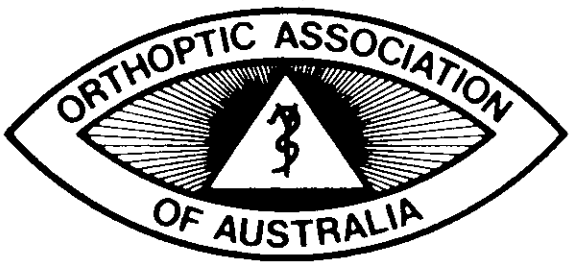
AUSTRALIAN ORTHOPTIC JOURNAL



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ORTHOPTIC ASSOCIATION OF AUSTRALIA

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PRESIDENT'S ADDRESS 1985

This year, as you are well aware, the theme for the 1985 Conference is "Communication". It was chosen predominantly because it is an area of involvement for each of us in whatever aspect of practise we find ourselves. On consideration of the topic, it seemed appropriate to look at how orthoptists have developed their lines of communication over the past 42 years, both within the profession, from the aspects of communication between orthoptists, and outside the profession in terms of communication by orthoptists with people for whom our profession has developed their skills. I would therefore, like to speak on communication with regard to the association, the training of orthoptists, patients seen by the orthoptists and professionals with whom the orthoptist is in contact.

Firstly, looking at the Orthoptic Association of Australia:

It is interesting to see that in 1942, when there were only 10 practising orthoptists, it was considered necessary to discuss the formation of an Association of Orthoptists. Its purpose being to establish links (or communicate) with practitioners throughout Australia, who shared similar expertise and had mutual interest in elevating standards to the benefit of the patients' care. In 1942, the Orthoptic Association of Australia was inaugurated and the first formal steps in communication were made.

Since 1943, the membership of 10 has steadily increased and currently over 300 orthoptists take the advantage of the benefits of communicating with their colleagues, to share in the mutual areas of scientific development and administrative gain. With the passing years, the Association, whilst being mindful of its goals and through communication with its members has seen the need to modify its structure and develop a range of membership categories which acknowledges the various needs of its members. These include:

HONORARY MEMBERS

(Established in 1942) For orthoptists and medical practitioners who the Association wishes to

acknowledge for their activities which have enhanced the profession.

Fellows (Established in 1973) For members whom the Association wishes to honour for their contribution to the profession.

Associate Members (Established in 1950) For orthoptists not working, but wishing to remain in communication with their colleagues.

Student Members (Established in 1980) A category which is intended to encourage orthoptists in training to join their future colleagues and so become involved in the communication process aimed at improving expertise and maintaining optimal standards for the benefit of the patients and the community as a whole.

In 1967 the Orthoptic Association of Australia represented by Pat Lance, joined in with several other orthoptic associations from various countries to form the International Orthoptic Association. Thus, communication of orthoptist to orthoptist became available throughout the world.

In Australia where vast distances separate practitioners, an essential method of communication for members of the Orthoptic Association is the written word which, on a regular basis is through the Australian Orthoptic Journal and the Association's News Letter. Of these, the Journal has developed in a similar manner to that of the Association. Pat Lance, in her President's address to the Association in Canberra in 1973, referred to the proceedings of early meetings as being "duplicated and sent out as typed notes to members and interested ophthalmologists". 1966 saw the first printed journal being published and today the Association supports and enjoys a journal of high calibre which continues to be circulated to all members of the OAA, all members of RACO and, as well, to subscribers from within Australia and overseas. As the journal represents the profession in Australia, the maintenance of a high standard is essential and requires each member's support both financially and in scientific endeavours.

On a less regular, though it would seem increasing basis, a more subtle form of written communication is the surveys which are circulated to members. These time consuming, often seemingly illogical pieces of paper, which require skills not taught in student days, provide valuable information to administrators and educators. Such surveys are a tool of the future as they yield relatively objective information regarding the development of new skills and the redundancy of other skills. Surveys additionally provide information to Government bodies which is useful in the acceptance of education programs proposed by, and on behalf of, the profession. They have also provided positive evidence in the current discussions for the possible inclusion of orthoptic services in medicare.

The Association plays a major role in another important aspect of communication: the education and training of orthoptists, so that the standards for patient care are maintained.

