each series.

Any score on either test means that the patient's real stereoacuity is at least as high as the score indicates, but is less than the value of the next item beyond it in the series. To take one example, a TNO score of 480" means that the patient has 480" acuity at least, but falls short of 240" acuity. A Titmus score of 400" comes within this range; therefore the cell TNO 480" x Titmus 400" is enclosed in heavy lines. Outside these lines are score pairs which cannot be reconciled in this way.

These points being understood, we may proceed to compare the test results.

COMPARISON OF RESULTS ON TITMUS AND TNO

There is appreciable difference in the score distributions for the two tests. The Titmus distribution (right hand column of fig. 5) has 3 maxima, at zero, 200", and at 40". The TNO distribution (bottom line of table) has two maxima, at zero and at 60". It appears that anyone who passes TNO at the 480" level is apt to go right on to the 60". The Titmus scores are more evenly distributed.

If we ignore four pairs of equivalent, or nearly equivalent, scores near the centre of the table, we have 4 distinct groups to consider. In two, all paired scores show equivalent acuity. The other two groups are of scores consistently higher on one test than on the other.

FIGURE 1

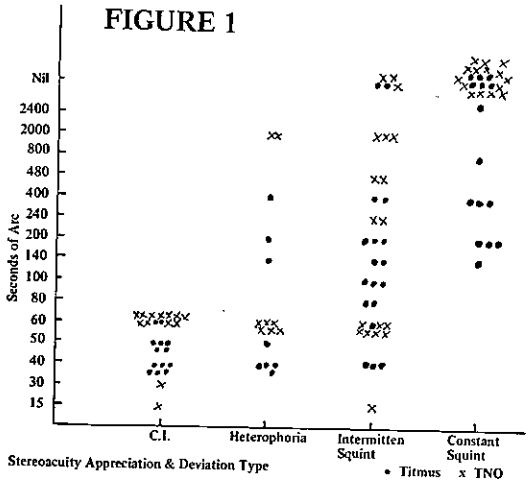
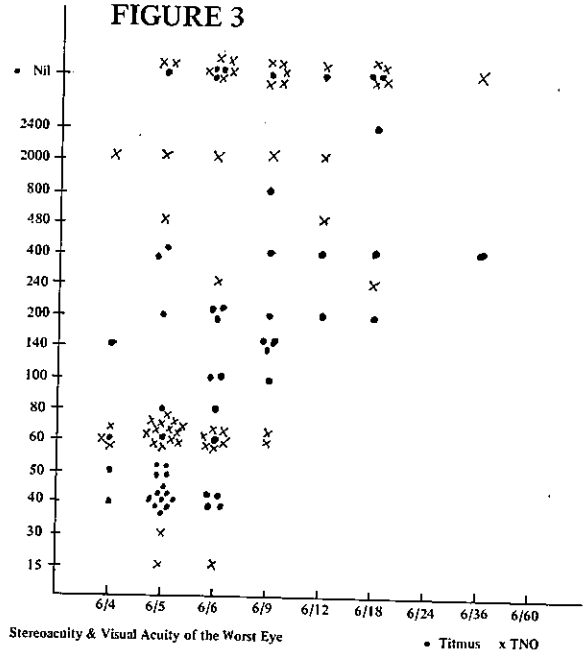


FIGURE 3



ZERO OR MINIMAL ACUITY ON BOTH TESTS

This group of 9 cases is within heavy lines, at the bottom right corner of the table. In all of these, there was manifest squint in the near testing position, 7 being cases of constant squint. Visual acuity of the worse eye ranged from 6/5 to 6/18.

One patient in this group was credited with a pass on the Titmus Fly plate. Walraven (2) claimed that patients responsive to the Fly but failing on the screening plates of the TNO test, could not be proved to be using binocular single vision. This confirms a personal view that without supportive evidence, stereoscopic appreciation of the Fly must generally be viewed with scepticism. Exceptional are the cases of young children who spontaneously reach forward to grasp the wings, with fingers well above the plate. This evidence is convincing enough.

HIGH STEREOACUITY ON BOTH TESTS

The paired scores within heavy lines at top left of the chart record 23 cases with stereoacuity of 60" or better on both tests. They include all cases of convergence insufficiency, five of the eight cases of heterophoria, and four of the seventeen with intermittent squint. All had vision of 6/6 or better in both eyes.

STEREOACUITY HIGHER ON TITMUS THAN ON TNO

This group comprises 16 cases. (See right hand columns above heavy lines in Figure 5.) Nine cases, of which eight were classed as constant squints, showed no stereopsis on TNO tests; the remaining five could manage a TNO screening test (2000"), but no more. All showed stereoscopic ability on the Titmus test, their scores ranging from 800" to 100".

The study of Frisby, Mein, Saye, and Stanworth is relevant here. Patients who demonstrated some response to the Titmus test were selected for the study, which examined their responses to two random dot stereogramms. Both stereograms subtended 12 minutes of arc (720") and were projected in turn on to a screen in front of the patient. A contour outlined the area of disparity in one stereogram; the other had no contour but was otherwise identical.

Frisby's results disclosed that in the presence of fully functional binocular single vision, contours were of no advantage. In patients with reduced binocular function (typically, with microtropia) contours were necessary to enable stereoscopic appreciation, and in patients with more severe binocular defects, contours were again of no advantage. It is at least probable that in the group of cases we are now discussing, the absence of contour lines was the reason for low stereo ability on the TNO tests.

STEREOACUITY HIGHER ON TNO THAN ON TITMUS

The fourth group came as a surprise. Five patients, all scoring at 60" on the TNO series, failed to reach the same level on Titmus. Their Titmus scores ranged from 200" to 80", their ages from 5 to 8 years, and vision of worse eye was 6/6 or better.

Why should these children find the TNO tests easier? Possibilities are that they are helped by the greater area of the disparate elements in the TNO plates, and that the hard-line definition of competing forms on the Titmus plates actually distracts attention from disparity. The emphasis on form definition while a child is learning to read may to some extent inhibit attention to depth. It is not uncommon for a child to give up at the 100" or 80" level on Titmus, and then to complete the test accurately when asked to "guess" the rest.

CONCLUSION

The Titmus and TNO stereotests are both effective measures of stereoacuity where binocular single vision is well established.

The Titmus test will allow stereo appreciation to be recognised if any is present, regardless of the patient's maturity, deviation type or angle, and visual acuity. It is fairly discriminating in the middle range of disparities, providing a means of demonstrating improvement under treatment.

The TNO may be used with advantage to screen visual defects, being effective where bifoveal single vision is used. Its range extends to higher stereoacuity levels than does the Titmus series.

FIGURE 2

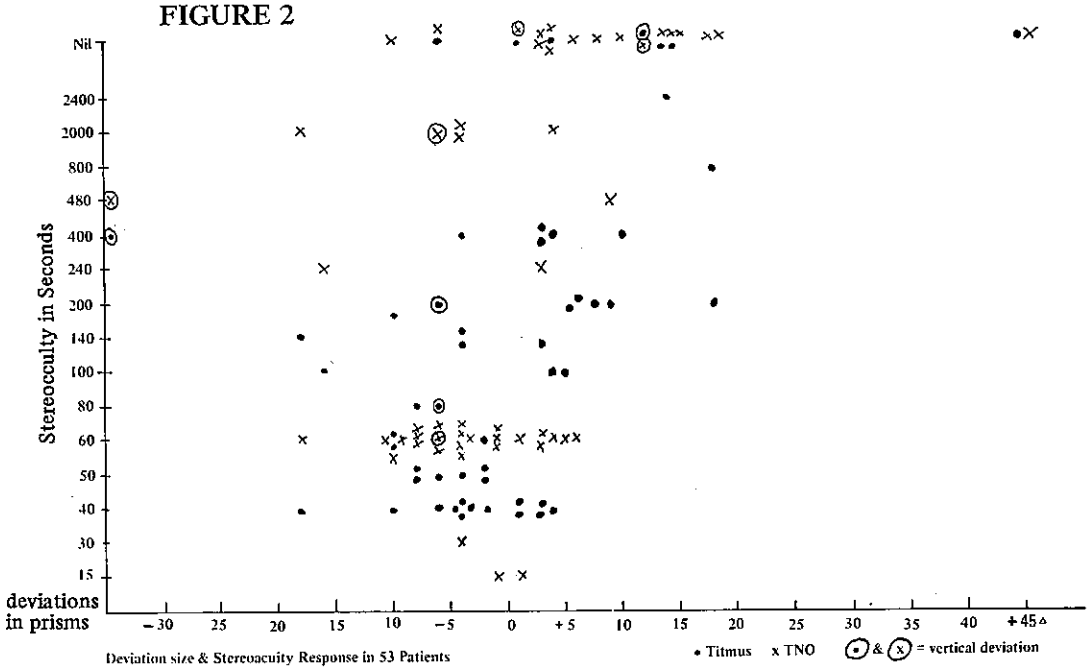
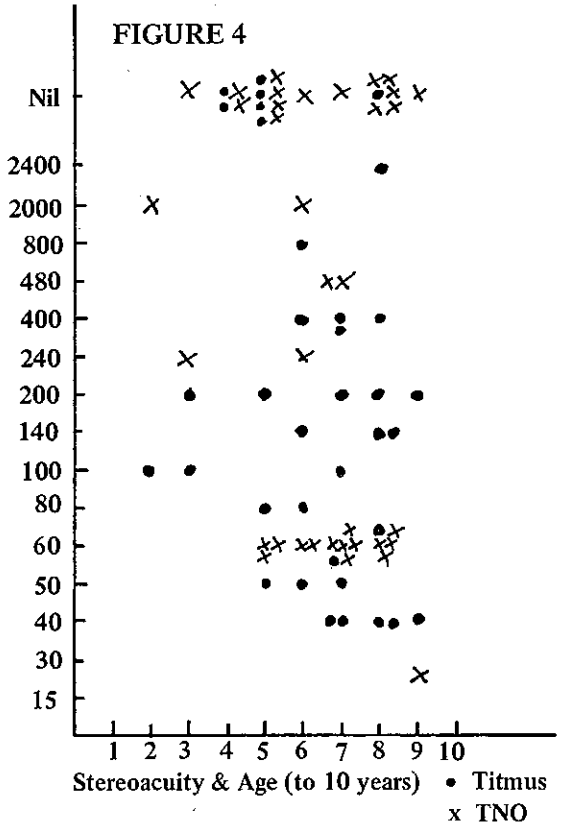


FIGURE 4



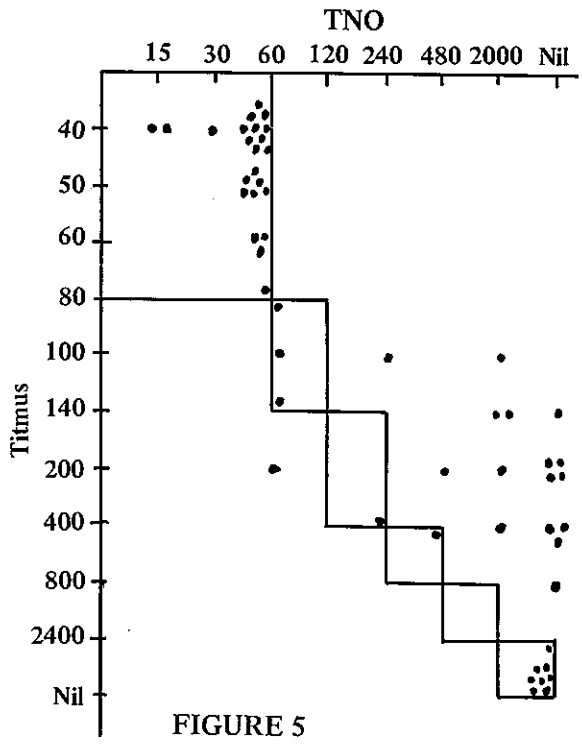


FIGURE 5

REFERENCE

Frisby, J. P., Mein, J., Saye, A., & Stanworth, A. (1975: Brit.J.Ophthal., 59, 545.

Walraven, Frances (1975): Amer. Med. J., vol. 8, no. 5, 873.

STEREOACUITY IN KINDERGARTEN CHILDREN

Jane Pardey
Sydney Eye Hospital

'Stereoscopic vision' , said Hermann Von Helmholtz in 1857, 'is the necessary foundation for all our actions, from threading a needle . . . to leaping from cliff to cliff when life itself depends on the right measurement of distance.' Early records show an interest in the phenomenon by Euclid in the fourth century B.C., Leonardo da Vinci, and Oliver Wendell Holmes in the nineteenth century.[1]

The physiology and neurophysiology of stereopsis has been widely discussed over recent years. The aim of this paper is not to look at these aspects of stereopsis but to look briefly at the gross features in a school screening survey of over 5,000 kindergarten children.

Stereoscopic vision, according to Lyle and Wybar, is 'the ability to fuse with the appreciation of depth, similar images falling on points of the retinae which are slightly disparate laterally.'[2] Steroacuity is the smallest amount of horizontal retinal image disparity that gives rise to a sensation of relative depth.[3] Normal stereoacuity according to Ogle and Parks is 40 seconds of arc. Romano, Romano and Puklin[3] , Brown and Jones[4] and Dunlop[5] have all described an Age-Stereoacuity link.

The statistics used for evaluation were provided by the Division of Health Services Research, Health Commission of New South Wales. The personnel, sample and tests performed have been described by Brown and Jones[4] .

RESULTS AND DISCUSSION

The scores were determined on the Titmus (R.) Test, and have been divided into the following groups for comment.

Key to Stereoacuity Levels

	Test Level	Stereoactuity level
Group 1 	No Score.	
	Wirt Fly	At least 1000 " of arc
Group 2 	Circle 1	800 " of arc
	Circle 2	400 " of arc
	Animal A	400 " of arc
	Circle 3	200 " of arc
	Animal B	200 " of arc
Group 3 	Circle 4	140 " of arc
	Circle 5	100 " of arc
	Animal C	100 " of arc
	Circle 6	80 " of arc
Group 4 	Circle 7	60 " of arc
	Circle 8	50 " of arc
	Circle 9	40 " of arc

Figure 2 shows the range of scores. It can be seen that nearly 60% of the children scored in Group 4 (60" of arc or better). In fact, 31.8% of the children scored 40" of arc. This compares favourably with the findings of Romano, Romano and Puklin.[3]

FIGURE 2

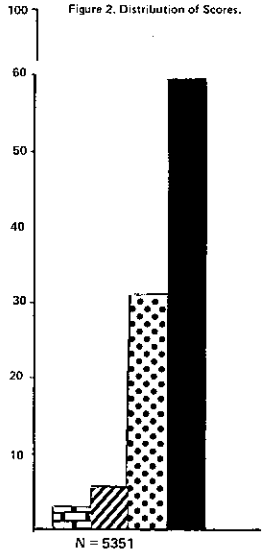


FIGURE 3

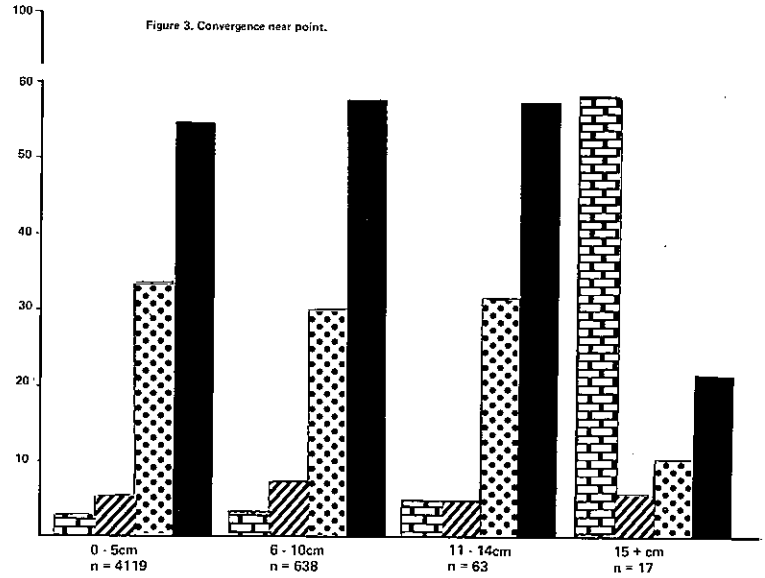


Figure 3 shows the relationship between the convergence near point and stereoacuity. More than half of those whose convergence near point was 15cm or more remote showed stereopsis on the Wirt Fly only, or none at all. These were presumably children with manifest strabismus for near vision. The figures indicate that for all who converge past 15 cm, whether to 14 cm, 5 cm, or anywhere between, the probability of good stereoacuity is equally high.

Figure 4 compares the stereacuities of children with and without defective ocular movements. The large number of group 1 and group 2 scores among those with defective movement are probably related to loss of bifoveal fixation in the primary position.

FIGURE 4

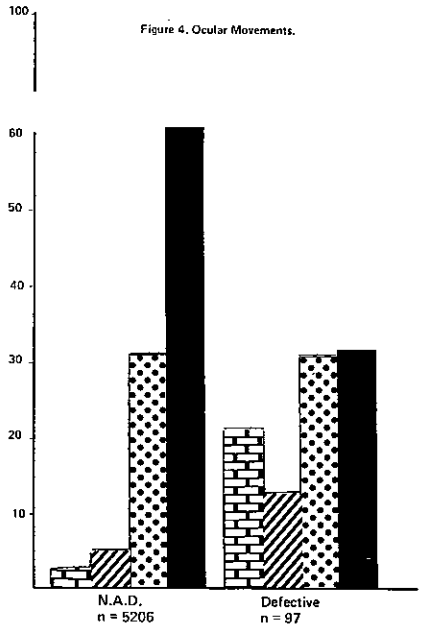
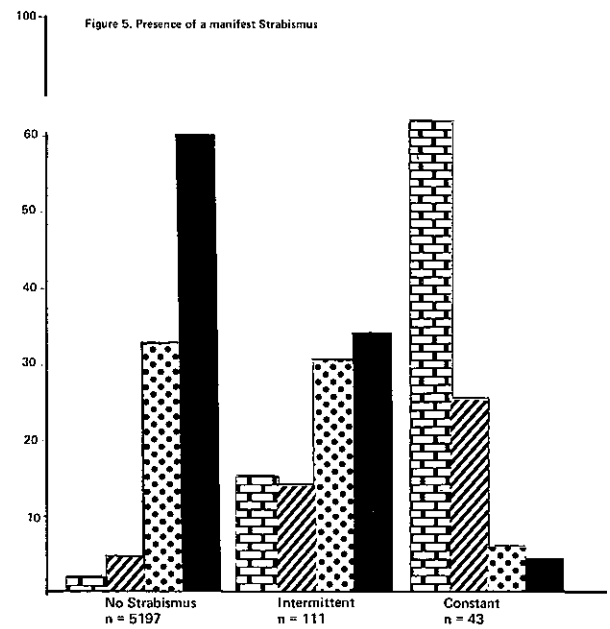


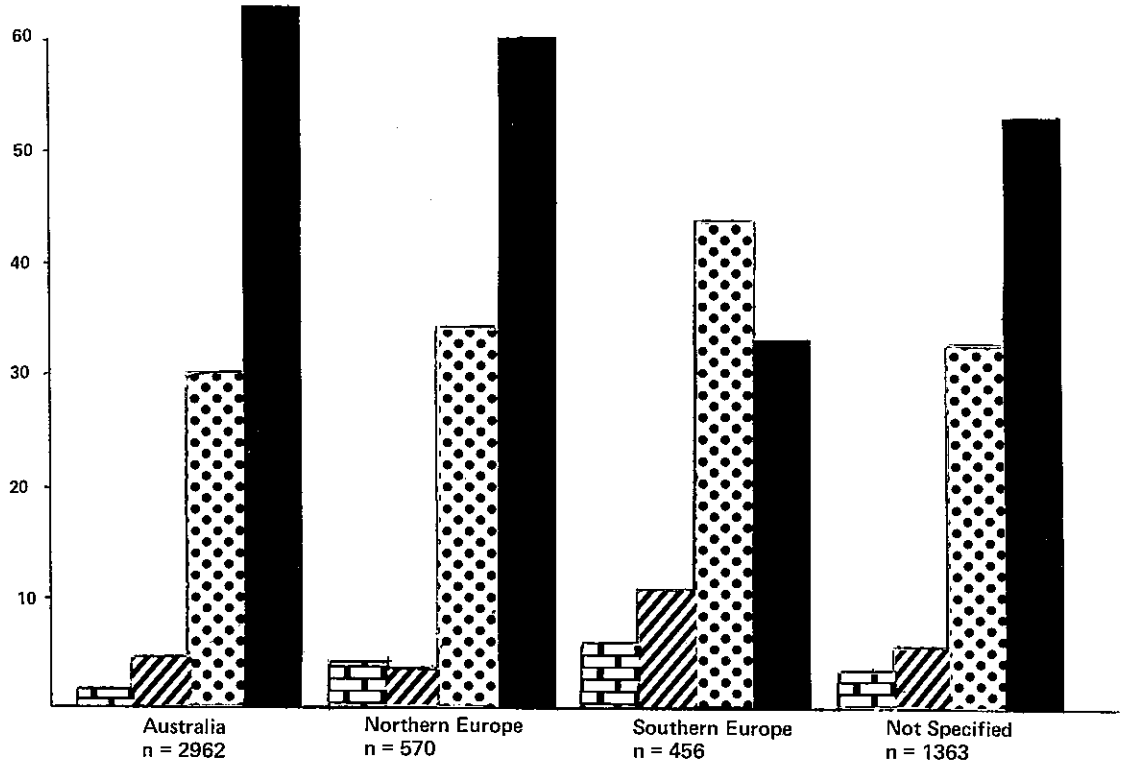
FIGURE 5



As might have been imagined, presence and constancy of manifest squint have considerable effect on steroacuity (Figure 5). A number of microsquints were included in the figures for constant deviations, in the survey. The 8 constant cases with stereoacuity of 140" or better are probably microsquints.