The education process is complex, it is continuous and fluctuates in intensity throughout our professional life. At all stages it involves communication, be it in written form through journals or verbal form through continuing education programs and general discussion with colleagues. Education can be direct, as on a program organised to deliver specific information, or indirect through the previously mentioned surveys, where, in order to answer questions in the survey, a sorting and sifting process can be established which helps to reshape our ideas.

In Australia, the basic training of orthoptists has developed through various stages. Initially, training was through "on the job experience", then a 12 months training program was adopted, which developed into a 2 year program supported by the Orthoptic Board of Australia. With the advent of Government support for the training schools, our profession was awarded an Associate Diploma, which has since further developed to a comparatively sophisticated 3 year Diploma program. The future holds promise of a degree program in the area of basic training and adventurous post graduate programs such as satellite linked continuing education courses,

both of which will enable the profession to continue its goal of elevating standards on an international level.

The final area of communication which I would like to discuss relates to the most important issue, that of the purpose of our existence in the health care services, which is our role with people outside our profession, namely patients and other professionals.

The communication patterns with patients have shown considerable change over the years. For instance, initial clinics in Victoria and RAHC in NSW were predominantly for children.

In the 1970s, orthoptists commenced work with both mentally and physically handicapped patients, offering skills in the assessment of their visual function and thus enabling improved management of other defects that the patients may experience. This has been an area of practise that has proved to be of definite value to the patient's well being and to other professionals in a multi-disciplinary team involved in the patients' overall care.

This ability to communicate with patients who find such skills difficult has also been of assistance to members of the non-English speaking community and in providing accurate assessment of eye conditions.

With regard to communication with other professionals, we are most fortunate to have strong links with ophthalmologists from whose guidance we have gained so much. In recent years orthoptists have modified their skills and provided a wider range of services to assist with the eye care of the patients and, consequently, have become more involved in work with ophthalmologists in areas other than in ocular motility.

Additionally, orthoptists are becoming more actively involved in their self regulation and direction, as is evident by representation to Government Bodies such as 'Better Health Commission' and 'Medicare Review Committee' for possible support of the profession.

Recent years have seen the development of communication links with many other professions such as Community Nurses, where assistance is given in teaching visual screening techniques. Additionally, with other therapists, communication on a two way basis has resulted in improved understanding of patients capabilities, ultimately leading to a total care program which has been shown to result in increased independence for the patient.

Having thus refreshed our memory on the development of communication skills within the various facets of the profession, it seems fitting for each of us in the Orthoptic Association to turn our thoughts to the future and to continue expanding our channels of communication so that the community patients, fellow professionals and colleagues may gain the full advantage of our skills.

Neryla Jolly

Australian Orthoptic Journal 1986; 23: 1-5

THE NEWBORN FOLLOW-UP CLINIC: A preliminary report of ocular abnormalities*

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Abstract

The Newborn Follow-up Clinic (NFC) at The Royal Alexandra Hospital for Children follows the development of "high risk" infants. 132 infants were enrolled in the Clinic in July 1985.

Ninety five of the NFC infants have been assessed in the Orthoptic Clinic. Their gestation ages ranged from 23.5 weeks to 42 weeks and their birth weights from 570 grams to 4440 grams.

Visual impairment was present in four infants (4.21%). Fifteen infants were found to have strabismus (15.7%).

The NFC Clinic plans to continue with review assessments until the children reach the early school years.

Key words: *Multidisciplinary team, retinopathy of prematurity, visual function, strabismus.*

The radical changes in methods of management that have been introduced into the intensive care nursery in recent years, have resulted in a significant improvement in the survival rate of premature and sick infants.[1,2] Technology has meant that the infant's progress can be monitored and problems detected and treated with far greater skill than was previously possible. In addition, the new attitude of the Neonatal Intensive Care Unit (NICU) recognizes the importance of close parental involvement with the sick neonate.

High levels of handicaps have been reported amongst the survivors in "high risk" groups in earlier studies.[1] However, more recent reviews suggest a more optimistic outlook for survival of these infants without handicap.[3,4,5]

At the Royal Alexandra Hospital for Children (RAHC), as in many of the other major world units responsible for the care of high risk infants[6,7,8,9,10] the need for a long-term follow-up of NICU "graduates" has been recognized. The task of the Newborn Follow-up Clinic (NFC) is to monitor the developmental outcome of the most vulnerable graduates in order to identify specific medical and psychosocial needs of these children and their families, and to act as a medical audit for neonatal intensive care services. The current programme has been running since early 1982.

The criteria for selection of infants for the NFC includes those who display one or more of the following characteristics:—

— Less than 1500 g birthweight.
— Less than 34 weeks gestation.
— Significant neurological problems (seizures, meningitis, grade III & IV intracranial haemorrhage, perinatal asphyxia).
— Chronic oxygen dependancy.
— Prolonged hospitalization.

Excluded are:—
— Major congenital malformations.
— Babies enrolled in other specialist clinics.
— Babies being followed developmentally at other hospitals.

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*This paper was awarded the Emmie Russell Prize for 1985.

TABLE 1
Newborn follow-up clinic, July 1985

Follow-up programme:		Assessments								
		Medical	Physiotherapy	O.T.	Audiology	Orthoptics	Speech	Psychometric	Educational	Social work
Corrected ages	4 months	X	X	X						X
	10 months				X	X				
	12 months	X	X	X						X
	12 months	X	X	X						X
	2½ years	X	X	X		X	X			
Chronological ages	3½ years	X	X	X	X	X				X
	4½ years	X	X	X				X??		X
	7½ years	X	?	X	?	X	?	X??	X	X

As the NICU graduate is known to be at a high risk for suffering an extensive range of disorders, a multidisciplinary team approach to follow-up is seen as most valuable.[12,13] At present, assessment for the NFC is performed by Neonatologists, Paediatricians, Occupational Therapists, Physiotherapists, Audiologists, Orthoptists, Speech Pathologists and Social Workers. Each therapist involved performs an evaluation of the infant at the appropriate age, as indicated in Table 1. The information is correlated by the physician, following the medical examination, and the parents are advised of the infant's progress.

The involvement of the Ophthalmic Unit in the high risk infant's follow-up is essential. In particular, there is an early need for ocular examination of the premature baby, to determine the presence of Retinopathy of Prematurity (ROP). Despite the careful monitoring of arterial oxygen levels, ROP continues to be a major cause of blindness in the very low birth weight infant.[12,13] Ophthalmic examination is performed on all infants who were considered to be at risk of developing ROP. This examination was usually conducted prior to the infant's discharge. There is also a higher incidence of strabismus, cataract and myopia in low birthweight infants.[7]

The Orthoptist's role in the NFC assists in assessment of the level of visual function and the status of binocularity.

Orthoptic Assessment:

All NFC infants are routinely screened at the corrected ages of ten months, 2½ or 3½ years, and 7½ years.

Determining visual function is regarded as a priority in the Orthoptic assessment. The Catford Drum, Stycar Balls and other clinical observations are the usual methods employed for testing vision in the ten month group. Although these methods do not give an accurate standard of acuity, they provide an evaluation of visual responses from each eye. The majority of 2½ and 3½ year olds have been able to perform vision tests using Sheridan Gardiner letters or Single Pictures. Confrontation visual fields are performed, where necessary.

Cover testing was used to diagnose strabismus at distances of ⅓ m and 3 m, where possible 6 m and where appropriate, far distance. Binocular co-ordination was assessed with a 15 Δ BO Prism, or preferably, the Lang's Stereo Test. Several of the older children co-operated to perform the TNO Stereo Test. Extraocular muscle movements were also examined.

Where defects were identified, the physician in charge of co-ordinating the child's care was informed and ophthalmic referral arranged.

Results and Discussion:

In July 1985, there were 132 infants and children enrolled in the NFC. Ninety five of these had been seen in the Orthoptic Clinic at a corrected age of ten months, 19 had been rechecked at 2½ or 3½ years. The following results relate only to those infants who have been orthoptically assessed.

The infant birth weights ranged from 570 g to 4440 g and their gestation ages from 23.5 weeks to 42 weeks. The distribution of birth weight compared to gestation is shown in Figure 1.