As regards the stereoacuity patterns among children grouped according to birthplace of parents, the most obvious group is the European one (Figure 6). It may be that the children concerned are culturally less equipped to deal with the English language and/or with the individual testing situation.

FIGURE 6 – Birthplace of Parents



CONCLUSIONS

Although most of these findings are in line with popular orthoptic thoughts on stereoacuity, there are perhaps areas where more investigations may prove interesting; for example a more detailed look at the effects of convergence on stereoacuity, and the difficulties of migrant children in test situations.

ACKNOWLEDGMENT

The author would like to thank Shayne Brown for all her generous help.

REFERENCES

1. *MUELLER, C., RUDOLF, M., et al, Light and Vision, Nederland, Time-Life International, 1967*
2. *LYLE & WYBAR, Practical Orthoptics in the Treatment of Squint. 5th edition. 1967. p. 621.*
3. *ROMANO, P.E. et al, "The Development of Normal Binocular Single Vision in Children". Orthoptics, Past Present and future. ed. Moore, Mein & Stockbridge, Stratton, N. York, (1976). p. 11.*
4. *BROWN, S., and JONES, D. "A Survey of the Incidence of Defective Vision and Strabismus in Kindergarten Age Children – Sydney, 1976". Australian Orthopt. J. Vol. 15, 1977 p. 24.*
5. *DUNLOP, P. "Visual Development of 148 Pre-School Children over a Period of 3 Years". Australian Orthopt. J. Vol. 15. 1977. p. 19.*

PURPOSEFUL PERIMETRY

Belinda Paul
Prince of Wales Hospital

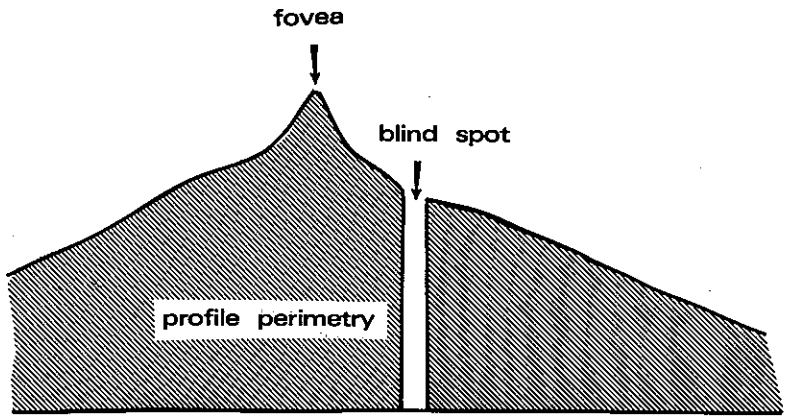
The plotting of visual fields has now become part of the routine work of many Orthoptists. This paper will outline the method currently in use at The Prince of Wales Hospital Eye Clinic, using the Goldmann Perimeter which we feel provides the most accurate, reproduceable visual field in glaucoma management.

Harrington describes the visual field as "that portion in space in which objects are simultaneously visible to the steadily fixating eye". "Objects act to stimulate the various portions of the retina and, through the conducting nerve fibre bundles of the visual pathway, the visual cortex in the calcarine areas of the occipital lobes."

The cortical response to the patient's awareness of these stimuli depend upon

1. their proximity to the visual axis
2. their size
3. their brightness
4. movement
5. the integrity of the receptor of the stimulus and the visual pathway.

"Retinal sensitivity is greatest in the foveal area and decreases in proportion to the distance of the rods and cones from the macula" and so does the visual acuity within the visual field. One is thus able to imagine Traquair's comparison of the visual field to "an island of vision surrounded by a sea of blindness' (figure 1). The peak is the fovea and the coastline the periphery. Objects near the fovea are easily seen but only become visible near the periphery if they increase in size and intensity, becoming larger, brighter or moving.



Characteristic changes in the visual field make it possible to localise within the visual pathway the site of the lesion that has produced the changes, and thus determine diagnosis, prognosis and possible therapy.

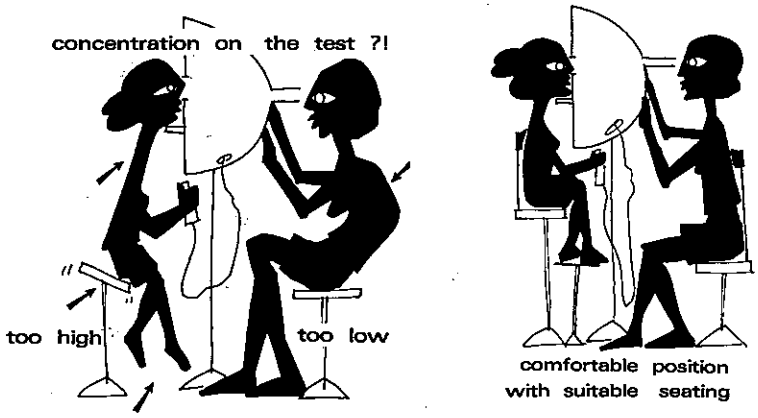
Harrington states the purpose of perimetry is "to detect these revealing defects in the visual field, to measure them quantitatively in terms of visual acuity, and to chart them as normal or abnormal contour lines or isopters".

A patient's neurological or ocular future may rest in our hands and we need to approach perimetry with due responsibility, and with enthusiasm. As we are trained to obtain detailed subjective and objective data, at times from very uncooperative patients this training should enable us to become well equipped to perform perimetry.

At the Prince of Wales Hospital Eye Clinic we have found it particularly helpful to follow a visual field screening technique (as adapted from Armaly and Rock, Drance and Morgan), using the Goldmann Perimeter. The Goldmann is constructed from a half shell in which the test object is projected and where the eye is at the centre of rotation of a hemisphere that has a radius of curvature of 33cms. Visual fields must be tested in a quiet area and illumination should also be constant. We have a rheostat which allows us to dim the lights evenly and the walls and floor of our room are black. One should not have a lamp on in the room even though this may be time saving as it is distracting for the patient. The calibration of the Goldmann must be done routinely.

For simplicity I have outlined our technique in ten steps:

1. Explanation is terribly important. Fixation of the central target must be stressed. Some patients require the examiner to remind them to re-fixate after each sighting of the target because they involuntarily fix the target.
2. Relaxation can and should be happening while you are explaining the test. The stressed patient will perform the test poorly as concentration will be reduced. It would be better to perform the test another day if the patient is particularly tense. The rapport between patient and examiner is built at this time and is important to ensure maximum results. Comfort is also necessary for concentration as seen in figures 2 and 3.



3. Refraction should be recent and the lenses used should include a near correction if testing with the Goldmann perimeter. A lens holder is fitted in front of the chin rest and the appropriate correction added using thin rimmed lenses. If the patients refraction is not known an approximate correction can be found by trial and error.

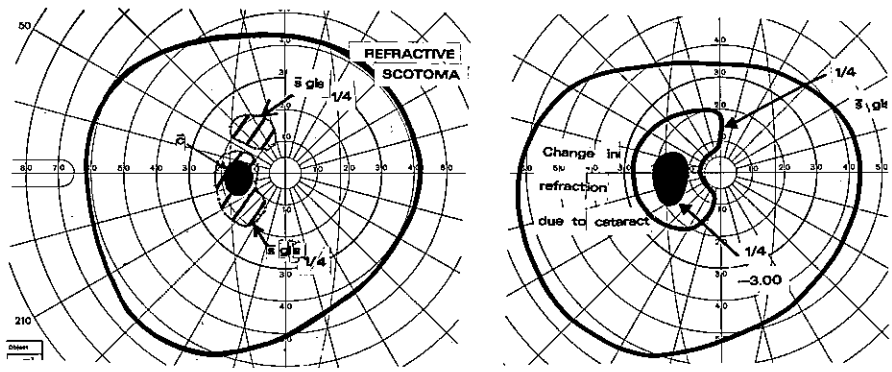


Figure 4 shows a typical refractive scotoma. These can be very similar to relative glaucomatous arcuate scotomata.

Figure 5 shows a refractive scotoma of a patient who became myopic (-6.00 D) in six months because of progressive cataracts. Previously she had been emmetropic. With the appropriate corrections the scotomata could not be reproduced. Any central defects can be refractive and lenses should be checked, cleaned and at the correct distance from the eyes. You should try stronger or weaker lenses if you suspect a refractive scotoma.

4. Threshold (see figure 6 No. 4) is found by presenting the target at 25° from fixation starting with 1/2 (1 = size, 2 = intensity) and increasing intensity and then size until seen by the patient, i.e. 1/2, 1/3, 1/4, 11/4, 111/4, 1V/4, V/4. Obviously a relative scotoma may be missed if the target is greater than threshold.

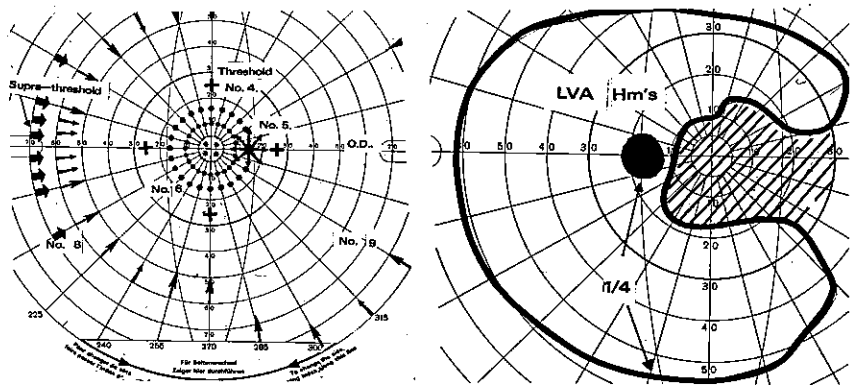


Figure 6 shows our procedure for glaucoma screening. We also check other areas of the visual field randomly. We adapt this procedure for neurological testing eliminating the supra-threshold target and using a red target as well as white. Visual loss to red occurs earlier in the course of neurological

disease than visual loss to white. The usual threshold used is 111/4 red. With glaucoma we are particularly concerned with changes on the horizontal meridian and within the central area, whereas with neurological fields the concern is for changes at the vertical meridian particularly when looking for disease behind the chiasma.

Figure 7 demonstrates how a visual field can be obtained with a low threshold target even with poor central vision. This patient had an anterior ischaemic optic neuropathy of the left eye with hand movements vision. We used the projector (Goldmann) which utilizes the fixation of the other eye to align the pupil of the eye being tested.

5. **The Blind Spot** (see figure 6 No. 5) is tested first starting at 15° temporal to fixation along the horizontal meridian and moving from blind to seeing in at least eight directions. Any defect at this stage can alert the examiner to recheck some of the possible causes. Failure to demonstrate a blind spot is a good indication of error on the part of examiner or patient. I have not only found a blind spot on the nasal field but I have also found one when the occluder was not on! Obviously occlusion and fixation must be checked.

6. **Seventy six Points** (see figure 6 No. 6) are marked in the central area and these should all be "spotted" using static (stationary) perimetry allowing one second for each presentation of the target. It is important to use static perimetry here as it detects relative scotomata whereas kinetic (moving) perimetry may not. It is important not to move (wiggle) the target. A scotoma here may be the keystone in the glaucoma patient's therapy. Roenne's nasal step is usually associated with a small paracentral scotoma and this may be missed if the central area is not thoroughly checked.

7. The refraction correction is removed. This can be a rest and reassurance period for the patient.

8. **The Nasal Isopter** (see figure 6 No. 8) is plotted, paying particular attention to 15° above and below the horizontal meridian. A supra-threshold target (one with increased intensity or size) should be used to check for nasal step in glaucoma screening.

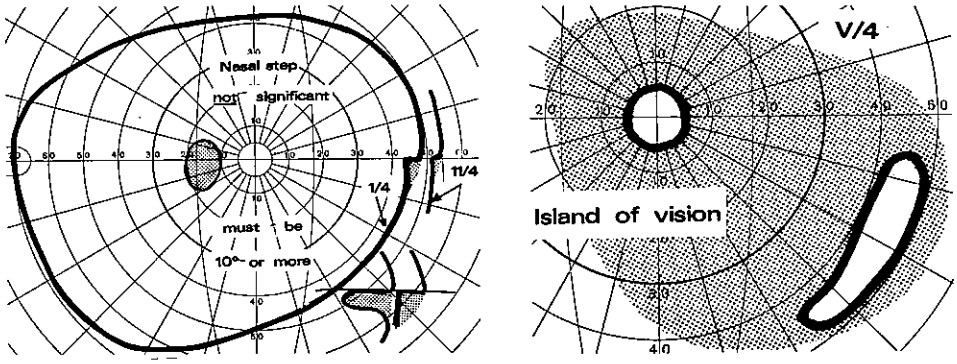


Figure 8 shows a nasal step which is not significant even though both the threshold and supra-threshold targets suggest it. The step must be 10° or more horizontally to be significant and you may find that in testing for nasal steps it is difficult to find a definite scotoma. A nasal step found should precipitate a more extensive search of the central field.

9. **The Temporal Isopter** (see figure 6 No. 8) is tested.

Figure 9 shows typical end stage chronic simple glaucoma using the brightest, largest target (V/4) This is referred to as the island of vision and one can see that the temporal field or island could be missed if this area was not checked thoroughly.

10. **Abnormal Field** is noted and one should note any particular methods used to detect scotomata – whether they are vague suggestions of scotoma, or relative or absolute. A comment on the patient's fixation and status while performing the test should be made especially if you felt this interfered with the results of the field.

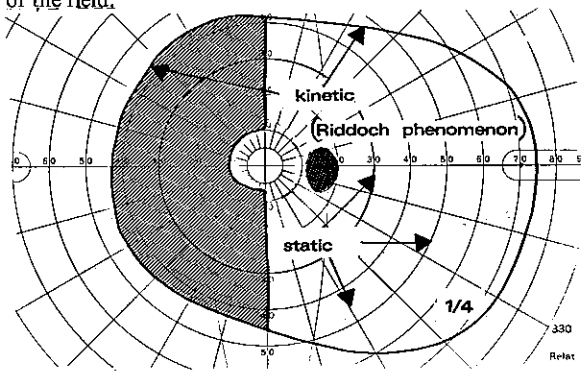


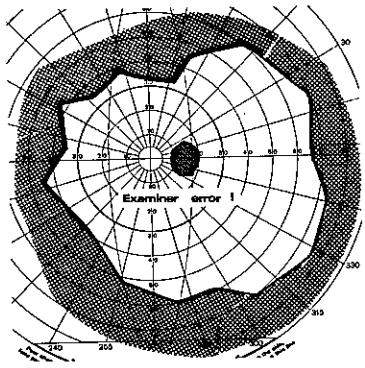
Figure 10 is a field of a patient with a lesion of the striate cortex of the occipital lobe. He had a complete homonymous hemianopia with static testing but showed a normal peripheral isopter with kinetic testing. This man could see only moving objects on his "blind" side.

At our clinic we use the Goldmann perimeter for screening because we feel it is best suited for use by multiple examiners and as several artifacts can be eliminated.

1. The fixation distance is set and the patient's head can be strapped in, whereas our Bjerrum screen relies on positioning of the patient without the help of a chin rest.
2. The patient's fixation can be watched through the telescope.
3. The target can be static or kinetic.
4. The patient cannot anticipate the target or its position.
5. The size and colour of the target can easily be changed without any noise or knowledge of the patient.
6. The diameter of the pupil can be measured through the telescope and recorded routinely. This is important if there is change in contour or size of the visual field and it may be because the pupil has changed in diameter. A miotic pupil produces a smaller field than a dilated pupil.
7. The central and peripheral field is tested routinely.
8. Last but not least the examiner may sit comfortably for the test.

We use the Bjerrum screen when we feel it will best suit the patient or if the patient has a known central scotoma. Confrontation test or any visual field recording is more helpful than the words "could not perform test". Some visual fields recorded have been done solely on the eye movements made to take up fixation and this is also helpful to remember when testing a malingerer. They will involuntarily fix peripheral targets if the examiner persists in "testing", for example, moving from the central area to peripheral area in spotting or changing colour of the target.

Techniques must be varied for each individual patient. The perimetrist must be aware of the limitations of testing and should be ready to adapt techniques to suit each patient. It is easier however if a format is applied initially, particularly as in most cases the patient may be tested the next time by another examiner. There are many subjective and objective artifacts possible so that on the same day the same patient may not respond to the same examiner in the exact same way. Not only should visual fields be reproducible it is our responsibility to perform them purposely. Knowing how a field should look is important. My first field resembled figure 11. Obviously my speed must have varied and I did not know to check any dips in the peripheral contour. If you imagine the outline of the island of vision, one can see that the isopter plotted should be symmetrical unless there is a definite defect.



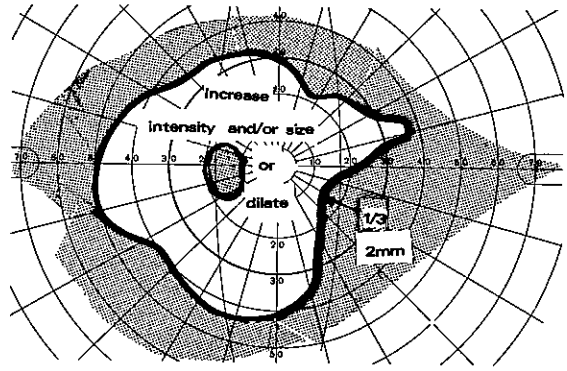


Figure 12 shows another poor field result. Here possibly the speed of the examiner was too fast or the patient's reflexes were poor. The target is only 1/3 and this should have been increased until a normal peripheral isopter could be elicited. The field looks defective but in actual fact would not help in pinpointing any disease.

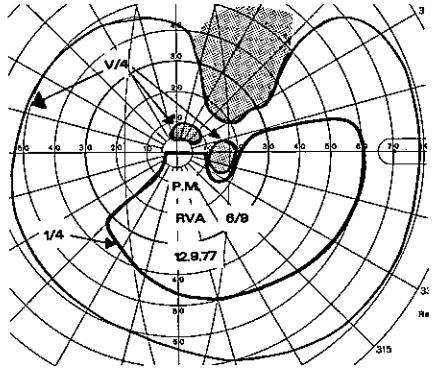
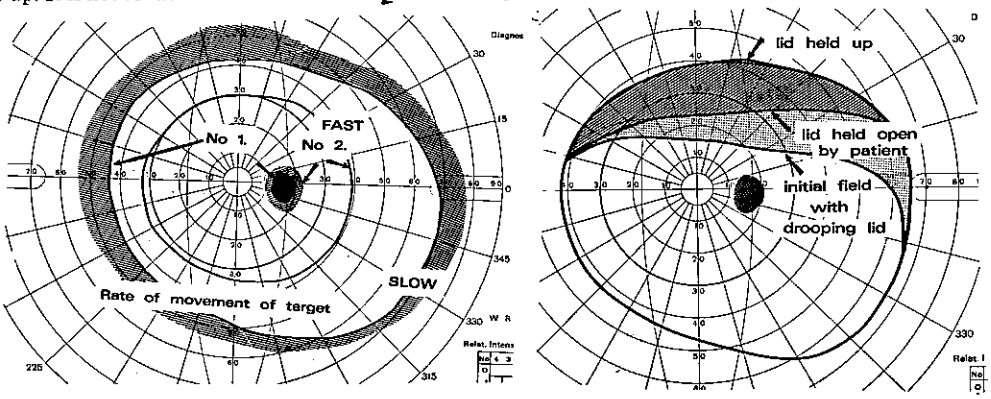


Figure 13 shows that if the field plotted with the 1/4 target was the only isopter plotted, one would not know whether the superior loss was relative or absolute. An increase in target intensity produced an increase in field plotted.

Figure 14 illustrates how speed of the target and reflexes of the patient may affect the result of the visual field. The reflexes of the patient must be known and can be tested using static spotting at the beginning of the test. The examiner should move the target with an even movement relative to the reflexes of the patient. An examiner moving too fast will produce a small visual field. One must also not anticipate seeing areas by slowing down movement or test the same area repeatedly without presenting the target elsewhere.

Figure 15 shows how superior field loss may be due to the lid or brow and this should be held or taped up. It is not always sufficient to ask the patient to "open wide".



It is important to know what you are looking for but it is often expectation of a defect in one area which leads to failure to find another defect. One must be forever reminded that the purpose of perimetry is to determine visual fields accurately and to discover any defect present. Non-existent defects are no help in diagnosis just as overlooked defects can hinder diagnosis and management. Finally, enthusiasm for perimetry should be a must if we are to be responsible professional perimetrists rather than technicians just doing a job.

I would like to thank Dr Ivan Goldberg and Dr Glen Gole for their help and Professor Fred Hollows who introduced me to the pleasures of perimetry.

A REVIEW OF 100 CASES OF CHILDREN WITH READING PROBLEMS

Shayne A. Brown
Orthoptist in Charge
Orthoptic Clinic
Sydney Eye Hospital

Since 1972 when the Newcastle research on Specific Learning Difficulties was first published, over 1,000 children have been referred by teachers to the Orthoptic Department of Sydney Eye Hospital for ocular assessment. The apparent break-through in the treatment of children who had not responded to conventional forms of educational help, led some teachers to believe that a solution to the whole reading disability problem had been found. The presenting symptoms and signs were multitudinous – general reading difficulties, reversals, poor spelling, lack of motor co-ordination – poor comprehension, untidy writing, etc. At first, children were frequently referred without a psychological assessment and without evidence of reading retardation. Quite often the parents did not know why the eye examination was requested.

During the past eighteen months, the standard of referrals has been more satisfactory. Children are being referred specifically because of a suspected visual component in the learning difficulty, or to rule out the possibility of visual processing problems.

We are well aware that a multidisciplinary approach to the investigation and treatment of learning difficulties is desirable, but this is not always easily implemented. Dunlop states that "the orthoptists' role is limited to ocular factors – she can have no part in the difficult parallel task of assessing relative intellectual potential or in the detailed analysis of writing and reading abnormalities".

As matters stand, the decision whether to treat, and how to treat, is left to us. We are expected to judge whether the ocular condition is a significant cause of failure, without the benefit of discussion with others who have studied the child.

Accordingly, the aims of this paper are

I) to identify, in general terms, the various types of reading problems,
ii) to review the cases of learning problems assessed and treated at Sydney Eye Hospital, and
iii) to discuss the difficulties faced by the independent practitioner.

1. IDENTIFICATION OF READING PROBLEMS

Most authors agree that remediation of a child's learning difficulties is mainly educational, but that there is a place for orthoptists, in both assessment and treatment. We must establish what this place is, if we practise independently of a multidisciplinary team. Barbara Cassin[6] said "Ophthalmologists and orthoptists should show interest in this type of problem (learning disability), learn how it relates to the eye, and become knowledgeable about learning disabilities as an educational handicap. Otherwise, the parents will have the child's eyes re-examined by someone more sympathetic, but not necessarily more able to give relevant help."

There appear to be many different classifications of learning disabilities. The classification of the University of Florida[6] appears to be the most practical in terms of relating the patient's history to the various categories. The six categories are:

Category I – Developmental Learning Disability (specific developmental dyslexia). Primary.

The characteristics of this category are – normal birth, normal motor and intellectual development for the first five years of life. At school (kindergarten) there may be difficulty in drawing and copying. In first grade these children have letter and word reversals and great difficulty learning to read, which persists despite average to high I.Q., normal cultural stimulation, absence of brain damage, intact senses, adequate motivation and normal schooling. Additional characteristics include familial incidence and a greater incidence in males.

Category 2 – Minimal neurologic dysfunction (M.N.D.). Secondary. M.N.D. does not show on tests and the history does not always give clear evidence of brain damage, but usually includes at least two of the following:– difficulties during pregnancy, prolonged labour, difficult birth, postnatal problems. The child may have delayed developmental milestones and hyperactivity but not the type of hyperactivity due to frustration. At school he may exhibit the same difficulties as children with development dyslexia, with these added signs: very limited concentration span, clumsiness and poor motor co-ordination (e.g. difficulty with scissors). Characteristic of this group is the continuation of perceptual motor deficits into adolescence despite remedial help. The I.Q. level is often low average; there is emotional lability, defective use of language, and difficulty with abstract reasoning and comprehension.

Category 3 – Brain damaged. This category includes children with manifest brain damage. These children have a positive history of seizures, abnormal E.E.G.'s, cerebral palsy or depressed I.Q.

Category 4 – Social and Emotional Learning Disability or both. The characteristics of this group are normal birth, normal milestones, but unlike the specific developmental dyslexics, there are no problems such as difficulty with copying or reversals. These children present as under-achievers, and the cause is emotional deprivation for lack of normal parental attention. The parents do not realise that they are the primary teachers of the child in the pre-school years. The problem in these cases is that the child is emotionally ill from disturbed human relationships.

Category 5 – Cultural Educational Deprivation. Mainly children from the low socio-economic level fall into this category because of poor prenatal care, a midwife rather than gynaecological delivery, or poor nutrition in the foetal and neo-natal state. The children are deprived culturally, or educationally because the parents are not capable of stimulating the child at an early age. Because their parents are failing intellectually, motivation for the children is lacking. Although they have problems with the alphabet, reading and spelling, they do not have associated neurologic and neuropsychologic deficits.

Category 6 – Combination of at least two of the above categories.

2. REVIEW OF THE ASSESSMENT AND TREATMENT OF CHILDREN WITH LEARNING PROBLEMS

A. SELECTION OF CHILDREN FOR REMEDIAL TREATMENT

The classification of learning difficulties given above provides a framework in which to consider our own role. We are familiar now with two binocular disabilities which affect reading. One is symptomatic convergence insufficiency, a state likely to discourage serious attempts at reading. The other is crossed lateral dominance, as tested by Dunlop's reference eye test, which shows a statistically high correlation with reading difficulties associated with reversals of letters and words. There is a school of thought which contends that lack of "visual perception" is a cause of reading problems. According to Herman Goldberg, Visual perception is the intellectualising of visual images. Thus the letters d o g become dog, a four-legged animal. The letter and word reversals common to Categories 1 and 2 (above) mean that visual images are incorrect or inconsistent, making intellectualisation more difficult.

Since Dunlop found that treatment of crossed laterality reduced reversals, with an 80% rate of improved reading ability, we have used crossed lateral dominance, as determined by the reference eye test, as our prime criterion for undertaking treatment, by occlusion and exercises. Other cases showing reversals were also accepted, for exercises only, as were cases of symptomatic convergence insufficiency.

B. STUDY OF FINDINGS IN CHILDREN TREATED

Although approximately 1,000 children have been referred to the Orthoptic Department, many attended for one assessment visit only; others did not complete treatment, and many are currently undergoing treatment. In order to assess the effectiveness of treatment, it was necessary to select a random sample, not from the 1,000, but from a group who had completed treatment and who had been under treatment for not less than three months. This time limit was chosen as it was felt that any scholastic improvement, as a result of ocular treatment would not be evident under this time period. The group who fulfilled this criteria numbered approximately 350, and of these, a group of 100 was chosen randomly on the advice of the senior medical records librarian at Sydney Eye Hospital.

All had been referred because they were not reading to the expected standard for their I.Q. All of them had completed treatment and were discharged at the time of considering the results.

The information taken from the medical records included age, sex, reasons for referral, visual acuity, the state of ocular motility and binocular function; handedness, eye dominance (both sighting eye and reference eye test as described by Dunlop[10]), the treatment prescribed; length of treatment and follow-up; and whether remedial reading was given elsewhere during the treatment period.

Improvement as a result of visual treatment was impossible to assess scientifically because psychological tests were not available either before or after treatment. Therefore, improvement (for the purpose of this study) could only be defined as that improvement in the child's performance which, as assessed by the teacher, was a direct result of the visual treatment. Unfortunately, even on this point, information was not available in every case.

SUMMARY OF STATISTICS

The group of 100 children consisted of 81 boys and 19 girls. This finding is in line with results of other studies, indicating that reading difficulties are more common amongst boys. Bettman[4], Cassin[6], Goldberg and Schaffman[12], etc.

The ages ranged from 5 years to 15 years. The average age being 9.2 years.

All children had been referred, by either a teacher or school counsellor, because they were not performing to their expected level. Only 4 complained of asthenopia associated with close work. The remaining 96 were referred because of poor reading, poor spelling, reversals, or a combination of all three. Of the group, 77 were right-handers, 19 left-handers, 2 were ambidextrous and 2 were not recorded. No girls were left-handed, one was ambidextrous. In the normal population one in eight is a left-hander, so in this sample the proportion of left-handers is almost twice as high. Of the number recorded, 32% had a history of familial left-handedness or ambidexterity.

The visual acuity was measured with either the Snellens Test Type or the Sheridan Gardiner chart. 91% had normal equal visual acuity (34% 6/6; 57% 6/5). Three cases had unequal vision due to refractive error; the remaining six had only slightly unequal vision, i.e. 6/6, 6/5. Of the 9 children with unequal vision, none had laterality problems, i.e. none of them had difficulties with reversals. According to Dunlop and Dunlop[11], equal vision is a characteristic of children with laterality problems. The presence of a deviation was determined by the cover test, prism cover test and Maddox Wing. 78% of the children were orthophoric, or exophoric for near only, which agrees with the 1977 survey of 5 year old children[5].

10 children had exophoria for near and distance
9 children had esophoria for near and distance
3 children had intermittent divergent strabismus.

Of the total number (863) of children referred for reading problems who had been assessed and recorded at our clinic, 1.5% had strabismus. In other series (Casin[6], Bettman[4]), the proportion of strabismus was not higher than in the normal population. In our experience the percentage of strabismus is lower than in the normal population. None of those with intermittent divergent strabismus had laterality problems.

Convergence was noted according to the R.A.F. rule divisions. Convergence ability was classed as normal (0-5 cms), reduced 16-10 cms) and defective (11 cms +).

	5,000 5 yr. olds	100 (average age 9.2 yrs)
Normal (0-5 cms)	86.4%	58%
Reduced (6-10 cms)	10%	33%
Defective (11 cm+)	1.8%	9%

The convergence ability of this group is very poor, as compared with the group of 5 year olds tested in 1977. Benton et al[1] found convergence insufficiency in 20% of cases and Nicholls[14] found convergence insufficiency to be significantly more frequent in children with reading problems. The latter also states that convergence is the least stable of all co-ordinated ocular movements and is easily disturbed by poor health or emotional upsets. So perhaps the convergence insufficiency in these cases is the result of the reading difficulty.

35% of children had less than full stereo acuity, measured either on the synoptophore or the Titmus Stereo test. Romano et al[15] and Dunlop[9] have found that the proportion of stereoacuity improves with ocular maturity. As the average age of this group was 9.2 years, it appears that, as a group, stereoacuity was significantly defective. One of the findings of Dunlop and Dunlop[10] is that children with crossed lateral dominance characteristically have less than perfect stereoacuity.

Eye dominance was tested monocularly by asking the child to look through a tube, and binocularly by the Dunlop method[11] as stated previously.

	Sighting Eye Test	Reference Eye Test (Dunlop Test)
Same as hand	53%	10%
Crossed or mixed	39%	87%
Not known or not applicable	8%	3% (one child could not do test; 2 children had defective vision)

C. TREATMENT AND RESULTS

Treatment consisted of convergence exercises only, occlusion only or occlusion and exercises. The children were instructed to wear the occlusion for all school work and homework. The average length of occlusion was 6/12. The total percentage of improvement was 59%. 14% did not improve and unfortunately, in 27% of cases the result of treatment was not recorded.

	Results of Treatment			
	Occlusion	Exercises	Exercises & Occlusion	Total
Improved	25	9	25	59
Not improved	7	0	7	14
Not known	15	4	8	27
Total	47	13	40	100

Of the 13 who had convergence exercises only, 9 (69%) improved, and 4 (31%) are not known.

The number of children treated for laterality problems by occlusion or occlusion and exercises was 87. The percentage of improvement was 57%; 16% did not improve and in 27% of cases improvement was not known.

The majority of those who improved with occlusion, or occlusion and exercises had been given remedial reading as well. This is to be expected as remediation MUST be educational as well as medical.

Again it is important to stress that accurate diagnosis of each child's specific type of learning problem had not been made known to us. But, interestingly, there was a useful percentage of improvement combined with a change of reference eye, amongst that group whose symptoms included reversals, which may indicate that these children fall into category one or two of the classification quoted earlier. In retrospect it is probable that some children would fit into one of the other 3 categories of learning difficulties (excluding the brain damaged category). Therefore, in some cases crossed lateral dominance would not have been a causative factor.

It is also important to recognise the psychological factors which may influence the prescribed treatment. It is reasonable to suggest that in the group who improved without alteration of reference eye (25% of improved group), some children were helped because the parents' attention was focused on a tangible reason for the learning difficulty. The child may have responded to treatment, because a reason for the learning difficulty was no longer due to his lack of trying, etc., but due to a "physical problem".

The average length of time of the follow-up period was 6/12. During the follow-up period, nine children who had changed reference eye after occlusion reverted to the original reference eye. The average duration of occlusion for these nine children was less than 6/12, probably too short a period.

DIFFICULTIES FACED BY THE INDEPENDENT PRACTITIONER

It is important to recognise the various types of learning disabilities and perhaps the classification of the University of Florida will be helpful. This is not to suggest that orthoptists are capable of diagnosing learning difficulties but some understanding of the many factors involved should help to put this problem into perspective for those of us who work independently of a multi-disciplinary team.

To decide when to stop occlusion is difficult, but these figures indicate that the length of occlusion should be not less than 6/12, and perhaps better results would be obtained with occlusion of 9-12/12 (Dunlop[11]). We have found that to avoid familiarity with the Dunlop reference eye test, it is advisable to see the child at intervals of not less than eight weeks, unless convergence exercises are prescribed as well. In such a case the initial visits are closer, but the Dunlop reference eye test would only be done every eight weeks. We have round the reference eye test difficult to perform accurately in a busy and noisy clinic, and therefore the decision to cease occlusion is difficult. We have found that it is easier when we combine 2 sets of the small house slides and use a pair with the same sized controls. But an objective test would be preferable.

CONCLUSION

Remediation should be mainly educational and treatment in conjunction with remedial reading appears to be the most beneficial.

Refractive or ocular motility problems do not cause learning difficulties, but strabismus, phorias, significant refractive errors and convergence insufficiency, should be treated so that the child has good symptom-free vision when learning to read.

Considering that this group of 100 children consisted of unspecified learning difficulties, 59% overall improvement is very encouraging. This study tends to show that occlusion is most beneficial when one of the symptoms is reversals, which suggests that such children may belong ing the University of Florida's Category 1, and perhaps 2.

Visually evoked cortical potential electrical tests have been suggested[13] as an alternative to the Dunlop reference eye test. This would be a desirable test, but it is very time consuming and would seem to be impractical at this stage.

When evaluating these results the psychological effect of treatment must not be overlooked. I do feel that while occlusion did alter the reference eye and the reverslas were stopped, that in some cases the psychological value of treatment must not be discounted or underestimated.

REFERENCES

1. *Benton Curtis D. Jr., McCann J. W. & Larsen M., Dyslexia and Dominance. AM. Journal Paed Ophthalmology 2: 53-57 July, 1965.*
2. *Benton Curtis D. Jr., McCann J. W., "Dyslexia and Dominance – some second thoughts" AM. J. Paed Ophthalmology 6: 220, November, 1969.*
3. *Benton Curtis D. Jr., McCann J. W. & Larsen M., "Dyslexia: A Practical Approach for the Ophthalmologist." Journal Paed Ophthalmology, 5: 25-29 February, 1968.*
4. *Bettman Jerome W. Jr., Stern E. L., Whitsell L. J. & Gofman H. F., "Cerebral Dominance in Developmental Dyslexia". Arch Ophthalmology – Vol. 78, 722-728, December, 1967.*
5. *Brown S., Jones David, "A Survey of the Incidence of Defective Vision & Strabismus in Kindergarten Age Children – Sydney 1976". Aust. Orthoptic Journal Vol. 15, 24-28, 1977.*
6. *Cassin Barbara "Strabismus and Learning Disabilities" Symposium: Strabismus Plus: AM. Orthoptic Journal Vol. 25, 1975, 38-45.*
7. *Dunlop P. – "The orthoptist's contribution to the multi-disciplinary group concerned with learning difficulties." Aust. Orthoptic Journal, Vol 14, 9-15, 1975.*
8. *Dunlop D. B., Dunlop Patricia – "A new Orthoptic Technique in Learning Disability due to Visual Dyslexia". Orthoptics – Past, Present and Future Ed Moore Main & Stockbridge, Stratton N. York, 11, 16. 1976.*
9. *Dunlop Patricia "Visual Development of 148 Pre-School Children over a period of 31 years" Aust. Orthoptic Journal Vol. 15, 19-23. 1977.*
10. *Dunlop D. B., Dunlop Patricia "Orthoptic Assessment of Children with Learning Difficulties". Aust. Journal of Ophthalmology. 2(3), October, 1974: 113-116.*
11. *Dunlop D. B., Dunlop Patricia "New Concepts of Visual Laterality in relation to Dyslexia" Aust. Journal of Ophthalmology. 2(3), October, 1974: 101-112.*
12. *Goldberg Herman K. et al "Dyslexia – Problems of Reading Disabilities" Grune & Stratton, New York & London.*

13. *Lux James P. "Detection of Learning Disabilities using the Visually Evoked Cortical Potential". Journal of Paediatric Ophthalmology. July-August, Vol. 14 No. 4, 248-253.*

14. *Nicholls, John V. V. – Part 3 Ophthalmic Disturbance – pp 49-52. In: Keeney & Keeney Ed. "Dyslexia – Diagnosis and treatment of Reding Disorders". The C. V. Mosby Company, Saint Louis, 1968.*

15. *Romano, P. E., Romano, J. H. & Puklin, J. E. (1976). "The Development of Normal Binocular Single Vision in Childhood" in Orthoptics, Past, Present, Future ed. More, Mein & Stockbridge, Stratton, N. York, 11, 16.*

ACKNOWLEDGEMENTS

I wish to thank Miss Jill Horsburgh, Senior Medical Records librarian for her advice on the statistical selection of the cases, and Mr. David Rivers for his advice on how to conduct this study.

A NEW ROLE FOR ORTHOPTISTS IN CEREBRO - VASCULAR ACCIDENT ASSESSMENT

Ann Macfarlane
Ted Longhurst
Eye Clinic Lidcombe Hospital
Joseph Street, Lidcombe. N.S.W.

SUMMARY

An orthoptic visual assessment programme, established as a preliminary to rehabilitation therapy for cerebro-vascular accident patients, is described, with case histories illustrating the advantages of the programme.

This paper describes the extended role of orthoptists at Lidcombe Hospital. This is a general hospital in the Western Suburbs of Sydney, having a strong emphasis on gerontology and rehabilitation.

From the time we began work as orthoptists in the eye clinic, we found we were being asked many general questions pertaining to vision such as "is 6/18 good vision?" and "what does N5 mean?", "should the patient wear glasses for therapy?" by occupational therapists, physiotherapists, speech therapists and therapy students. After discussing the therapists' concern about the visual problems of their patients, especially cerebro-vascular accident (C.V.A.) patients, we were encouraged by our ophthalmologists and supported by the medical superintendent, physicians and neurologists to initiate a visual screening assessment for these patients prior to their commencement of rehabilitation therapy.

Previously, when these patients were referred to the Eye Clinic it was with a note stating "this patient complains of being unable to read – for glasses check please." Most C.V.A. patients seem to complain of a deterioration in vision and being unable to read. They generally attribute this to the need for a change of glasses, but a C.V.A. does not alter refraction. More often than not their problem is caused by one of the other conditions such as nystagmus, loss of fixation, poor convergence, diplopia, field loss, overlapping images, etc. It is usually better to delay refraction until drugs are stabilized and the patient feels well enough to co-operate in subjectively assisting refraction.

After determining which tests gave therapists the visual information directly related to therapy tasks, we gave a further lecture to explain the testing and its significance.

At present we are screening each newly admitted C.V.A. patient. We have also been included in ward rounds and weekly conferences.

Screening begins with a detailed history:

1. history of C.V.A. – date and nature
2. past medical history (diabetes, hypertension, medication)
3. ocular history (age of glasses, previous ocular treatment)
4. symptoms (including questions about reading ability)

In the screening routine examination:

1. external observation (lids, conjunctiva, cornea etc.) is made
2. glasses are neutralized,
3. vision is tested for near and distance (and with pinhole if indicated)
4. reading technique is checked, noting words missed or misread and skipping of lines,
5. visual fields are plotted,
6. pupil responses are tested,
7. cover tests are done for near and distance,
8. ocular motility is tested for
   i) presence of muscle palsy
   ii) siting of lesion – a) pursuit b) saccadic c) position maintenance d) vergence – to test all eye movement systems.
9. Nystagmus is evaluated
10. Wirt Titmus Test for stereo acuity is given.
11. tensions are tested, as glaucoma is prevalent in the age group.
12. colour testing

A report is placed in the patient's history file to be seen by the patient's physician who may then refer to the ophthalmologist if there is the slightest evidence of an ocular defect. We make a point of seeing the therapist personally to explain significant findings.

We give the therapists the following results –

1. If it is necessary for glasses to be worn for therapy tasks.
2. The amount of vision present for near and distance and how this would affect certain tasks.
3. We explain the problem a patient may be having with reading i.e. the words at the beginning of a line are lost by left hemianopias and letter and word sequences are difficult for right hemianopias.
4. Fields are plotted as accurately as possible because many patients have a "unilateral neglect". "This neglect ranges from a passive to an active neglect of the affected side of the body, the neglect being visual, motor or sensory or a combination of these. The patient may not use the affected limb, i.e. when asked to raise his arms he will raise only one arm although motor power is normal. The unaffected arm may not cross the midline to wash affected side of the body".[1] The therapists need

to know whether the patient has an actual field loss or a neglect. Macular sparing is noted for reading and fine skills. Fields are repeated to observe changes as recovery takes place.

5. We explain the significance of control of phoria or problems resulting from a squint (recently acquired due to C.V.A. or present prior to C.V.A.) such as diplopia.

6. Ocular motility defects mean an explanation of whether A.H.P. should be retained. The normal therapy technique is to have straight head, to help balance. For nystagmus we are able to arrange an E.N.G. for positioning the head where nystagmus is least worrying.