TABLE 2
Neonatal Conditions (N = 95)

Condition	No. Affected	Condition	No. Affected
Respiratory Distress Syndrome	56	Patent Ductus Arteriosus	17
Apnoea	24	Persistent Transitiona Circulation	5
Bradycardia	11	Anaemia	12
Chronic Oxygen Dependancy	1	Hyperglycaemia	4
Chronic Lung Disease	8	Hypocalcaemia	3
Perinatal Asphyxia	23		
Pneumothoraces	9	Seizures/Convulsions	12
Jaundice	31	Sepsis	23
		Meningitis	2
Intracranial Haemorrhage		Pneumonia	2
— non specific	13		
— intraventricular	13	Necrotising Entercolitis	12
— subarachnoid	21		
— subependymal	11		
— subaponeurotic	1		

Table 2 lists some of the problems encountered by the infants in the neonatal period, which placed them "at risk". This list is by no means complete.

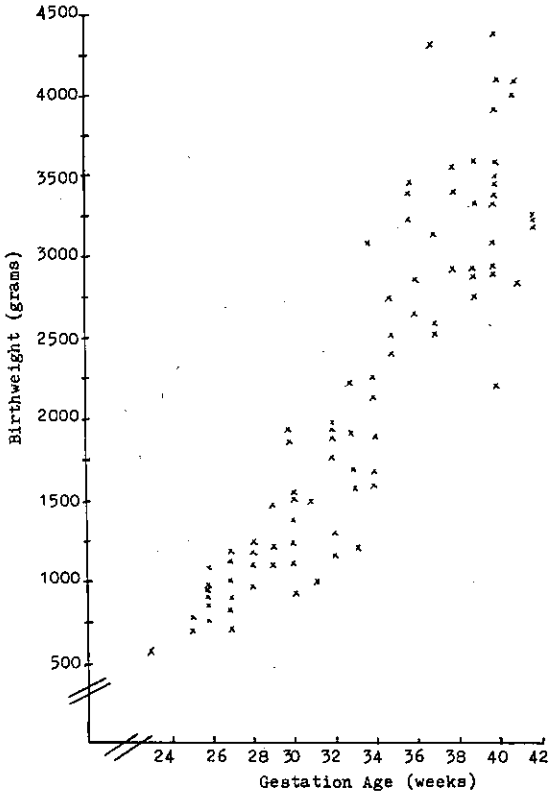

Figure 1: Birthweight vs Gestation Age of NFC Infant.

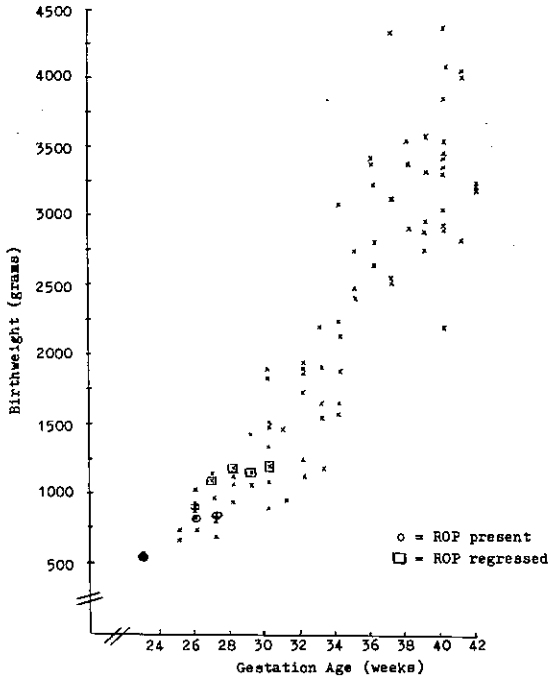

Figure 2: Incidence of Retinopathy of Prematurity.

Ophthalmic Examination:

Eighty one of the infants were checked for the presence of ROP. Of these, only three have been found to have severe visual impairment due to ROP. Five other infants on initial examination, showed some signs of retinal changes, consistent with early ROP, but this regressed at subsequent follow-up. In all three cases, ROP was present

TABLE 3
Orthoptic Abnormalities (N = 95)

Gestation	Visual Impairment	Esotropia	Exotropia	Amblyopia
25 weeks	1		1	
25-30 weeks	1	4	3*	4
31-35 weeks		3		3
36-40 weeks	1	3		1
40 weeks		1		1

in both eyes. The infants born at 23.5 and 26 weeks gestation have Grade V ROP, while the 27 week gestation baby shows Grade II ROP.