7. We relate Wirt Titmus to tasks requiring finer degrees of depth perception (judgement with stairs etc.)

8. Colour testing is important because much therapy especially speech therapy depends on colour cues.

Orthoptic problems are referred back to us by the ophthalmologist and the usual orthoptic techniques of occlusion, Fresnel prisms and lenses, convergence insufficiency exercises, progressive Hess charts etc. are used.

As therapy for reading with hemianopias we suggested the use of a piece of black cardboard placed on the left side of the page. This ensures that the patient comes back to the beginning of the line with a left hemianopia. Large print books are suggested for right hemianopias to learn to read letters in sequence. If lines are skipped the cardboard is placed under each line and moved down line by line. This is reinforced by the occupational therapist.

Special adaptations of tests for C.V.A. patients

1. History

History with expressive aphasic patients has to be reduced to simple 'yes' or 'no' answers; but at times these responses are not accurate so the therapist has to be consulted first.

2. Glasses

It is important to ask if the glasses belong to the patient as several of our patients were found to be wearing other people's spectacles. We also label the near and distance glasses, as confusion often occurs.

3. Vision Testing

The patients, who are unable to respond to any other form of testing are tested by Catford drum. Those aphasic patients with expressive problems are tested with Sheridan Gardiner board and near vision is tested with reduced Snellen's chart. Also we use a reading passage, and give the patient descriptive pictures, to be matched correctly to the passage.

Figure 1. Demonstrating use of Sheridan Gardiner board, to aphasic patient.

4. Ocular Motility

Ocular motility for saccadic movements can be tested using opto-kinetic nystagmus, if patient will not concentrate on fixating two separated targets.

Examples –

a. Mrs. R.J. (C.V.A.)
Her hyperphoria became manifest after C.V.A. Resulting diplopia was joined by prisms. Reading and walking both improved remarkably from then on.

b. Mr. C.T. (C.V.A.)
Mr. C.T. was found to have an almost complete right homonymous hemianopia with very little macular sparing. His difficulty in reading caused such frustration that he was becoming violent with his family. By repositioning his reading material and taking each word slowly, Mr. T. gradually improved his reading ability and his whole attitude changed.

c. Mrs. M.B. (C.V.A.)
This lady developed a squint with diplopia which could not be joined by prisms or abnormal head posture. When given alternate occlusion, she was able to cope well with therapy sessions.

d. Mr. S. (C.V.A.)
Mr. S. was referred complaining that recently acquired glasses did not give clear vision. However, his near vision proved to be R.N5, L.N6. We found that the present glasses were adequate, and were able to convince him that the "loss of focus" after reading a few words was due to fine but irregular left beating nystagnmus. This nystagmus fortunately disappeared spontaneously.

e. Mrs. E.H. (C.V.A.)
She had bilateral cataracts, with VR. 6/60, VL 6/60, which she accepted as "just a part of being old". She was referred by a physician to the eye clinic, and the therapist was informed of the state of vision and the limitations it imposed.

f. Mr. B. (C.V.A.)
Was a right leg amputee and had a blind left eye due to an accident. He had a complete right homonymous hemianopia, and needed to use a pronounced face turn in order to make the most use of his small visual field.

We feel very strongly that the orthoptist has an essential role in the rehabilitation of C.V.A. patients. Orthoptists have the necessary background knowledge in neurology to contribute to their total assessment. We can assist the therapist to plan the treatment programme. At the same time we can save the patient from discouragement, by ensuring that he shall not be asked to perform a task which is beyond his visual capability. We also give orthoptic treatment where this is applicable.

In each hospital where C.V.A. patients are admitted for sufficient time to begin rehabilitation training, provision for orthoptic assessment should automatically be included. We must be ready to offer our services as an integral part of the therapy team.

Our thanks are expressed to Dr. Shirley Sarks and the other Ophthalmologists in the Eye Department at Lidcombe Hospital, Dr. G. Carter, the Medical Superintendent, the physicians, neurologists, registrars and residents and all the therapists for the help and support they have given to us.

*REFERENCES **

1. *Australian Association of Occupational Therapists Journal*

2. *Parietal Lobes – McDonald Critchley.*

THE ORTHOPTIST'S ROLE IN A TEAM APPROACH TO VISUALLY HANDICAPPED CHILDREN

Jane Pardey
Orthoptist, Sydney Eye Hospital
Mary Guy
Occupational Therapist, Royal Blind Society of NSW

THE ORTHOPTIST'S ROLE

The idea behind this paper is to acquaint orthoptists with the services of the Child Development Unit and the role of the orthoptist in the assessment of visually handicapped children. The involvement of the orthoptist in the Child Development Unit has led to the development of a Low Vision Programme to aid partially sighted children.

The structure of the Child Development Unit is as follows. It is headed by the Honorary Medical Advisory Panel.

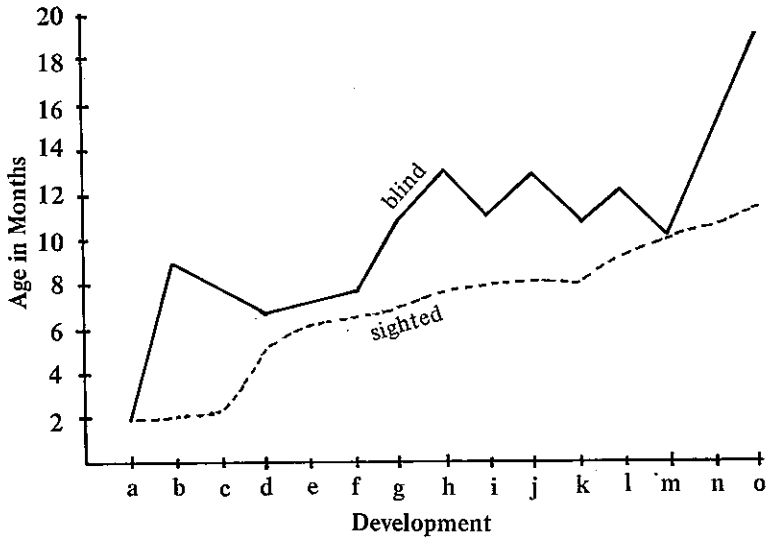
Regular home visits are made by members of the next seven groups.

Case Co-ordinators: social workers, who provide support for the parents, and act as links between the Advisory Panel and all other groups,
Fraiberg Programme: occupational therapist,
Low Vision Training Programme: occupational therapist in consultation with orthoptist,
Stimulation Programme for mobile children: occupational therapist,
Psychotherapist Consultant
Psychologist
Kindergarten Teacher: – fosters integration into local kindergartens
– provides support/back-up to these kindergartens
– offers individual pre-kindergarten programme.

The Co-ordinators further arrange
Referrals to the Education Department, re assessment for appropriate schooling,
Co-ordination with general practitioners, opthalmologists, paediatricians, to explain services,
Onward referrals to community support systems, e.g. playgroups, counsellors, field welfare officers.

The services of the Royal Blind Society's Child Development Unit need to be viewed within the context of society in general, as blind children have to grow up and learn to live in a sighted community. The aim is to provide support and guidance to the family as a whole, so they may help the growing child develop his abilities to the full. The programmes are mostly home based and the aim is to encourage the child to follow the normal patterns of development as far as possible. The parents are involved in this programme through various methods such as daily physiotherapy where necessary.

Figure 1 illustrates the development of a blind child in relation to a normal child. The Child Development Unit deals primarily with children in the pre school age range from the time of diagnosis and referral to school placement begins. Early referral is important. From the developmental point of view, during the first two years, the child's sensorimotor development is focused upon as without vision a blind child relies on sound and touch for his incentive. Hand development is an important area as blind people need to be adept in the use of their hands, so a variety of sensory stimuli are used, for example finger painting.



KEY

a. adjusts to mother's position when held.
b. elevates self in prone.
c. hands to midline
d. sits alone momentarily
e. rolls independently
f. sits alone steadily
g. four point kneel
h. crawling
i. raises self to sitting
j. pulls to standing
k. stepping movements
l. walks holding
m. auditory-tactile co-ordination
n. stands alone momentarily
o. walks alone

Figure 1. Development of Blind vs Sighted Children

The staff of the Unit soon felt the lack of basic information regarding the particular visual abilities of individual children. As a result, an orthoptist was seconded from the Sydney Eye Hospital on a one-day-a-week basis for a six-month trial.

The orthoptist's role in the Unit is to determine the amount of usable vision so that the Low Vision Programme can work towards developing visual skills to the highest possible level in order to supplement the child's other skills.

The programme at present is still very much in the planning stages and evolving slowly as more children are assessed and their needs become apparent. Visual learning programmes by Marianne Frostig, Natalie Barraga, Marvin Effron and Beth Reilly Du Boff to name a few, are being investigated as a basis for the programme, and at this stage it appears that there will be three main levels, each one being co-ordinated with the child's present programme:–

1. A visual stimulation programme for the younger children and those with minimal vision. Making the child aware of position of light sources for motility and orientation.

   Bringing colours and large objects to their attention.

   Identifying their toys, clothes and household objects,

   Identifying their body parts and movements,

   Introducing concepts of in-out, up-down.

2. The next stage for children over three with minimal vision and under three with useful vision e.g. gross and fine motor activities,

   Developing an accurate perception of body awareness and movement,

   Discrimination of colour, shape and size,

   Understanding 'different and same' with sorting shapes and objects.

3. For children over three years with useful vision. In this group it is sometimes necessary to decide whether the child will need special schooling or whether he will be able to fit into the normal school system. It is in this group that it is hoped to use visual perception developmental tests and programmes.

The function of the orthoptist at the Child Development Unit is to determine the visual status of the children, which is quite a different matter from visual acuity, as with many of them, although their acuity is very poor, their visual ability is quite good. The decision of whether a child is capable of undertaking a low vision programme is made on a complete visual assessment which may take several visits, together with the recommendations made by the occupational therapist involved.

The children are assessed in a variety of situations, at the Child Development Unit's playgroups, at home, at kindergarten and school. In the visual assessment of the child, a number of variations are used.

A questionnaire answered by the parents or a person such as the kindergarten teacher aids in the testing. The following questions are included:

At what distance does he hold objects?
Which eye does he hold objects closest to?
Does he screw up his eyes trying to see?
Does he close one eye?
Can he recognise colour? All colours?
Can he see a large ball (basketball size) at 1m, 2m, 3m, 6m?
Can he recognise pictures of familiar objects e.g. house, car, dog?
Does he turn his head to one side when observing? Which side?
Does bright light worry him?
Does he rub either eye often?

Observation can sometimes be misleading, but it is often useful in seeing how a child copes and can give an indication of any field defects. Ocular appearance may give information as to the type of defect e.g. microphthalmia. An abnormal head position should be noted, also non-visual habits such as smelling unfamiliar objects.

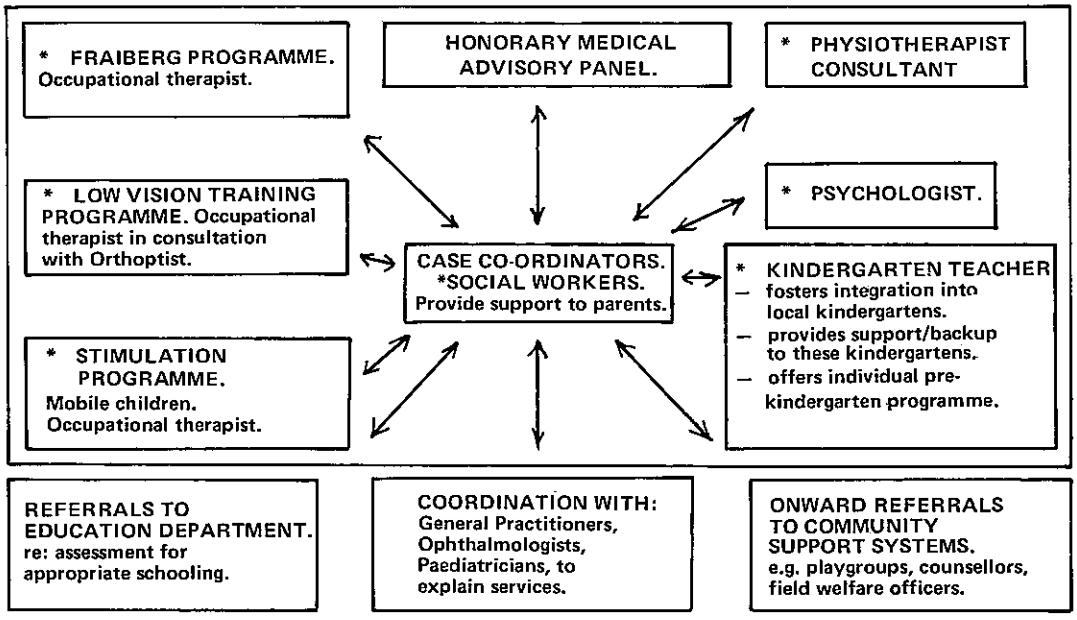
The tests for determining the visual acuity are the same as those used in a normal clinic for use with children under five, i.e. Stycar box, E cube and matching E, Catford Oliver drum, and pictures (chart and singles). The tests are often adapted and used at a closer than standard distance.

The Catford Oliver drum is used extensively, but with the frequency of pathological nystagmus, and the amount of pathology limiting the area of functional retina, this is not always accurate. Also the Stycar box, incorporating the Sheridan Gardiner balls and letter tests with 5, 7, or 9 letters, and the Stycar toys are widely used. The visual safety acuity is more often tested with both eyes open.

The visual field is approximated by observation and confrontation, with older children. Colour vision is tested with the Matsubara Colour Vision Test for Children.

Many of the children are not using binocular vision, and depending on the aetiology of the visual impairment, many of the findings on a routine orthoptic investigation may change from one visit to another. The accurate assessment of vision is of primary importance.

The ever expanding role of the orthoptist is well illustrated in the team approach to the treatment and management of the visually impaired child. The ongoing programme to enhance the child's abilities depends on the orthoptist's assessment of useful residual vision. This is challenging work in an unusual field which will bring great satisfaction to any orthoptist who may be called upon to work with these children.



KEY. * Makes regular home visits.

SCHEMATIC REPRESENTATION OF THE MULTIDISCIPLINARY CHILD DEVELOPMENT UNIT.

ACKNOWLEDMENT

The authors gratefully acknowledge the assistance of the staff of the Child Development Unit and the Sydney Eye Hospital Orthoptic Clinic.

BIBLIOGRAPHY

1. *BINOVEC, M. (1977) Orthoptics and Squint Management of the Cerebral Palsied Child. Aust. Orthopt. J. 15. pp 15-18.*

2. *ELLIOT, V.C. (1975) Cerebral Palsy and Orthoptics. Aust. Orthopt. J. 14 pp 7-9.*

3. *EFRON, M. and DU BOFF, B.R., (1975) Vision Guide for Teachers of Deaf-Blind Children.*

4. *FRAIBERG, S. (1971) Intervention in Infancy; a programme for blind infants. J. Americ. Acam. of Child Psychia. 1971. 10 (3) 381-405*

5. *ZIMMERMAN, D.r. (1977) Birth Defects and Visual Impairment. Vis. Impair. and Blind. Jan. 1977.*

ORTHOPTICS IN ARGENTINA

Alberto O. Ciancia, M.D.
Callao 1395, Buenos Aires (1023) Argentina

I highly appreciate the honour implied by The Orthoptic Association of Australia's invitation to participate in this bright Congress.

It will be a great pleasure for me to tell you about "The present state of Orthoptics in Argentina", as Miss Carter kindly asked me in your name.

The Argentine Republic, as you know, occupies the southern-most end of South America. Its Capital, Buenos Aires City, is almost at the same latitude as Sydney.

Two outstanding personalities of Argentine Ophthalmology in the past were Dr. Pedro Lagleyze and Dr. Jorge Malbran. Both showed special interest in strabismus and wrote interesting books about ocular motility. These works reveal their great interest in the sensory aspects of strabismus, perhaps due to the influence of the European schools of that time.

This interest in the sensory problems persisted among Argentine ophthalmologists until recently.

Howevery, the development of orthoptics as a professions was slow, and employed rather few people.

Two Universities included courses on Orthoptics: The University of Buenos Aires, the Capital of the country, on the shore of the Plata River; and the University of Mendoza, at the foot of the Andes.

The course at Buenos Aires University was recently discontinued.

Fortunately, the course at the University of Mendoza, created by Professor Roger Zaldivar, is very active. The graduates have the title of Auxiliaries of Ophthalmology and show a very good level of knowledge in orthoptics, tonometry, and other special techniques of modern ophthalmology.

Many opthalmologic centres of the country benefit from the assistance of these efficient professionals.

The evolution of orthoptics in Argentina followed, as was to be expected, the fortunes of orthoptics in other parts of the world.

When interest in the classical English techniques began to fade the works of Cuppers, Bangerter, Bagolini and others, widened the field of orthoptics, and its practice increased significantly. These developments produced an improvement in the knowledge of the physiopathology of binocular vision and new possibilities of treatment began to appear.

However, it is necessary to accept that the therapeutic results did not come up to initial expectations.

The disillusionment produced in time damped the initial enthusiasm and generated a contrary wave movement. As often happens, this reaction was excessive and the value of orthoptics as a whole was questioned.

This is a world-wide phenomenon, and an indicator of it is the great proportion of papers dealing with surgery that can be read in Orthoptic Journals.

Meanwhile, some new developments created certain points of interest. The so-called treatments in free space of Cuppers, Hugonnier, Lavat, Bagolini and others, excited the interest of some ophthalmologists and orthoptists. Specially, the appearance of the wafer and Fresnel light prisms was received with great enthusiasm. At our Hospital, in Buenos Aires, we tried largely to overcome ARC with prisms.

Unfortunately, ARC, versatile and unseizable, continues eluding our efforts to normalize it. It seems as if correspondence were comfortable in that state which we insist on calling anomalous and try to change.

The present crisis of the treatment of ARC, often confused with a crisis of orthoptics, is a world wide phenomenon.

In the professional work of orthoptic technicians this crisis provoked, at least in Argentina, on the one hand, a decrease in the work available, and on the other, a diversification of the tasks performed by orthoptic technicians in hospitals and consulting rooms.

This diversification, paradoxically, led to an enrichment of the professional background of orthoptists, because they have gained insight on certain motor aspects previously the province of ophthalmologists.

On the other hand, a closer collaboration can be observed between ophthalmologists and orthoptists in the evaluation of the patients and even in the tasks of clinical investigation.

The present generalization of the EOG and EMG studies also found dedicated and highly qualified assistants among the orthoptic technicians.

This shift of the orthoptist technician's activity towards areas which are out of the scope of classic orthoptics leads the orthoptist to occupy part of the time, and sometimes most of it in activities not directly related to the cure of binocular vision. Undoubtedly, this is a widening of the professional field of work and from this point of view it is both positive and desirable. But it would be a pity if orthoptists should discontinue the search

for new and better ways of diagnosis and therapeutics of the sensorial disfunctions of binocular vision, a wide and fascinating field which is indeed their specific task.

I shall briefly review some of the present orthoptic activities performed in Buenos Aires by orthoptic technicians.

Concerning orthoptic diagnostic procedures I would like to emphasize two points:

The first is the detection of small angles of anomaly. Although this diagnosis is frequently established by the ophthalmologist, it is rather often necessary, particularly in small children, to carry out thorough and repeated examinations that lie mainly in the hands of the orthoptist. Her skill in detecting very small angles of anomaly is a point of justified pride for the orthoptist.

The second point is the longitudinal study, after surgery, of the sensory readjustments of patients with ARC. Binocular vision with ARC is so complex and, let us confess it, so unknown, that we should use every opportunity to widen our knowledge on this matter.

Concerning this point, and as an example of what can be done in this field, you can read the findings made in a group of overcorrected esotropias in a paper presented by Ciancia and Melek, at the Third International congress of Orthoptiste held at Boston.

This reveals only a very little portion of a much wider field. The varied types of sensorial changes after surgery, and sometimes after the wearing of glasses or prisms, show possible lines of research open to orthoptists.

Conversely, the influence of the type of sensory changes on the motor changes is a wide and almost unexplored field.

Let us say a few words about the work of the orthoptists in the field of therapy.

Of course, as in the rest of the world, orthoptists in Buenos Aires treat patients with accomodative esotropia and convergence insufficiency.

We discontinued the treatment of ARC in esotropias. Only in special cases we undertake this treatment with prismatic overcorrection. Close control of the sensorial motor changes of these patients is performed with the aid of the orthoptic technician.

But now I would like to mention more in detail two ways of treatment that we use in Buenos Aires which have not become widely known: one is the treatment of amblyopia with excentric fixation by controlled inverse occlusion, and the other is the treatment of anomalous correspondence in intermittent exotropia.

The treatment of amblyopia is one of the main fields of work for the orthoptic technicians.

Many amblyopic patients are cured with direct occlusion, that is, occlusion of the sound eye. The various hues of this treatment, the practical problems it poses and, especially, the necessary care to avoid damage to the occluded eye in infants, make necessary a very close care that only the orthoptic technician can provide.

In amblyopic patients over the age of four, showing excentric fixation, direct occlusion frequently fails. The pleoptic treatments in which we put such great hopes some years ago, did not produce therapeutic results worthy of the enormous effort they demanded.

At the Children's Hospital of Buenos Aires we developed one method of treatment of amblyopia with excentric fixation in children over four years of age, called "controlled inverse occlusion".

The technique is roughly as follows:

The amblyopic eye is permanently occluded. The fixation is reviewed weekly by means of the visuscope. In the majority of cases a shift of the zone of fixation towards the macula is observed. Sometimes, an increase of the wandering movements is seen in the first weeks. When the fixation movements are centered in the foveal zone, the fixating eye is occluded. The patient is seen again the following day. Sometimes the fixation goes back in the direction of its previous excentric zone. In this case the amblyopic eye is occluded again for some weeks. When, after 24 hours of occlusion of the good eye, the fixation stays in the foveal zone, occlusion of the good eye is continued until the cure is complete.

Uncontrolled inverse occlusion is ineffective: it can be either too short and consequently insufficient. or too long. In the latter case the fixation becomes temporal, and the prognosis worsens.

Controlled inverse occlusion is frequently successful in excentric fixation in the maculo-papilar area. It generally fails in parapapilar, temporal and far vertical fixators.

When, after three months of inverse occlusion, the fixation does not show a definite shift toward centralization, the treatment is discontinued. In such cases we change the occlusion to the sound eye and in some cases the fixation improves.

In a statistic presented by Gurovich and Fisher at the Latin American Council of Strabismus we found that 48.52% of the cases regained central fixation with inverse occlusion, and 27.94% with inverse followed by direct occlusion. The total of cured cases reached 76.4%

Study of the patterns of change in eccentric fixations under occlusion of the amblyoptic eye is a fascinating task performed by orthoptic technicians at the Children's Hospital.

Another interesting point is the treatment of anomalous correspondence in intermittent exotropia.

Strangely enough, in almost all the papers dealing with intermittent exotropia, when speaking of the sensorial disfunctions, only hemiretinal suppression is named, and ARC is disregarded.

In spite of that, the presence of ARC in intermittent exotropia is easily shown when the patients are studied with the visuscope maculomacular test, or by means of the diplopia provoked with a red filter and prisms. Less frequently, ARC can be also found with the synoptophore and with the after-images tests.

In Buenos Aires, Melek found ARC with at least one of the tests in as much as 40% of the cases of intermittent exotropia.

The frequent recurrences of the exotropia after surgery led us to try to fully correct the sensorial anomalies, as a means to avoid such recurrences.

The "Elimination" treatment of Hugonnier allows us to correct ARC in children above 7 years of age. But in younger children it is difficult to get the collaboration this method demands.

Together with Melek we presented a new treatment of ARC in intermittent exotropia based on the therapeutic use of the visuscope.

In short, the technique is as follows:

The patient looks with one eye at a spot light located at 5 m distance. The visuscope star is projected on the fovea of the other eye. The eyes are in a divergent position. The patient sees the visuscope star some times not only in homonymous diplopia, but also not on the wall but at a few cms from his eye. We say that the correspondence is anomalous both in direction and distance. Then we ask the patient to handle the visuscope, and to try to superimpose the star and the light. The orthoptist observes the position of the star in the patient's retina.

Frequently the repetition of this movements provokes the super-position of star and light while the star is projected on the fovea; the correspondence is now normal.

If this does not happen, a minus three lens is put in front of the eye fixating the spotlight, so that it appears as a small reduced image at 33 cm. As a rule in this situation the exotropia disappears and both eyes are directed towards the near (apparent) position of the spot light. Normal correspondence then reappears. The minus three lens is now removed and the visual axes gradually swing apart: exotropia reappears. In the first attempts, when the eyes deviate anomalous correspondence returns. Finally the patient superimposes star and point of fixation while the observer sees the star in the patient's fovea: normal correspondence. The fact that the patient's hand moves the visuscope actively trying to superpose the star and the fixation point, has a definite effect toward the normalization of the correspondence.

As a rule, when the correspondence becomes normal with the visuscope, the patient has spontaneous diplopia when the eyes deviate, but the relative projection of the diplopic images shows that the correspondence is, in this condition, still anomalous. The last step is to correct also the anomaly in this condition. This is performed with techniques derived from Pemberton's propioceptive reorientation, that you all know so well.

Unfortunately our hope that normalization of the correspondence should prevent the recurrences after surgery was rather seldom realised. Anyhow, it is important to know that anomalous correspondence in intermittent exotropia does exist and can be cured, and a slight residual postoperative exotropia with normal sensorial relationship is a much more bearable situation that a variable and uncontrollable exotropia with anomalous correspondence.

It was a great pleasure for me to talk with you about these matters concerning our daily work. May I just wish you a pleasant stay in Singapore and a happy and successful future.

REFERENCES

1. *Ciancia, A. and Melek, N :The Sensorial Evolution of Overcorrected Esotropias, In Orthoptics, Past, Present, Future Pag. 501-506 –*
Stratton Intercontinental Medical Book Corporation New York, 1976.

2. *Gurovich, L. Tratamiento de la ambliopia con fijacion excentrica mediante oclusion inversa controlada. Actas del III Congreso del CLADE, pag. 33, 1971.*

3. *Ciancia, A. and Melek, N.: Nuevo tratamiento de la correspondencia anomala en la exotropia intermitente. Arch. Oft. Bs. Aires XLIV, 108-119, 1969 –*

4. *Melek, N.: La exotropia intermitente. Observaciones clinicas y quirurgicas. Buenos Aires, 1977.*

SOME ASPECTS OF PAEDIATRIC EYE PROBLEMS IN SINGAPORE

Professor Wong Hock Boon, (FRCP Edin) FRACP, DCH (Lond)
Department of Paediatrics
University of Singapore

The theme of this Congress is "The Eyes of Three Cultures". Unfortunately, it is extremely difficult to define the word culture, which has many connotations. It is usually interpreted rather facilely as "learned behaviour", and hence embraces many aspects of acquired attributes. In fact, many equate it with "civilisation". Even if culture is mainly acquired, the "acquiring" of many things is tied up with the genetic make-up of the individual. Hence, culture also depends on inheritance. I shall therefore discuss some aspects of eye problems in Singapore, which as you know is the meeting point of 3 great cultures, viz. Chinese, Malay and Indian. There will therefore be differences in genetic constitution, as well as customs, but, fortunately, a lot of these latter differences have been ironed out by the benevolent and effective health policies of the Government of Singapore, in terms of eye problems.

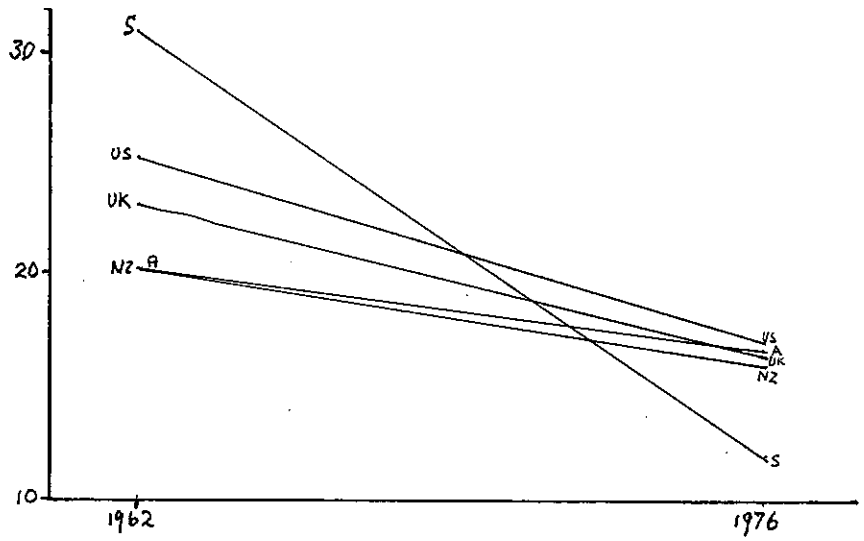
Before I discuss the various eye problems, it is useful to recapitulate very briefly the changes which have occurred in the delivery of paediatric health care over the years in Singapore. Only, in this way, can the present position be appreciated. I will touch only upon the infant mortality rate, i.e. the number of deaths per 1000 liveborn babies during the first year of life, and the neonatal mortality rate or the number of deaths per 1000 liveborn infants during the first month of life. The infant mortality rate (IMR) and the neonatal mortality rate (NMR) are indices of the level of child health care, and accepted by all countries. In 1962, when the University Department of Paediatrics was first established, the IMR and NMR of Singapore are compared with those of USA, UK, Australia and New Zealand (1):

CHILD HEALTH IN 1962

	USA	UK	AUSTRALIA	NZ	SINGAPORE
INFANT MORTALITY RATE	25.3	23.0	19.8	20.9	31.2

It is true that the developed countries have improved on their figures somewhat, but the improvement in child health in Singapore has overtaken these developed countries. This is summarised in Fig. 1.

Infant Mortality Rate (Deaths Below 1 years/1,000 Live Births)



Such an improvement in child health in Singapore would also reflect on the paediatric eye problems.

BLINDNESS:

The definition of blindness has had a chequered career. In practice, it is usually accepted that a child is considered blind if he has insufficient sight to receive education in a normal school, and that an adult is considered blind if he has insufficient sight to enable him to carry out a job for which eyesight is essential. This broad definition is as good as any, and anyway, it is a practical one. In 1972, the World Health Assembly (2) noted that there were 65 different definitions of "blindness" and tried to lay down some guidelines so that figures from different countries may be compared. It is estimated that there are at least 10 million blind people in the world today (3), and if the definition covers those whose vision is defective enough for normal activity, then many more millions must be added to this. Of this large number of blind people, there are 3 common causes of their condition.

1. TRACHOMA due to an organism, CLAMYDIA TRICHOMATIS
2. KERATOMALACIA due to Vitamin A deficiency
3. ONCHOCERCIASIS due to a microfilaria or worm carried by a species of fly.

In Singapore, the prevalence of blindness has never been accurately assessed. Most of the figures through the years have been obtained by the Eye Department of the Singapore General Hospital, and such patients were subsequently referred to a Voluntary Organisation – the Singapore Association for the Blind. It is true that such figures are biased, but most figures from various countries are equally biased. In the immediate post-Second World War (4), the main causes of blindness are seen in the following figures, which depict new admission cases into the Eye Department, which served as the only centre for the treatment of eye diseases during that period in Singapore:

NEW ADMISSIONS

	1946	1947	1948	1949	1950
TRACHOMA	15	32	53	23	75
CORNEAL ULCERS	18	33	33	46	23
CATARACTS	167	311	151	269	217

Most of the cases of corneal ulceration were due to Vitamin A deficiency keratomalacia, and included large numbers of babies and children, and in the Annual Report of the Health Department for 1947 (4), it was stated that "there has been an alarming increase of keratomalacia, and that this was due to the increased use of sweetened condensed milk." It is rather interesting that the change-over from breast-feeding to artificial feeding was already increasingly evident even in 1947 in Singapore due to the activities of milk firms, so much so that breast feeding in Singapore now is even worse off than in the West (5). Legislation was then introduced forcing milk firms to add Vitamin A to canned milk, and this almost totally eradicated xerophthalmia from Singapore, and today, no new cases are seen. (6) Medical students today are unable to diagnose keratomalacia and have no idea what Bitot spots are, though such cases are extremely common in the surrounding neighbouring countries.

With regard to trachoma, it was extremely common at that time in Singapore. Again, it was so alarming that the Annual Report for 1949 has this to say: "Experience has shown that all Sikh immigrants from India, without exception, show signs of trachoma of varying severity, and immigration is thought to keep this disease alive. Should immigration from India and China ever begin on any scale, the question of barring all immigrants with active trachoma at ports of embarkation would have to be revived." Of course during those days, Singapore was still a British colony.

Coming to more recent times (7, 8, 9), the prevalence of blindess in Singapore is as follows:–

YEAR	NO. REGISTERED	RATE PER 100,000 POPULATION
1964	1015	55
1972	1442	67
1978	1520	66

The prevalence rate for Singapore is therefore about 60-70 per 100,000 of the population. It is very difficult to compare prevalences among different countries because of the differences in definition of blindness and the assiduity demonstrated in detection and registration. WHO tried to get some estimate of prevalence in different countries in 1966 (10) but it was obvious that the figures obtained cannot be meaningfully compared because of differences in materials and methodology. Estimates in Africa, at the moment, reveal the following (2):–

COUNTRY	PREVALENCE RATE PER 100,000 POPULATION
Ethiopia	380 – 450
Kenya	1050 – 1150
Malawi	724
Sierra Leone	952
Uganda	1842

In the United States, it is about 214 with a total blind population estimated at 385,000. In South America, the rates for Argentina and Uruguay are about 80-90 per 100,000 and in Cuba it is 969. Brazil and Mexico have 147 and 101 respectively. Estimates in Asia are as follows:–

COUNTRY	PREVALENCE RATE PER 100,000 POPULATION
Japan	248
Saudi Arabia	3000
Yemen	4000
Iraq	500 – 1000
Pakistan	1000
Sri Lanka	470
Vietnam	428
Indonesia	239
China	450
Hong Kong	1392

To give an idea of the rates in Europe, the following table is revealing:–

COUNTRY	PREVALENCE RATE
Belgium	51
Spain	56
Netherlands	50 – 60
Federal Republic Germany	60
Poland	66
Romania	77
Portugal	93
Yugoslavia	100
Hungary	100
France	107
Switzerland	145
Greece	170
England and Wales	209
Iceland	272
Gibraltar	647

and in the Soviet Union, 179,317 people out of 200 million have been recorded as blind.

With regard to age at registration in Singapore, approximately 23% are below 15 years, 34% from 15-55 years and 43% from 56 years and above. A more detailed breakdown from the figures obtained in 1972 (11) is shown below:–

AGE	NO.
Birth – 6 years	22
6 – 16 years	136
16 – 21 years	62
21 – 44 years	261
Above 44 years	990

Prevalence figures for blindness in any country often are not accurate but give only estimates. Figures for actual causes of blindness as estimates of aetiological prevalence are even less accurate. For what it is worth, the causes of blindness in Singapore from 1950-1972 are as shown in the following table (8):–

CAUSE	NO.	%
Congenital and developmental	268	13.7
Corneal diseases	302	19.6
Uveitis	82	4.2
Other inflammation involving the Uvea	75	4.0
Retinal degeneration (including myopia and detachment)	180	9.2
Cataract	88	4.5
Glaucoma	380	19.4
Optic atrophy	389	19.8
Trauma	24	1.2
Tumors	6	0.3
Unknown	88	4.5
Double-groupings	77	3.9

At the moment, the commonest causes of blindness in infants and children are the following:–

A) GENETIC DISEASES:

This forms the largest group and include the following categories:

1. Mendelian monogenic diseases such as autosomal dominant diseases, e.g. Marfan's Syndrome, certain retinoblastoma, aniridia, etc., autosomal recessive diseases such as certain types of optic atrophy, cataracts, choroido-retinal degenerations, mucopolysaccharidoses, etc.
2. Chromosome diseases of various types
3. Multifactorial, comprising diseases where polygenes interact with the environment such as severe myopia, congenital glaucoma, colobomas etc.

B) INTRA-UTERINE ACQUIRED DISEASES:

This could be due to drugs and intra-uterine infections congenital rubella, toxoplasmosis are examples. Blindness due to congenital syphilis or due to gonococcal ophthalmia is non-existent in Singapore.

C) ACQUIRED DISEASES IN INFANCY AND CHILDHOOD:

This is rare as Vitamin A deficiency keratomalacia, and trachoma and onchocerciasis are now not encountered in Singapore. However, some cases of retrolental fibroplasia have been seen due to excess oxygen therapy in the respiratory distress syndrome in newborns. No accurate incidence figures are available in Singapore.

In a survey in England and Wales (12), it has been estimated that the causes of blindness in childhood are as follows:–

CAUSE	NO.		%
Genetic: Mendelian			
Autosomal Dominant	158		
Autosomal Recessive	130		
Sex-linked	39		
Total		327	43
Acquired:			
Prenatal	44		
Perinatal	258		
Postnatal	77		
Total		379	50
Others:	59		7
Grant Total		765	

It is interesting to see that about ½ are genetic, and almost all the remainder are acquired during intra-uterine life and during birth itself. Of the 258 perinatal cases, 177 were due to retrolental fibroplasia as a result of excessive oxygen therapy for small premature babies with hyaline membrane disease. Hence, about 90% of causes of blindness in infants and children occur before they are born, after fertilisation and in the early days of life

The incidence of causes of blindness in Singapore roughly resemble these figures in UK, i.e. our causes are very much like the causes in the developed countries. This is not surprising since the health of our infants and children is as good as that in the West.

What can be done therefore to cut down blindness in infants and children? The answer is genetic counselling which is being carried out in the Department of Paediatrics, University of Singapore, for the whole country. It also depends on good perinatal and neonatal care. In Singapore, rubella vaccination is now offered to schoolgirls (13). The careful use of oxygen therapy in respiratory distress syndrome should prevent retrolental fibroplasia.

COLOUR VISION:

The role of genetic evolution in producing different frequencies of colour blindness in various ethnic groups is speculative. For example, (14) Mann gives the following percentages of red-green defective males among some ethnic groups:–

ETHNIC GROUP	% RED-GREEN BLINDNESS
Australian Whites	7.3 – 12
American Whites	8
Europeans	8.4
Australian Aborigines	2.32
Japanese	3
Chinese	8.4

the highest incidence of red-green colour blindness is seen in Caucasians with lower frequencies in Japanese and Australian aborigines. What is the significance of this difference? We have found that the incidence in Chinese in Singapore is much lower than the 8.4% figure given by Mann.

Over a period of 3 years, 1974-1976, the School Health Services (15) had screened 300,000 Chinese males and females, 60,000 Malay males and females and 20,000 Indian males and females, by the use of Ishihara charts. The results are as follows:–

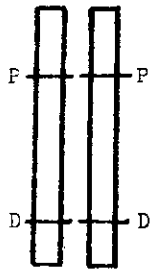
ETHNIC GROUP	% RED-GREEN BLINDNESS	
	Males	Females
Chinese	3	0.1
Malays	2.9	0.1
Indians	2.6	0.1
Others	4.9	0.5

There were 4,500 'other races' mainly Eurasians and Caucasians. There is no doubt that the Chinese, Malay and Indian frequencies are 3% in males and 0.1% in females, and these figures resemble those of the Japanese and is 1/3 less than for the Caucasians. It is important to screen colour vision in children because, in the present stage of social evolution, colour plays a great part in the process of everyday living, e.g. learning from books, electronics, chemistry, medicine, art, printing, etc.

The gene/genes for colour vision are situated in the X-sex chromosome. Because there are different types of colour blindness, it has been postulated that there is more than one locus and that for each locus there are alternative mutant genes. The various phenotypes arising from the different mutant genes have been given terms which have been confusing. Essentially, there are 2 loci, each locus occupied by a pair of genes. One pair is termed the PROTAN gene because it allows us to see the primary colour RED – it can be represented as P^N (or for normal). The other pair is termed the DEUTAN gene because it allows us to see the secondary colour GREEN – it is represented by D^N. In colour-blindness, if the defect is one in discerning red, it can be severe or a mild type. If severe, it is termed PROTANOPIA and if milder, it is termed PROTANOMALY, represented by the symbols, P^P and P^L respectively. Similarly, if the defect is one in discerning green predominantly, it also can be severe or it can be mild. If it is severe, the term DEUTERANOPIA is used, and if mild, it is DEUTERANOMALY, and can be represented by D^P and D^L respectively. Therefore, various combinations of normal and mutant genes may be present in each or both loci. However, there is a precedence or dominance of one gene over the other in respect of phenotypic express, i.e. $P^N > P^L > P^P$ and $D^N > D^L > D^P$. Hence, the situation can be seen diagramatically, as in Fig. 2:–

GENE LOCI FOR

COLOUR-BLINDNESS



P^N; PROTANOPIA(P^P); PROTANOMALY(P^L)

D^N; DEUTERANOPIA(D^P); DEUTERANOMALY(D^L)

DOMINANCE

$$P^N > P^L > P^P$$

$$D^N > D^L > D^P$$

Among the male colour-blind children in Singapore, the commonest is the DEUTERANOMALOUS TYPE which comprise 63% of all colour-blind boys. Deuteranopia, protanomaly and protanopia comprise about 12% each. When there is total inability to see any of the colours, it is termed ACHROMASIA. The actual incidence in Singapore is as follows:–

	% ACHROMASIA
Chinese	0.25
Malay	0.22
Indian	0.26

It has been suggested that colour-blind males would be selected against when our ancestors were living by hunting, but that with assumption of farming, the colour-blind individuals would not suffer from such a selective disadvantage. However, this is still speculative (16, 17).

MYOPIA:

Of all the so-called refractive errors, myopia is the commonest and most troublesome problem in Singapore. The detailed studies of Sorsby et al (18) established that the refraction of the eye depends on at least 5 factors, viz. corneal power, depth of the anterior chamber, lens thickness, anterior and posterior powers of the lens and axial length. In the normal emmetropic eye, they found that there is a high correlation of axial length with both the corneal power and lens thickness. Therefore, the normal eye grows in a co-ordinated manner. The ametropic eye becomes abnormal not so much because of abnormal constituents as because of a faulty correlation of the individual components and the degree of ametropia is a measure of the degree of faulty correlation. Most refractive errors are due to disturbance of correlation between axial length and corneal power and not a disturbance of correlation between axial length and lens power. Lens power is more highly correlated with axial length irrespective of refractive anomalies.

In Singapore, schoolchildren have been routinely examined annually for vision using the simple Snellen Chart (15). Those aged 7, 9, 11, 13 and 15 were examined, and the numbers respectively were 41,578; 43,663; 51,870; 40,421; and 36,915. The percentages of each group with the different visual acuities (expressed as 6/6.75 – 6/12; 6/18, 6/24, 6/36, 6/60 and <6/60 and normal) are as follows:–

AGE	6/6.75-6/12	6/18	6/24	6/36	6/60	<6/60	NORMAL
7	13.7%	1.7%	1.3%	0.8%	0.55%	0.54%	80.9%
9	9.8%	2.2%	1.6%	1.4%	0.87%	0.88%	83.1%
11	8.3%	3.4%	3.5%	4.4%	3.9%	3.2%	73.2%
13	11.0%	3.6%	3.7%	6.8%	6.4%	8.8%	59.7%
15	9.6%	3.8%	4.4%	8.5%	8.7%	13.3%	51.7%

When the children commence normal primary school 81% have normal vision. By the age of 11 years, i.e. 4 years later, this has fallen to 73%, and by 15 years, 50% have abnormal vision, and it is common to find that half the class in secondary schools are wearing glasses. This is in sharp contrast to the situation in 1950 (4), when it was reported that "2.91% girls and 2.52% of boys had visual defects", and in 1951 that "3.95% of girls and 1.31% boys had visual defects". There has been a significant deterioration of eyesight over a period of 25-30 years even when the state of nutrition of our children has improved considerably.

This throws some light on the aetiology of myopia. It is true that the aetiology of high myopia is probably different from that of mild and moderate myopia. However, if we compare the percentages of children with myopia in Singapore with those in the West (19), the difference is striking:–

AGE	% WITH MYOPIA USA	SINGAPORE
7	3	19
9	7.5	17
11	12	27
13	15	40
15	17	48

Since myopia is caused by changes in the five components of the eye listed above, but the growth changes being inco-ordinated, it is obvious that these inco-ordinated changes are due to both environmental and genetic factors. There obviously will be genetic factors, but the low incidence of myopia in Singapore children 25 years ago compared to now seems to point to environmental factors, and the commonest change in the environment has come with greater competition in school learning. There is more vision fixed to books, i.e. concentrated near vision for long periods of time, and this may in the long run cause the increase in myopia. One also cannot discount genetic factors because in Singapore, it is common to see myopia running in families. I realise that many ophthalmologists believe that excessive reading will not contribute to myopia, but, I beg to disagree.

ACQUIRED EYE DISEASES:

Since eye infections are uncommon now in Singapore children, most of the eye diseases seen are secondary to other systemic disease, e.g. the collagen diseases, neoplasms such as leukaemia, bleeding disorders, etc.

CONCLUSION:

Therefore, the eye of the child among the 3 main ethnic groups in Singapore, has been affected by pathology, which has changed considerably over the years, with increasing improvement of the general health of the child. Within a period of 2 decades, the common infectious eye diseases and blindness due to malnutrition have been totally eradicated. But myopia, instead, is a greater problem.

REFERENCE:

1. *Wong, H.B. (1977): Changing Patterns of Paediatric Diseases in S.E. Asia Proc. 2nd Asian Congress of Paediatrics, Jakarta p. 79*
2. *Bietti, G.B. (1976): The Role of WHO in Blindness Prevention. Bulletin of the Pan American Health Organisation, 10, 65*
3. *Mabler, H.T. (1976): Foresight Prevents Blindness. Bulletin of the Pan American Health Organisation, 10, 62*
4. *Annual Reports of the Medical Department of the Colony of Singapore, 1946, 1947, 1948, 1949, 1950. Government Printing Office, Singapore.*
5. *Wong, H.B. (1971): Breast Feeding in Singapore. Choon Kee Press, Singapore.*
6. *Loh, R.C.K. (1964): The Decline in Incidence of Xerophthalmia in Singapore. Trans. Asia Pacific Acad. Ophthal. 2, 65*
7. *Loh, R.C.K. (1968): Changing Pattern of Blindness in Singapore. Trans. Asia Pacific Acad. Ophthal. 3, 441*
8. *Lim, K.H. (1975): Registration of Blindness in Singapore. 1950-1972. Singapore Med. J. 16, 62*
9. *Singapore Association for Blind (1978): Personal Communication*
10. *World Health Organisation: Epidemiological and Vital Statistics Report. 19, 441*
11. *Singapore Association for the Blind: Annual Report, 1971-1972.*
12. *Fraser, G.R. and Friedmann, A.I. (1967): The Causes of Blindness in Childhood. John Hopkins Press, Baltimore, USA*
13. *Report of Expert Committee on Immunisation, Ministry of Health, 1975.*
14. *Mann, I. (1966): Culture, Race, Climate and Eye Disease. Charles C. Thomas, Springfield, Illinois, USA*
15. *Annual Report (1974, 1975, 1976): School Health Services, Ministry of Health, Singapore*
16. *Pickford, R.W. (1965): Human Color Vision. In "Color Vision". Editors Renick, A.V.S. and Knight, J. Ciba Foundation Symposium. J and A Churchill. London*
17. *Health, G.G. (1964): Color Vision. In "Vision of Children". Editors Hirsch, M.J. and Wick, R.E. Hammard, Hammard and Co. Ltd., London.*
18. *Sorsby, A., Bengamin, B., Davey, J.B., Sheridan, M. and Tanner, J.M. (1957): Emmetropia and its aberrations. Medical Research Council Special Report Series, 293. Her Majesty's Stationery Office.*

AUSTRALIAN ABORIGINAL EYE HEALTH _ AND WHAT MUST BE DONE

Professor Fred Hollows
Director, National Trachoma and
Eye Health Program

The fact that Aborigines in Australia suffer poor eye health has been well attested, notably by Flynn, Mann, and most recently, the work of the National Trachoma and Eye Health Program.

During 1976 and 1977, the program, organised by the Royal Australian College of Ophthalmologists, visited more than 320 Aboriginal communities throughout rural Australia.

Almost 75,000 people were seen, of whom 49,781 were Aborigines. Of the latter, some 15,621 show signs of trachoma. Trachoma is a form of conjunctivitis with characteristic signs that are readily detectable. In childhood, these signs are follicles, papillae and limbal follicles, which proceed through the teenage years into adult life to scarring, Herbert's Pits, pannus and trachiasis.

The basic infection – often a non-troublesome conjunctivitis – is associated with an obligatory intra-cellular bacterium called *Chlamydia trachomatis.* This poorly antigenic "bug" appears to have the following attributes:

1. It is probably harboured without great inconvenience by many mammalian hosts;
2. Strains of chlamydia can be found in the linings of all portals of entry, that is in eyes, noses, ears, the respiratory system, the urethra, vagina, cervix and rectum.
3. Whether or not a particular clinical entity is associated with the presence of chlamydia appears to be due to a concurrence of environmental circumstances that favour their development. Some such diseases are non-specific urethritis, ocular trachoma, chlamydial respiratory syndrome and inclusion conjunctivitis.
4. It seems likely that humans will always harbour the organism.

For each portal of entry chlamydial disease, there is a cluster of conditions necessary for its development.

In the Australian context, trachoma, as an ocular portal of entry disease, requires:

1. Prolonged exposure to the organism;
2. Repeated exposure to the organism;
3. Eye to eye secretion swapping;
4. Hot, dry, bright climatic conditions;
5. Prolonged body contact, especially between women and children and children and children; and
6. Poor individual and group hygiene.

Trachoma manifests itself in childhood as a local immune overgrowth which brings in its train certain indirect deleterious consequences. Follicles – immune hypertrophy – are followed by scarring. A scarred conjunctival membrane appears to be a second-rate protecting membrane.

Eyes infected by chlamydiae are prone to repeated infections by other bugs.

Again, eye infections and injury, including surgery, have much more serious consequences than in eyes that are not affected by trachoma. There are three possible explanations:

1. The scarred trachomatous conjunctiva is not able to provide the normal mechanical and humoral protection;
2. The trachoma organism may directly inhibit macrophage activity, thus enhancing infection;
3. Previous trachoma infection may sensitise ocular tissue to subsequent non-trachomatous infections.

Trachoma provides the support pathology for most Aboriginal blinding corneal disease. Corneal disease is the major cause of incurable blindness in Australian Aborigines.

Monocular and Binocular blindness – National figures: Aborigines

	No. seen	Monocular	Binocular
All Aborigines	48,782	1,420 (2.9%)	844 (1.7%)
Aborigines over 60	3,160	658 (21%)	644 (20%)

One in every 21 Aborigines we have seen is blind in one or both eyes. When one looks at the figures for people over 60, more than four in ten people are blind in one or both eyes. In many communities and areas we visited, the figures were much worse.

The trachoma organism appears to be sensitive to many readily available antibiotics – antibiotics available for both systemic and topical application.

We have an organism that infects most portal of entry mucosal surfaces of most persons in most Aboriginal communities: the children in these communities show trachoma follicles – a good index of trachoma infection – and the adults have high rates of blindness due to trachoma-induced corneal disease.

We can treat people so that they reduce the rate at which they shed and spread trachoma organisms to and from each other. Probably the best method of giving the medication is by mouth, and it is probably better to treat all persons in trachomatous communities, with the exception of those under three months of age, and women who are pregnant.

All persons in trachomatous communities are part of the infectious pool. It is the infectious pool we must treat.

The success of trachoma treatment programs can readily be gauged by rescreening children after treatment, and measuring the extent to which follicles have been reduced. The aim of a treatment program is to achieve children without follicles and scarring – alliteratively, follicle-free five year olds.

In 1972, I was involved in a treatment program at Engonnia, in upper western NSW. There we have now almost achieved our objective of follicle free children. Ninety-five per cent of children there are free of follicles, and all children born since 1972 are free of follicles.

ENGONNIA TREATMENT TRIAL CHILDREN 2–11 YEARS

Follicle grade	Before treatment 1972		After treatment 1975	
	No.	Percentage	No.	Percentage
0	9	25	36	95
1	16	44	2	5
2	10	28	0	0
3	1	3	0	0
	36		38	

Trachoma treatment must be followed up. To prevent or lessen the effects of non-trachomatous added infections, topical antibiotics should be applied widely in the communities, especially in those whose anterior eyes are prone to damage, that is in those with significant trachoma scarring, and especially during periods of acute infective conjunctivitis and conjunctivo-keratitis. Epidemics of acute conjunctivitis and conjunctivo-keratitis frequently sweep through trachomatous Aboriginal communities.

TRACHOMA SCARRING IN ABORIGINAL COMMUNITIES

Age	Total number	Scarring 1, 2, & 3	Scarring 2 & 3
Under 11	19,708	2,218 (11%)	573 (2.9%)
Over 11	29,055	14,693 (51%)	6,125 (21%)
Over 60	3,160	2,570 (81%)	1,713 (54%)

As Professor Barrie Jones, one of the world's leading authorities on trachoma, said in his report to the Royal Australian College of Ophthalmologists on the activities of the trachoma program:

> "The provision of family-based immediate topical chemotherapy of every red or sticky eye is an objective that can be achieved only within the context of a substantial community-based move towards health, arising within the Aboriginal communities."

Of course trachoma is one of the environmental diseases, and environmental disease must be tackled at its base. Aborigines have traditionally moved in small groups over vast areas. Their culture has provided them with the hygiene rules necessary for small mobile groups. For such small groups there is a small infectious pool and only infrequent cycling disease.

At present, however, most Aborigines live in large, stationary groups, forming large infectious pools and suffering constant cycling disease.

They do not have incorporated in their style of living the hygiene rules necessary for large stationary groups.

A large group, for example, needs to be able to utilise certain basic health amenities. Three such amenities are:

1. Water for washing at the rate of at least 100 litres per person per day;
2. Separated sleeping and breathing space;
3. Hygienic above-ground accommodation.

The provision of these amenities is an urgent necessity, but no less urgent is the accomplishment of the utilisation of these amenities.

Therefore to deal with trachoma we have three requirements:

1. The need to mass treat trachomatous communities.
2. The need for follow-up and for readily available treatment for secondary conjunctivitis; and
3. The provision of basic health amenities and the utilisation of these amenities.

How can this be done?

It will certainly not be done by cultural invasion, by attempts to superimpose white methods and techniques willy-nilly on the Aboriginal communities. It will not be achieved by demanding of Aborigines a pattern of activisation or an order that is foreign to them.

We must use the existing group structures!

We must find out who and what the Aboriginal organisation is around and about the Aboriginal communities. This requires detailed knowledge from people in the group, so local personnel must be employed from the start.

Having identified the groups, the first duty of liaison workers, there must be consultation, discussion, explanation, familiarity and empathy with the group.

This will take time and effort and it will often come about in a way both strange and inconvenient to the white health resource person but it is essential that the authority structure of the target groups be "levelled with" completely.

Aborigines will quite quickly establish the bona fides of any health agent if he or she sits down and talks with them on their terms.

Then follows identification with and commitment to the group. Once this commitment is made, and the Aboriginal structure understands and agrees the program may become the base of a community based health service.

In this, the National Trachoma and Eye Health Program agrees with and is carrying out the policy of the Government. As the Minister for Aboriginal Affairs, Mr Ian Viner, said last year, there will be no headway made in improving Aboriginal health

> "until the Aboriginal people are involved in solutions to the basic health needs of their life at the grass-roots level".

Again, as Mr Viner has said, the basic principles must be:

> "self-management, self-sufficiency and Aboriginality".

The communities will choose and supervise Aboriginal health workers, chosen, as a leading trachomatologist, Chandler Dawson, has said

> "by cultural and other local attitudes".

Again, however, there is often a great reluctance on the part of individuals to subject themselves to prolonged "whitefella" type vocational training, so, as Chandler Dawson has said –

> "Training for village health workers must be short, simple, and carried out in the community, since there may be a high rate of turnover of these workers".

This high turnover serendipitously provides a wide base of health knowledge.

It is insufficient that Aboriginal health workers be employed and trained. The health service must be community based. That is, resources must be directed by and administered by the Aboriginal people. Aboriginal people must decide policy, give directions and utilise the necessary white resource personnel.

As Chandler Dawson has put it:

> "In the final analysis, trachoma control programs should not be considered only in the narrow context of preventing blindness but as the beginning of a sustained effort to deliver eye health care to rural communities."

Everywhere the National Trachoma and Eye Health Program has moved, we have attempted, albeit insufficiently, to proceed via consultation, discussion, explanation, familiarity and empathy. Whatever the trachoma program has achieved has been due to this.

In 1978, the trend in Aboriginal health is moving from earlier, mostly unsuccessful attempts to train Aborigines to use white health structures into methods that involve whole communities in their own health care.

It is good to see community-based Aboriginal health services taking over or beginning to take over in places such as:

Redfern, Sydney
Kempsey, NSW
Melbourne
Adelaide
Port Augusta
Perth
Fitzroy Crossing
Carnarvon
In the Pitjantjatjarra homelands
At Alice Springs
Papunya
Utopia
Townsville
Brisbane

REGISTER OF FINANCIAL MEMBERS

ADAMS (Wyatt), Mrs. C.	2 Pearl Place, Blackman's Bay, Tas. 7152	
ALEXANDER (Jones), Mrs. J.	69 Commonwealth St., Bathurst, NSW 2795	063-314 111
ALLWORTH (Sheldon), Mrs. P.	165 Condamine St., Balgowlah, NSW 2479	949 2774
ANGUS (Manning), Mrs. J.	P.O. Box 170, Manuka, ACT 2603	73 2345
ARMSTRONG (Coddington), Mrs. J.	94 Cabbage Tree Lane, Fairy Meadow, NSW 2519	84 2629
ARTHUR, Miss J.	147 Kent St., Epping, NSW 2121	86 2695
ASHTON, Miss C.	14 Gordon Street, Clontarf, NSW 2093	
BALFOUR, Miss B.	48 Hilda St., Shenton Park, WA 6008	81 2980
BALL, Miss V. M.	4/51 Domain St., South Yarra, Vic. 3141	267 1549
BARBOUR (Large), Mrs. T.	P.O. Box 18, Evandale, Tas. 7212	
BARLOW, Miss S.	16 William St., Botany, NSW 2019	
BEAUMONT, Mrs C.	9 Girilang Ave., Vaucluse, NSW 2030	337 4442
BERASTROW, Mrs. J.	1A/5 Lower Boyle St., Cremorne, NSW 2090	
BINOVEC (Hansor), Mrs. M.	27 Fraser Rd., Normanhurst	48 0274
BOAG, Miss E.,	16/7 Manningham St., Parkville, Vic. 3052	329 0272
BOORNE (Odillo-Maher)	2 Gladstone Ave., Turramurra, NSW 2074	
BRENNAN, Miss C.	"Widgeree", Warren, NSW 2824	
BRENT (Lipman), Mrs. L.	"Varuna", Armidale, NSW 2350	75 0134
BRIERLY, Mrs. L.	5 Bristol Court, Glen Iris, Vic. 3146	20 1033
BRODIE (Paul), Mrs. B.	11a Howard St., Randwick, NSW 2031	399 7840
BROWN (Sullivan), Mrs. M. A.	1 Charman Ave., Maroubra, NSW 2035	349 4650
BROWN, Miss S.	2/3 New Beach Rd., Darling Point, NSW 2027	328 6421
BROWNFIELD (Sadler). Mrs. R.	227 Mill St., Ballarat, Vic 3350	32 1655
BROWNING (Reggio), Mrs. R.	10/67 Broome St., Maroubra, NSW 2035	661 6682
BUCHANAN, Miss J.	8/19 Roddards Rd., Wollstonecraft, NSW	
BURGESS (Lake), Mrs. G.	Lot 4, Harley St., Bendigo, Vic. 3550	43 2028
BURKE (Williams), Mrs. K.	"Dunreath", Loomberah, NSW 2340	
BURR (Morris), Mrs. A.	31 Bolingbroke Grove, Toorak Gardens, SA 5065	332 1291
CAMERON, Mrs. V.	50 Elizabeth St., Malvern, Vic. 3144	20 1425
CARTER, Miss M.	P.O. Box 470, South Yarra, Vic. 3141	26 5562
CHILDS, Miss C.	West Australia	
CLARK (Innes), Mrs. S.	455a Bronte Road, Bronte, 2024	387 1459
CLERK, Miss J.	7 Star St., Sandy Bay, Tas. 7005	34 4251
CLUGSTON, Mrs. H.	7 Silex Rd., Clifton Gardens, NSW 2088	977 4377
COLLINS, Mrs. L.	16 Carolyn St., Hampton, Vic. 3188	598 0371
COLLINS, Miss S.	237 Blaxcell St., Granville 2142	637 2510
CORNELL (Smith), Mrs. E.	58 Wycombe Rd., Neutral Bay, NSW 2089	90 3610
CORT, Miss S.	5 McMillan Ave., Winston Hills, NSW 2153	629 8330
COULSON (Knight), Mrs. C.	94 Sydenham Rd., Norwood, SA 5061	
COWLE, Miss S.	27 Mills St., Asquith 2078	
DALY (Tindale), Mrs. S.	6 Dommett Place, Kenmore, Brisbane, Qld. 4069	378 3588
DALZELL, Joseph	78 Beryl St., Coffs Harbour, NSW 2450	
DENNISON (Lewin), Mrs. B.	2 Valley View Cres., North Epping, 2121	86 2270
DEVIDAS (Ross), Mrs. B. J. .	153 Neale St., Bendigo, Vic. 3550	
DICKINSON, Mrs. A.	12 Mavis St., St. Lucia, Qld. 4067	371 4617
DIMOS, Miss C.	4 Doulton St., Beacon Hill, 2100	938 4471
DOSSOR (Sterling Lewis), Mrs. F.	84 Milray Ave., Wollstonecraft, NSW 2065	43 4384
DOUGAN (Hall), Mrs. M.	9 Highgate Rd., Lindfield, NSW 2070	467 1613
DOYLE (Kirkland), Mrs. M.	15 Burrabirra Ave., Vaucluse, NSW 2030	337 2810
DUGGAN (Colman), Mrs. T.	49 Amore St., Hornsby, NSW 2077	
DUNCAN (Hillsdon), Mrs. E.	15 Seamen St., Greenwich, NSW 2065	
DUNLOP (Maguire), Mrs. P.	61 Carrington Pde., New Lambton Heights, NSW 2305	57 2019
EARL, Mrs. M.	65 The Esplanade, French's Forest, NSW 2086	452 4661
EDWARDS (Woolley), Mrs. K.	12 Coree Court, Mooloolabah, Qld. 4557	44 1639
ELLIOT, Miss V.	4 Palmerston Place, Seaforth, NSW 2092	94 6200
ERBY (Morey), Mrs. J.	5 Oratava Ave., West Pennant Hills, NSW 2120	871 8954
ERIKSON (Pyrke), Mrs. P.	52/88 Wycombe Rd., Neutral Bay, NSW 2089	90 3088
EYRES, Miss M.	6/63 Darling Point Rd., Darling Point, NSW 2027	32 2220
FENELEY (Knight), Mrs. M.	52 Park St., Bulli, NSW 2516	84 7978
FERRARO, Mrs. K.	37 Red Bluff Rd., Black Rock, Vic. 3193	598 2860
FINKLESTEIN, Miss S.	19 Burton St., Mosman 2088	960 3068
FITZGERALD, Miss P.	44 Wyralla Rd., Eway Bay, NSW 2228	
FITZSIMMONS (Henning), Mrs. J.	10/20 Dutruc St., Randwick, NSW 2031	
FORREST, Miss P.	33 Ashgrove, Bingley, West Yorkshire, England	
FRILAY, Mrs. P.	31 Wilkinson St., Flynn, ACT 2615	58 5254
FRANCIS (Bulford), Mrs. P.	35 Richardson St., West Perth, WA 6005	21 4650
FYFFE, Mrs. J.	116 Kooyong Rd., Armadale, Vic. 3143	20 3965
GARFIT, Mrs. J.	54 Grayson St., Hackett, ACT	41 3493
GHENT, Miss R.	"Rosehill", Bonds Rd., Lower Plenty, Vic. 3093	41 4347

GILMORE (Croker), Mrs. D.	29 Carbeen Ave., St. Ives, NSW 2075	44 2155
GORDON (Blaubaum), Mrs. V.	5 Stonnington Place, Toorak, Vic. 3142	20 5568
GRAY, Miss Sharon	21 North Neringah Ave., Wahroonga, NSW 2076	48 3069
GREEN (Palmer), Mrs. E.	Boothenbar Rd., Dubbo, NSW 2830	
GREEN, Mrs. J.	98 Glyndon Rd., Hartwell, Vic. 3124	29 6225
GREGG, Miss S.	342 Bobbin Head Rd., North Turramurra, NSW 2074	44 1139
GREGORY (McDonald), Mrs. M.	30 Infantry Pde., Holdsworthy, NSW	611 1445
GYAW, Mrs. M.	72 Kenmore St., Mont Albert, Vic. 3127	88 5109
HALL (Jones), Mrs. P.	13 Frew St., Fullarton, SA 5063	79 1138
HANDLEY, Miss M.	63 Murray St., Tamworth, NSW 2340	067-66 4485
HARRIS, Miss A.	16 Hoadley Ave., Frankston, Vic. 3199	783 1832
HAYNES, Miss B.	6 Loloma Court, Burwood, Vic. 3125	29 5762
HEINZE, Mrs. G.	1 Selwyn Court, Toorak, Vic. 3142	20 6437
HENDERSON (Massey-Greene), Mrs. J.	17 Avenal Gardens, Menindie, SA 5081	44 7969
HERLIHY, Miss A.	78 Wolseley Rd., Point Piper, NSW 2027	36 7139
HICKS, Mrs. M.	7 Arnold Rd., East Brighton, Vic. 3187	92 5935
HITCH (Metcalfe), Mrs. A.	158 Riverview Rd., Clareville, NSW 2107	918 9852
HOBSON, Miss K.	74 Bendigo St., Richmond, Vic. 3121	429 2694
HOGAN, Mrs. W.	West Australia	
HOPKINS (Knoblanche), Mrs. A.	19 Marr St., Wollongong, NSW 2500	28 5118
HORTON, Miss F. M.	26 Murdoch St., Cremorne, NSW 2090	90 1856
HOWARTH, Mrs. P.	35 Bromborough Rd., Roseville, 2069	46 1465
HUNTER, Mrs. J.	1/140 Warringul Rd., Mentone, Vic. 3194	93 3724
JOHNSON, Mr. G.	1 Francis St., Fairfield, NSW 2165	
JOLLY (Heard), Miss N.	110 Copeland Rd., Beecroft, NSW 2119	84 2087
JONES, Miss L.	45 Cecil Ave., Castle Hill, NSW 2154	
KELLY, Miss J.	1/211 Military Rd., Neutral Bay, NSW 2089	
KIERNICKI, Mrs. R.	31 Bellinger Rd., Ruse, NSW 2560	
KIRBY, Miss J.	Andrew House, 1 Wickham Tce., Brisbane, Qld. 4000	31 1597
KRISTIC (Hamburg), Mrs. B.	26 Wellington Ave., Beaumaris, Vic. 3193	99 1773
LANGRELL, Miss C.	12 Agnes St., Strathfield, NSW 2135	642 2789
LAWSON, Mrs. A.	37 Kendall St., Pymble, NSW 2073	498 4984
LEWIS, Miss M.	254 St. George's Terrace, Perth, WA 6000	22 3454
LONGHURST, Mr. E.	16 Timothy St., Hurstville, NSW 2220	570 1498
LOOBY, Miss R.	16 Victoria Rd., Pennant Hills, NSW 2120	
LOVE, Miss S.	1 South St., Strathfield, NSW 2135	398 1001
LOWIS (Cameron), Mrs. M.	C/- G. H. Lowis Snr., 14 Cornel Rd., High Heaton, Newcastle-upon-Tyne, NE7-7PT. England.	
McCORMACK, Miss G.	210 Yarranabbe Gardens, 87/98 Yarranabbe Rd., Darling Point, NSW 2027	32 2517
McCORQUODALE, Miss D.	2/104 Kurraba Rd., Neutral Bay, NSW 2089	909 1731
McDOUGALL (MacGee), Mrs. B.	15 Princess St., Rose Bay, NSW 2029	371 9235
MACFARLANE (Fleming), Mrs. A.	3 Brush Rd., Eastwood	85 6895
McINDOE (Rickard), Mrs. A. K. R.	38 Bryson St., Canterbury, Vic. 3194	83 2139
McKENZIE (Santamaria), Mrs. L.	11/14 Warringal Rd., Mentone, Vic. 3194	
MACLARN, Miss L.	Woodbine Close, 1/85 Orange Rd., Toorak, Vic. 3142	
McNAMARA (Nicholson), Mrs. T.	22 Saxby Rd., Glen Iris, Vic. 3146	25-2460
MacPHERSON (Woods), Mrs. B.	2 Keith St., Roseville, NSW 2069	46 3632
MAHONEY, Miss A. M.	5/36 Power St., Hawthorn, Vic. 3122	86 5311
MANCHEE (Hannah), Mrs. C.	221 Hopetoun Ave., Vaucluse, NSW 2030	337 5967
MARSHALL, Miss B.	40 Waverley St., Bondi Junction, NSW 2022	389 6420
MARTIN (Whitten), Mrs. J.	3 Porters Rd., Kenthurst, NSW 2154	
MARTIN, Miss L.	14 Kenilworth Cres., Glen Waverley, Vic. 3150	232 4046
MERRICK, Mrs. F.	54 Clanville Rd., Roseville, NSW 2069	46 5066
MERRINGTON, Mrs. S.	19 Boonal St., Baulkham Hills, NSW 2153	624 6351
MESSERLI, Mrs. U.	36 Kardinia Rd., Mosman, NSW 2088	969 6476
MEYER (McKern), Mrs. K.	7/18 Collingwood St., Drummoyne, NSW 2047	81 2623
MIKUS (Southwell), Mrs. A.	4/102 Brighton Rd., Elsternwick, Vic. 3185	91 5469
MOLONEY, Miss J.	1 Parkside Ave., Box Hill, Vic. 3128	89 2324
MONTEATH, Miss P.	41 Scenic Drive, Merewether, NSW 2291	63 1199
MOORS, Miss S.	28 Ross St., Newport, NSW 2106	99 1405
MOUNTFORD, Miss L.	2 Rose Ave., Connells Point, NSW 2221	546 4728
MULHALL, Mrs. L.	181 Carrick Drive, Gladstone Park, Vic. 3043	
NEVILL, Miss M.	21 Iris St., Burwood, Vic. 3125	288 2069
NEWTON (Haddon-Smith), Mrs. J.	167 Kooyong Rd., Toorak, Vic. 3142	20 6177
NORTON, Mrs. P.	383 Napier St., Fitzroy, Vic. 3065	419 3559
O'DONNELL, Miss S.	C/- Dr. D. J. McAuliffe, P.O. Box 229, West Perth, WA 6005	
O'SULLIVAN, Miss G.	'Farnham House', 3 Farnham Ave., Randwick, 2031	393 1629
PARDEY, Miss S. J.	7 Bangalla St., Warrawee, NSW 2074	48 6799
PEBERDY, Mrs. J.	61 Wentworth Ave., Killara, NSW 2071	498 8597
PEOPLES, Miss M.	351 Chloride St., Broken Hill, NSW 2880	5135
PETTIGREW, Miss A.	187 Boundary St., East Roseville, NSW 2069	407 1458
PHILLIPS, Mrs. A.	32 Northcote Ave., Killara, NSW 2071	498 1593

PHILPOTTS, Miss A.	7 Trafalgar Ave., Roseville, NSW 2069	46 5125
PITTAR, Mrs. H.	69 Stanhope Rd., Killara, NSW, 2071	46 2722
PRIOR, Mr. C.	23 Frederick St., Ferntree Gully, Vic. 3156	758 5548
PUNCH (Dwyer), Mrs. A. M.	53 Glover St., Mosman, NSW 2088	90 5065
RICHARDSON, Miss B.	14 Meadow Grove, Balwyn, Vic. 3103	80 5761
RIVERS (Osborn), Mrs. M.	14 Acheron St., Doncaster, Vic. 3108	602 0211, 848 8385
ROBINSON (Muir), Mrs. C.	8 Seeana Place, Belrose, NSW 2085	451 5977
ROBINSON, Mrs. M.	9 Pembroke Rd., Mooroolbark, Vic. 3183	725 6127
RONA, Mrs. A.	83 Manning Rd., Double Bay, NSW 2028	36 7086
ROSE, Miss K.	9 Reynolds St., Balmain, NSW 2041	
ROSS, Miss J.	302/176 Glenmore Rd., Paddington, NSW 2021	(message service) 31 8681
ROYSTON, Miss S. K.	4/51 Domain St., South Yarra, Vic. 3141	267 1549
RUSSELL, Miss J.	72 Holdsworth St., Woollahra, NSW 2025	32 4689
SHANAHAN, Miss K.	28 Bolaro Ave., Gymea, NSW 2227	524 4357
SHEPPARD, Miss J.	1 Hunter Ave., Black Rock, Vic. 3193	99 4288
SMITS (Gers), Mrs. J.	8 Lido Court, Para Hills, SA 5096	264 6195
SPENCE (Gray), Mrs. S.	345 Bobbin Head Rd., North Turramurra, NSW 2074	
SPILIOPOULOS (Blake-Lane), Mrs. S.	Hotel Pelops, Olympia, Greece.	
SPROD, Miss D.	Royal Ave., Burnside, SA 5066	31 7424
SMITH, Mrs. S.	Victoria	
STANLEY (Kelly), Mrs. S.	'Locking', 49 Cobran Rd., Cheltenham, NSW 2119	869 2680
STEAD (Wood), Mrs. G.	1 Kerrie Rd., Dundas, NSW 2117	630 1083
STEWART, Miss J.	5/26 Stonehouse Ave., Camden Park, SA 5038	
STRENTZ, Mrs. J.	192 Seaford Rd., Seaford, Vic. 3198	786 3126
SULLIVAN, Miss E.	Arthur St., Beaudesert, Qld. 4285	
SYME, Miss A.	13/3, Boston Rd., Balwyn, Vic. 3103	836 3056
TAIT, Miss S.	14 Redgum Ave., Killara, NSW 2071	498 6079
TAYLOR, Miss E.	45 Malmsbury Rd., Kew, Vic. 3101	
TAYLOR, Miss J.	11 Cheltenham St., Highgate, SA 5063	74 1295
TERRELL, Miss A.	35 Richardson St., West Perth, WA 6005	450 2568
TIPPER (Morrice), Mrs. J.	149 Swan St., Morpeth, NSW 2321	33 5580
TOOGOOD (Lloyd), Mrs. P.	9 Swallow Rock Drive, Grays Point, NSW 2232	525 6145
TURNBULL, Mrs. R.	49 Colson Cres., Monteray, NSW 2217	588 3205
VINCE, Mrs. A. M.	6/8 Buller St., Artarmon, NSW 2064	411 5940
WALCH (Brown), Mrs. B.	12 George St., Unley Park, SA 5061	71 3754
WARHAM, Miss P.	C/- J. F. Finch, 4 Eltham St., Wembley Downs, WA 6019	
WATTS, Miss A.	13 Churchill Cres., Allambie Heights, NSW 2100	93 5011
WEBSTER, Miss V.	57 Yathong Rd., Caringbah North, NSW 2229	525 1520
WEIGHTMAN, Miss J.	10 Willawa St., Balgowlah Heights, NSW 2093	94 5645
WELLINGTON, Miss R.	5 Fiona St., Wahroonga, NSW 2076	48 1090
WHITNEY (Henderson), Mrs. R.	4 Mark Rd., Goonellabah, NSW	24 1022
WILDERS, Mrs. S.	124 Clyde St., Mosman Park, WA 6012	
WILKINSON, Miss R.	104 Montague St., Newtown, Tas. 7008	
WILLIAMS, Mrs. A.	15 Rosalyn St., Hawthorn East, Vic. 3123	82 2120
WILLIAMS (Bode), Mrs. B.	55 Philip St., Vermont, Vic. 3133	874 1752
WILLIAMS (Thompson), Mrs. J.	10 Jerome Ave., Winston Hills, NSW 2153	639 0465
WILSON (Bradshaw), Mrs. J. A.	9 Stephen St., Balmain, NSW 2041	827 2305
WISTER, Miss M.	248 Barrenjoey Rd., Newport, NSW 2106	
WISTER, Mrs. P.	8 Malakoff St., North Caulfield, Vic. 3161	509 5751
WRIGHT, Miss J.	(overseas)	
WONG Mrs. L.	37 Devon St., North Epping, NSW 2121	86 6562
WULFF (Hopkins), Mrs. J.	4 Antoinette Place, Warrawee, NSW 2075	
WRIEDT, Miss J.	32 Old Lower Plenty Rd., East Rosanna, Vic. 3084	435 9684
YAP (Cumines), Mrs. J.	2 Murrumbidgee Ave., Sylvania Waters, NSW 2224	522 9553

FELLOWS

CRAIG, Mrs. D. (Mann)	20 Merton St., Ivanhoe, Vic. 3079	49 1352
HAWKESWOOD, Miss H.	42 Newcastle St., Rose Bay, N.S.W. 2029	371 5394
LANCE, Miss P.M.	67 Bower St., Manly, N.S.W. 2095	977 2897

ASSOCIATE MEMBERS

BAIRD (Fraser), Mrs. S.	P.O. Box 1115, Canberra City, A.C.T. 2601
BALDICK, Miss J.	5 "Coromandel", Sutherland Cres., Darling Pt., N.S.W. 2027
BUCHANAN, Mrs. D.	8 Truscott St., Campbell, A.C.T. 2061

BURFITT-WILLIAMS (Davies), Mrs. G.	38 Wentworth Ave., Vaucluse, N.S.W. 2030	337 2922
CADWALLADER (Crawley), Mrs. S.	68 Springdale Rd., Killara, N.S.W. 2071	
CAMERON, Mrs. V.	50 Elizabeth St., Malvern, Vic. 3144	
COLLETT (Anderson), Mrs. S.	9 Crawford Crt., Dartmouth, Vic.	72 4262
DENNISON (Lewin), Mrs. B.	2 Valley View Cres., North Epping, N.S.W. 2121	86 2270
FLEMING, Miss K.	Orthoptic Clinic, North Canterbury Hospital, Christchurch, New Zealand.	
FORD (Allen), Mrs. Y.	10/73 Darley Rd., Manly, N.S.W. 2095	977 4598
GLUCKMAN, Mrs. J.	17 Wandeen Ave., Beecroft, N.S.W. 2092	84 6293
GRAHAM (Hall), Mrs. J.	"Fernhill", Adjungbilly, N.S.W. 2737	
HUDSON (Doghlan), Mrs. J.	7 Drumalbyn Rd., Bellevue Hill, N.S.W. 2023	36 4680
MARSHALL (Wilkinson), Mrs. A.	C/- P.O. Box 858, Alic Springs, N.T. 5750	2 1477
McDADE (Bryden-Brown), Mrs. L.	32 Castle Circuit, Seaforth, N.S.W. 2092	94 1573
O'CONNOR (McKillop), Mrs. B.	8 Keirnan Ave., Gwynneville, Wollongong, N.S.W. 2500	29 5434
ROTELLI, Mrs. M.	C/- Robertson Darling & Wolfenden, G.P.O. Box 1906 Sydney, 2001	
SMITH (Engwerda), Mrs. J.	95 Daglish St., Wembley, W.A. 6074	
TINWORTH, Mrs. J.	72 Peronne Ave., Clontarf, N.S.W. 2093	
VICARS, Mrs. S.	1602 Pacific Highway, Wahroonga, N.S.W. 2076	48 1311
WOODHILL, Mrs. R.	15 Wigram Rd., Austinmer, N.S.W. 2514	

ORTHOPTIC BOARD OF AUSTRALIA

Chairman	Dr. E. F. McGuinness
Secretary	Dr. W. E. Gillies
	Dr. E. M. Gregory
	Dr. J. Heinze
	Dr. G. C. Hipwell
	Dr. A. B. L. Hunyor
	Dr. P. J. Stobie
	Dr. J. Norton Taylor
	Dr. R. Westmore
	Dr. D. C. Wilson
	Mrs. V. Gordon
	Miss P. Lance
	Miss H. Hawkeswood
	Mrs. P. Wister

ORTHOPTIC BOARD OF AUSTRALIA – NEW SOUTH WALES BRANCH

Chairman	Dr. R. McGuinness
Secretary	Dr. R. Higgins
	Dr. A. B. L. Hunyor
	Dr. I. Kennedy
	Dr. F. Martin
	Dr. M. Strathdee
	Miss S. Brown
	Miss P. Lance
	Miss H. Hawkeswood

ORTHOPTIC BOARD OF AUSTRALIA – VICTORIAN BRANCH

Chairman	Dr. W. E. Gillies
Secretary	Dr. J. Norton Taylor
	Dr. R. West
	Dr. J. Heinze
	Dr. J. Reich
	Mrs. V. Gordon
	Miss A. M. Mahoney
	Mrs. P. Wister

ORTHOPTIC ASSOCIATION OF AUSTRALIA – NSW BRANCH

President	Shayne Brown
Vice President	Jeanette Yap
Secretary	Sandra Tait
Treasurer	Susan Gregg
Council	Elaine Cornell
	Patricia Dunlop
	Patricia Lance (Head of School of Orthoptics)

ORTHOPTIC ASSOCIATION OF AUSTRALIA – VICTORIAN BRANCH

President	Leonie Collins
Vice President	Beth Richardson
Secretary	Jenny Fyffe
Asst. Secretary	Amanda Harris
Treasurer	Barbara Haynes
Librarian	Jennifer Hunter

HONORARY MEMBERS

Fenton, Dr. F. G.	20 Collins St., Melbourne, Vic. 3000
Hipwell, Dr. G.	C/- Medical Centre, 66 Pacific Highway, St. Leonards, NSW
Livingstone, Air Marshall Sir Phillip	Maple Bay, R.R. Duncan, B.C. Canada
Lowe, Dr. Ronald F.	82 Collins St., Melbourne, Vic. 3000
Lyle, Mr. T. Keith	6 Chesterfield St., Mayfair, London, U.K.
Mein, Miss Joyce	Park View Cottage, Melmertoy, Ripon, Nth. Yorks. H945HA, U.K.
Pockley, Dr. J. A.	233 Macquarie St., Sydney, NSW 2000
Retalic, Mrs. L.	595 Glynburn Rd., Beaumont, SA 5066
Russell, Miss E.	281A Edgecliff Rd., Woollahra, NSW 2025
Ryan, Dr. H.	100 Collins St., Melbourne, Vic. 3000
Spooner, Miss V.	52 Mountbatten Close, Roath Park, Cardiff, CF2 5QH U.K.
Sterling-Levis, Dr. M.	324 Marrickville Rd., Marrickville, NSW 2204
Wesson, Miss E.	8 Mayfield Court, 59A Mayfield Rd., Moseley, Birmingham, B139HS, U.K.

ORTHOPTIC PRACTICES AND HOSPITAL CLINICS

New South Wales

Allambie Heights			
Spastic School Centre Industries		Mrs. M. Binovec Mrs. M. Binovec	451-5555
Armidale			
Private		Mrs. L. Brent 137 Dangar St., Armidale, 2350	72-3655
Armidale & District Hospital		Mrs.L. Brent	
Auburn			
Sponsor	Drs. Milverton, Steiner & Pickering 16 Mary St., 2144	Miss K. Rose	
Balmain			
Balmain & District Hospital,	Booth St., 2401	Mrs. E. Duncan	827-3211
Bankstown			
Private		Mrs. J. Wulff 17 Kitchener Pde., 2200	709-1910
Bankstown & District Hospital,		Miss Susan Collins	70-0444
Bathurst			
Sponsor	Dr. T. D. Leckie, 79 Bentinck St., 2795	Mrs. J. Alexander	31-3989
Blacktown			
Private		Miss S. Cort Suite 207A, Town Centre, 32 Campbell St., 2148	671-1651
Bondi Junction			
Private		Miss G. McCormack 253 Oxford St., 2022	389-0488
Locum		Miss C. Manchee	
Sponsor	Drs. Cher and Robinson 253 Oxford St., 2022	Mrs. J. Fitzsimmons Miss R. Wellington Miss C. Dimos	
	Dr. R. McGuiness	Miss K. Rose	
Broken Hill			
Broken Hill & District Hospital, 2880		Miss M. Peoples	
Burwood			
Private		Mrs. A. Macfarlane "Murray Place" 127 Burwood Rd., 2134	747-2518
Sponsor	Dr. D. V. Campbell, 86 Shaftesbury Rd., Burwood	Mrs. J. A. Wilson	74-8470
Cabramatta			
Community Health Centre		Mrs. J. Williams	
Camperdown			
Royal Alexandra Hospital for Children,	Bridge Rd., 2050	Mrs. M. Gregory Miss S. Gregg	692-6460
Royal Prince Alfred Hospital	Missenden Rd., 2050	Miss M. Eyres	51-0444
Campbelltown			
Private		Mrs. A. M. Vince Suite 2, 7 Lithgow St., 2560	046- 25-2422

Caringbah			
Private		Miss J. Cumines 3 NRMA Building. 30 President Ave.	525-5100
Castle Hill			
Sponsor	Dr. Hopkins & Partners, 268 Old Northern Rd., 2154	Mrs. S. Merrington	634-6364
	Baulkham Hills Health Centre	Miss R. Looby	
Chatswood			
Private		Mrs. M. Brown Chatswood Chambers, Help St., 2067	412-2130
	Locum	Miss L. Mountford	
Coffs Harbour			
Sponsor	Dr. C. Housego Fitzroy Chambers, Park Ave., 2450	Mr. J. Dalzell	52-1221
Crows Nest			
Mater Misericordiae Hospital, Pacific Highway, 2065		Mrs. B. Macpherson	929-7022
Croydon			
Western Suburbs Hospital, Liverpool Rd., 2132		Mr. E. Longhurst	747-5311
Darlinghurst			
St. Vincents Hospital, Victoria St., 2010		Miss M. Eyres	339-1111 Ext.2026
Dee Why			
Private		Miss P.M. Lance, Miss H.R. Hawkeswood	
		Locum: Miss C. Ashton 14/1 Redman Rd., 2099	98-9503
Eastwood			
Sponsor	Dr. F. Martin 208 Rowe St., 2122	Miss D. McCorquodale	85-0514
Epping			
Private		G. Stead, B. Dennison, J. Arthur Mrs. S. Stanley, Mrs. A. Hitch 41 Rawson St., 2121	86-5477 86-5855
Fairfield			
Sponsor	Dr. T. Keldoulis 4 William St., 2165	Miss R. O'Neil	724-4811
	Dr. J. Trenerry, 77 Ware St., 2165	Mr. E. Longhurst	
Community Health Centre		Joy Williams	
Gordon			
Private		Mrs. J. Wulff 5/20 St. Johns Ave., 2072	498-6887
Gosford			
Private		Mrs. A. Lawson William Court, William St., 2250	
Grafton			
Sponsor	Dr. M. Waugh 13a Prince St., 2460	Mrs. D. Whitaker	42-2476
Hornsby			
Private		Mrs. N. Jolly & Miss R. Looby 32 Florence St., 2077	477-2494
Hornsby & District Hospital, Palmerston Rd., 2077		Miss R. Looby	476-3622
Hurstville			
Private		Miss C. Muir 9/346 Forest Rd., 3220	451-5977 57-1203
Kempsey			
Sponsor	Dr. C.J. Housego 80 Belgrave St., 2440	Mr. J. Dalzell	62-6046
Kogarah			
Sponsor	Dr. V. S. Kotur, Suite 5, 1st Floor, Commonwealth Bank Building, 104 Railway Pde.	Miss V. Webster	

Liverpool			
Sponsors	Drs. E. Gregory & J. Kevin Specialists' Medical Centre, 17 Moore St., 2170	Miss S. Gray	602-8066
	Liverpool Hospital, Elizabeth St., 2170	Mrs. M. Binovec	600-0555
	Community Health Centre	Miss K. Rose	
Maitland			
Sponsor	Drs. McClelland, Heron & Gibbons, High St., 2320	Mrs. J. Tipper	
Manly			
Sponsor	Dr. Y. Barnard, 2 Wentworth St., 2095	Mrs. P. Erikson	977-5728
	Royal Far West Children's Health Scheme, 18 Wentworth St., 2095	Mrs. A. Mikus	977-4377
Marrickville			
Sponsor	Dr. M. Sterling-Levis 324 Marrickville Rd., 2204	Mrs. M. Beaumont	560-8589
	Merrylands Community Health Centre	Mrs. A. Macfarlane Miss F. Horton	682-3133
Miranda			
Sponsor	Dr. M. Silva, 17 Urunga Pde, 2228	Mrs. P. Toogood	525-4688 525-4747
Mona Vale			
Sponsor	Dr. J. Killick, 174 Pittwater Rd., 2103		99-2918
Mosman			
Spastic School		Mrs. M. Binovec	969-1666
Mt. Druitt			
Polyclinic		Miss S. Cort	625-6000
Newcastle			
Private		Mrs. P. Dunlop 66 King St., 2300	049- 2-3466 049- 2-3535
Sponsors	Drs. G. Stuckey & C. Joneshart, C.M.L. Building, Hunter St., 2300	Miss P. Monteath	049- 2-2029 049- 2-4429
	Dr. W. Porter, "Athcourt", Watt Street, 2300	Miss P. Monteath	049- 2-4380
	Royal Newcastle Hospital, Paediatric Dept. Learning Disorders Clinic	Miss P. Monteath Mrs. P. Dunlop Consultant Orthoptist	049- 2-0411
Parramatta			
Private		Miss J. Clark, Medical Centre, 154 Marsden St., 2150	635-3460
Sponsors	Drs. R. Moxham & C. Cumming, 20 Macquarie St., 2150	Miss K. Rose	635-8509
	Drs. F. Hopkins & Partners 152 Marsden St., 2150	Mrs. J. Dixon Mrs. L. Wong	635-7795 635-7077
	Parramatta District Hospital, Marsden St., 2150		
Penrith			
Sponsor	Dr. R. Chatfield, 314 High St., 2750	Mrs. A. M. Vince	047- 31-1811
Port Macquarie			
	Spiral Towers, 87 William St., 2444		83-2428
Randwick			
Private		Miss A. Dwyer (Mrs. Punch) Medical Centre, 66 High St., 2031	398-4001
Sponsor	Dr. I. Kennedy 66 High St., 2031	Miss S. Moors	398-4001
	Dr. R. McGuiness, 66 High St., 2031	Miss K. Rose	
	Prince of Wales Hospital, High St., 2031	Miss B. Paul Miss C. Langrell Miss G. O'Sullivan	399-2268

Redfern			
Rachel Forster Hospital, Pitt St., 2016		Mrs. P. Howarth	699-3222
Rockdale			
Sponsor	Dr. D. Swan, 27 George St., 2216	Miss K. Shanahan	59-8666
Ryde			
Private	Medical Centre,	Miss D. McCorquodale, Medical Centre, 219 Blaxland Rd., Top Ryde	808-1622
St. Leonards			
Private		Miss M. Hall, North Shore Medical Centre, 66 Pacific Highway, 2065	43-4614
		Locums: Pat Erikson, Mrs. B. McDougall	
The Royal North Shore Hospital, Pacific Highway, 2065		Miss J. Russell	438-0411 Ext.8373 8372
Sydney			
Private		Misses P. Lance, H.R. Hawkeswood & Miss S. Gray 106/67 Castlereagh St., 2000	232-6775
		Mrs. A. Rona, 195 Macquarie St., 2000	211-1714
Sponsor	Dr. P. Beaumont, 187 Macquarie St., 2000	Mrs. V. Messerli	
Medical Eye Service	27 Commonwealth St., 2000	Miss A. Philpotts	61-6534
Sydney Eye Hospital,	Sir John Young Crescent,	M/s S. Brown, F. Merrick	230-0111
		S. Tait, S.J. Pardey, A. Watts, F. Horton	Ext.2758
		Part-Time: A. Hitch, M. Doyle, J. Russell	
Tamworth			
Sponsor	Drs. Baker, Burnett & Wilson, 33 Dowe St., 2340	Miss C. Brennan	66-1622
Wagga Wagga			
Sponsor	Drs. D. Thomson, G. Nixon & M. Halley, 1 Trail St., 2650	Miss A.M. Mahoney	
Warrawong			
Sponsor	Dr. K. MacMillan 242 Cowper St.,	Miss V. Webster	
Westmead			
Marsden Hospital	Mons Rd., 2145	Mrs. J. Erby	635-8866
Wollongong			
Sponsor	Drs. Brown, O'Connor & Higgins 82 West Market St., 2500	Miss S. Barlow	042- 28-6666
Sponsor	Dr. Scott, 68 Market St., 2500	Mrs. J. Martin	042- 29-3655
AUSTRALIAN CAPITAL TERRITORY			
Canberra			
Sponsor	Dr. W. Pluschke, 66 Doyle Terrace, Chapman, 2611	Miss S. Cowle	062- 88-5936
Sponsor	Drs. J. Lodge & D. Coop, Canberra Arcade, West Row, Canberra City, 2601	Mrs. Garfit-Mottram	48-0084
	Drs. L. Shanahan & S. Saunders, P.O. Box 718, Canberra City, 2601	Mrs. P. Frilay	49-6000
VICTORIA			
Albury			
Sponsor	Dr. S. Goddard 573 Stanley St., 2640	Mr. C. Prior	
Sponsor	Dr. G. Howsam, 541 Kiewa St., 2640	Mr. C. Prior	

Ballarat			
Sponsor	Dr. M. H. Brennan & Dr. G. Mangan 16 Errard St., Nth. Ballarat, 3350	Mrs. J. Fyffe Miss E. Boag	321-7033
	Ballarat & District Hospital, Sturt St., 3350	Mrs. J. Green Mrs. J. Fyffe	32-1771
Balwyn			
Sponsor	Dr. W. Firth-Smith 174 Whitehorse Rd., 3103	Miss B. Richardson	80-4250
Bendigo			
Sponsor	Dr. R. Wilson, Shamrock Building, Williams St., 3550	Mrs. B. Devidas	43-1268
Sponsor	Dr. J. O'Sullivan	Mr. C. Prior	
	Bendigo & District Hospital, Lucan St., 3550	Mrs. B. Devidas	43-0411
Bentleigh			
Sponsor	Dr. J. L. Foster, 7 Bent St., 3204	Mrs. B. Williams, Mrs. M. Hicks & Miss L. MacLarn	97-1042
Blackburn			
Sponsor	Dr. R. Stasiuk 156 Canterbury Rd., South Blackburn, 3130	Rosemary Kable	
Boronia			
Sponsor	Dr. J. Sutton, Knox Consultant Centre, 275 Dorset Rd., 3155	Miss K. Hobson Miss R. Ghent	762-2419
Box Hill			
Sponsor	Dr. P. Hardy-Smith 50 Nelson Rd., 3128	Miss L. MacLarn	89-7916
Burwood			
Sponsor	Dr. P. Henderson, 258 Warrigal Rd., 3125	Miss M. Carter	288-3388
Brighton			
Sponsor	Dr. P. Cowen, 14 Male St., 3186	Miss E. Taylor	92-4863
Sponsor	Dr. R. Lindsay Jones 429 Bay St., 3186	Mrs. P. Wister	96-5556
Cheltenham			
Sponsor	Dr. N. Alpins, 7 Chesterville Rd., 3192	Miss J. Shepperd	550-6122
Dandenong			
Sponsor	Drs. J. Stewart & D. O'Connor, 236 Thomas St., 3175	Mrs. M. Hicks Miss E. Boag	792-2155
Department of Defence			
	Department of Defence, 3 Camp Hospital, Puckapunyal	Mr. C. Prior	
Doncaster			
Sponsor	Dr. S. P. Brumley, 619 Doncaster Rd., 3108	Miss M. Carter	848-7680
Fitzroy			
Sponsor	Dr. G. Serpell, 181 Victoria Pde., 3065	Mrs. J. Strentz	419-3393
	St. Vincents Hospital, Victoria Pde., 3065	Mrs. K. Ferraro	40-0221
Footscray			
Sponsor	Drs. E. Finkelstein & B. L. Wilburham, 34 Paisley St., 3011	Mrs. P. Norton Miss R. Ghent	68-1959 68-3501
	Western General Hospital, Eleanor St., 3011	Mrs. L. Mulhall	317-0211
Frankston			
Sponsor	Drs. A. Goldmann & D. J. Shuter, 42 Beach St., 3199	Mrs. B. Williams Mrs. J. Hunter	783-5245
Sponsor	Dr. J. B. Foster, 10 Davey St., 3199	Mrs. J. Strentz	781-1244

Geelong			
Sponsor	Dr. J. Bishop, 10 Wallace Ave., 3220	Mrs. M. Hicks	
Sponsor	Dr. J. Robinson, Ryrie St., 3220	Mrs. M. Hicks	
	Geelong & District Hospital, Ryrie St., 3220	Mrs. M. Hicks	
Glenroy			
Sponsor	Dr. C. A. Koniuszko, 6 Corio St., 3046	Miss M. Carter	306-6234
Glen Waverly			
Sponsor	Dr. P. L. Wong, 272 Springvale Rd., 3150	Mrs. V. Gordon	560-6818
Greensborough			
Sponsor	Dr. J. D. Cairns, 83A Main St., 3088	Mrs. J. Hunter	453-3388
Heidelberg			
	Austin Hospital, Studley Rd., 3084	Miss B. Richardson	45-0411
Kew			
Sponsor	Dr. T. F. Spring, 77 High St., 3101	Miss K. Hobson	862-1111
Korumburra			
	Korumburra District Hospital, 3950	Miss E. Boag	
Malvern			
Sponsor	Drs. J. Heinze & J.E.K. Galbraith, Cabrini Medical Centre, 5 Cooni Cres., 3144	Mrs. G. Heinze	509-7646
Melbourne			
Private	20 Collins St., 3000	Miss D. Mann (Mrs. J. Craig)	63-3115
Private	111 Collins St., 3000	Mrs. V. Gordon	63-2639
Sponsor	Dr. W. Chin, 20 Collins St., 3000	Miss S. Romston	63-1438
Sponsor	Prof. G. W. Crock, 32 Gisbourne St., East Melbourne, 3002	Miss A. M. Mahoney	662-2000
Sponsor	Dr. W. E. Gillies, 110 Collins St., 3000	Mrs. A. McIndoe	63-5869
Sponsor	Dr. G. Harley, 15 Collins St., 3000	Mrs. M. Hicks	63-4829
Sponsor	Dr. E. Ryan, 4 Collins St., 3000	Mrs. P. Wister	65-5393
Sponsor	Dr. H. Ryan, 100 Collins St., 3000	Miss M. Carter	63-5706
Sponsor	Drs. R.H. West & W.D. Counsell, 428 St. Kilda Rd., 3004	Mrs. L. Collins	267-2549
Sponsor	Drs. Borger, Coote, Markwick & Nave, 20 Collins St., 3000	Mrs. S. Bower, Miss D. Mann (Mrs. J. Craig) Mrs. M. Robinson	63-3115
	Prince Henry's Hospital, St. Kilda Rd., 3004	Mrs. P. Wister Miss V. Ball	62-0621
	Queen Victoria Memorial Hospital, 172 Lonsdale St., 3000	Mrs. L. McKenzie Miss K. Hobson	66-6046
	Royal Victorian Eye & Ear Hospital, 126 Victoria Pde., East Melbourne, 3002	Mesdames V. Cameron, L. Collins, D. Craig, J. Fyffe, J. Green, M. Hicks, J. Hunter, A. McIndoe, J. Newton, P. Norton M. Robinson, J. Strentz, A. Williams, P. Wister. Misses V. Ball, M. Carter, A. Harris, B. Haines, A-M Mahoney, L. Martin, B. Richardson, S. Royston, J. Wreidt, Mr. C. Prior	662-2000 Ext. 420
Moonee Ponds			
Sponsor	Dr. J. Long & M. Brumer, 88 Maribyrnong Rd., 3039	Mrs. L. Mulhall	370-7877
Moorabbin			
Sponsor	Dr. C. Gill, 324 South Rd., 3189	Mrs. J. Hunter	95-8888

Morwell			
Sponsor	Dr. A. Steward, 6 Church St., 3840		
Noble Park			
South Eastern District Hospital, Princes Hwy., 3174		Miss J. Sheppard	547-1000
Oakleigh			
Sponsor	Dr. N. Mellor, 12A Atherton Rd., 3166	Mrs. P. Wister	56-3706
Parkville			
Royal Children's Hospital, Flemington Rd., 3052		Mrs. J. Krstic, Mrs. L. Brierly, Mrs. S. Smith	347-5522
Prahran			
Alfred Hospital Commercial Rd., 3181		Mrs. L. Collins	520-2811
Preston			
Sponsor	Dr. B. Nicholls, 268 Murray Rd., 3072	Miss M. Carter	478-5060
Preston and Northcote Community Hospital 265 Bell St., 3072		Mrs. B. Williams	44-0241
Sponsor	Dr. R. Hudson, 39 Erin St., 3121	Mrs. J. Krstic	42-2425
Ringwood			
Sponsor	Dr. J. Mitchell, 21 Ringwood St., 3134	Miss A. Syme	83-3056
Sponsor	Dr. G. H. Vaughan, 43 Ringwood St., 3134	Mrs. B. Williams	870-8022
Swan Hill			
Sponsor	Dr. J. Yeung, Swan Hill Medical Centre, 3585	Miss B. Richardson	
Shepparton			
Sponsor	Dr. M. F. Roche, 65 Nixon St., 3630	Miss A. Harris	21-3455
Warragul			
Sponsor	Dr. B. Nicholls, West Gippsland Base Hospital, 3820	Miss M. Carter	
Warrnambool			
Sponsor	Dr. G. Jones, 200 Liebig St., 3280	Miss B. Richardson	62-5866
Windsor			
Sponsor	Dr. L. Cebon, 33 The Avenue, 3181	Miss V. Ball	529-1949

QUEENSLAND

Brisbane			
Private		Miss J.E. Kirby, 1 Wickham Tce., 4000	31-1597
Sponsor	Dr. H. Fraser, Bancroft Medical Centre, Chermside, 4032	Mrs. K. Edwards,	59-6288
Sponsor	Dr. M. Harrison, 137 Wickham Tce., 4000	Mrs. S. Daly	221-4972
	Dr. E.F. McGuiness, "Inchcolm", Wickham Tce., 4000	Mrs. A. Dickinson,	221-0437
Mater Children's Hospital, Annerley Rd., South Brisbane, 4101		Mrs. A. Dickinson	44-0141
Princess Alexandra Hospital, Ipswich Rd., Woolloongabba, 4102		Mrs. S. Daly	91-0111
Royal Brisbane Children's Hospital, Herston Rd., Herston, 4029		Miss J. Kirby, Mrs. A. Dickinson	52-0111
Nambour			
Sponsor	Dr. H. Fraser 18 Lamington Tce., 4650	Mrs. K. Edwards,	41-3061

SOUTH AUSTRALIA

Adelaide			
Private		Miss J. Taylor, 225 Melbourne St., North Adelaide, 5006	267-3304
Sponsor	Dr. D. Karunaratne, 183 Tynte St., North Adelaide, 5006	Miss D. Sprod	267-4677
	Drs. M. Moore, J. Murchland, D. Bennett & P. Munchenburg 163 Nth Terrace, 5000	Miss J. Stewart	51-9722
	Drs. J. Slade, R. Renton, A. Jordan, D. Tamblyn 79 Pennington Tce., North Adelaide, 5006	Miss C. Knight	267-1967
	Dr. P. Stobie, 182 Ward St., North Adelaide, 5006	Miss D. Sprod	267-3722
Adelaide Children's Hospital,	King William Rd., North Adelaide, 5006	Misses J. Taylor, C. Knight	267-4999
Royal Adelaide Hospital,	North Terrace, 5000	Mrs. J. Smits & Miss D. Sprod	223-0230
Bedford Park			
Flinders Medical Centre,	Flinder Drive, 5042	Mrs. P. Hall	275-9911
Regency Park			
Regency Park Centre for Handicapped Children,	Days Rd., 5010	Mrs. P. Hall	268-4444

TASMANIA

Hobart			
Private		Miss J. Clerk, Miss R. Wilkinson 201 Macquarie St., 7000	23-5306
Royal Hobart Hospital		Miss J. Clerk, Miss R. Wilkinson	38-8493
Sight Saving School		Miss J. Clerk	23-7510
Sponsor	Dr. G. Wise, 151 Macquarie St.,	Mrs. J. Adams	23-5118
Launceston			
Sponsor	Dr. Westmore, 66 York St., 7250	Mrs. T. Barbour	31-7951
	Dr. Quartermass, 5 Elphin Rd., 7250	Mrs. T. Barbour	31-5413
General Hospital,	Charles St., 7250	Mrs. T. Barbour	

WESTERN AUSTRALIA

Albany		
Regional Hospital	Miss B. Balfour	
Bunbury		
Private	Miss B. Balfour, Q.B.E. Building, 20A Stirling St., Bunbury, 6230	
Esperance		
District Hospital	Miss S. O'Donnell	
Fremantle		
Private	Miss B. Balfour, 26 Queen St., 6160	335-6048
Fremantle Hospital, Alma St., 6160	Mrs. W. Hogan	335-0111
Manjimup		
District Hospital	Miss B. Balfour	
Northam		
Regional Hospital	Miss B. Balfour	

Perth			
Private		Miss B. Balfour, 48 Hilda St., Shenton Park, 6008	381-2980
Private		Misses M. Lewis & C. Childs (part-time), 254 St. Georges Tce., 6000	322-3454
Private		Miss M. Lewis, 24 Washington St., Victoria Park, 6100	
Private		Miss A. Terrell, 35 Richardson St., West Perth, 6005	321-4650
Princess Margaret Hospital, Thomas St., Subiaco, 6008		Misses M. Lewis, B. Balfour, C. Childs (locum) and Mrs. S. Wilders	381-0222
Royal Perth Hospital		Misses M. Lewis, A. Terrell	
University Dept. of Ophthalmology Royal Perth Hospital		Miss A. Terrell	
St. John of God Clinic, Subiaco		Miss P. Warham 4 Eltham St., Wembly Downs, 6019	
Sponsor	Dr. D. McAuliffe, 169 Cambridge St., Wembley, 6014	Miss S. O'Donnell Mr. G. Johnson	
	Dr. D. C. Candy, Dr. T.McL. Wilson, Dr. C. Townsend, 169 Cambridge St., Wembly, 6014		
	Pilbara Region		
	Fremantle		
	Rockingham-Kwinana District Hospital		