Figure 2 demonstrates very clearly that the incidence of ROP is concentrated at the lower end of the gestation and birth weight scales — a finding that is common to the many other reports of this syndrome.[7,12,13]

In the group of infants ophthalmically examined, acute ROP occurred at a rate of 3.7%. When the minimal ROP changes are included, this rate increased to 9.87% of the group.

This study agrees with previous authors[12,13] who have suggested that ROP occurs predominantly in those infants whose birth weight is less than 1500 grams. Fifteen per cent of infants weighing less than 1500 grams showed signs of ROP. Acute ROP occurred in 9.38%. Stark[14] found the incidence of ROP to be 19% in infants whose birth weight is less than 2000 g, 22% when birth weight was between 1000 g and 1500 g, and 47% when the infants weighed less than 1000 g at birth. In Kitchen's study,[7] ROP caused visual abnormalities of 6.3% of the very low birth weight babies. Gole[12] states that in infants of birth weight less than 1500 g, 3% are blinded by the disease.

The arterial level of oxygen is also considered to be a significant influence in producing ROP.[14] Two of these infants were ventilated to the level of 100% oxygen, in order to overcome respiratory distress. The third infant, with Grade V, had no documentation of prolonged hyperoxaemia, but there were large fluctuations in the oxygen levels during respiratory distress.

Development assessments have shown marked motor and global delay in all three of these infants at the corrected age of twelve months.

One infant, born at 38 weeks gestation, weighing 2830 grams was found to have a microphthalmic right eye, with colobomata of the iris, choroid and retina. This child has several other dysmorphic features, and at twelve months was developmentally delayed.

Infants are being reviewed where necessary, to correct refractive errors.

Orthoptic Examination:

Table 3 shows the abnormalities that have been encountered on Orthoptic assessment of all the NFC infants.

Visual impairment was evident only in the four babies mentioned to have abnormal eyes — three with ROP and one with microphthalmos. The infant with Grade II ROP was showing visual interest in the environment. He has an esotropia with horizontal and rotary nystagmus, but uses "crossfixation" to reduce the nystagmus. One of the Grade V ROP infants was showing some visual interest, but this was very limited. He was exotropic. The second infant with Grade V ROP demonstrated even less ability to use vision, the clinical report commented "reacts to very bright light in a darkened room, but has no idea of fixing or locating the light". He was also exotropic. In the case of microphthalmic eye roving nystagmus was present, but the infant did make an attempt to fix and follow targets. She was esotropic.

The infants who had shown early ROP changes, were demonstrating a good level of visual function at ten months. All of these infants had straight eyes and binocular co-ordination was evident.

A total of 15 infants, or 15.7% of this group were found to have strabismus at the corrected age of ten months. This is a somewhat higher

incidence than is accepted for the general population.[15,16] The higher neurological risk of these infants may be responsible for this. Kitchen[7] found an incidence of strabismus to be 20-25% in a group of very low birth weight babies that have been followed to eight years, when intermittent and accommodative type deviations would have become evident.

In seven of the cases where amblyopia has been detected, treatment with occlusion has been undertaken and four of the infants have progressed to surgery.

Developmental assessment has shown that delay is a common feature amongst the strabismic cases. All four of the visually impaired infants were showing significant delay. Seven others in the strabismic group were also showing some developmental abnormalities, which ranged from minor delays, to more marked problems.

The nineteen cases where a review of ocular posture and visual acuity has been performed at 2½ or 3½ has revealed no new abnormalities. More accurate assessment has confirmed equal vision, of a good standard for age and straight, binocularly co-ordinated eyes.

ACKNOWLEDGEMENTS:
The assistance and advice given by Mr B. Cook, Miss H. Hardy and Mrs S. Horne has been greatly appreciated. The R.A.H.C. Departments of Neonatology and Ophthalmology have generously given permission for their patients to be used in this paper.

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VISUAL PERFORMANCE IN THE LOW VISION CHILD

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Abstract

The visual performance of pre-school children who have been assessed in the Child and Adolescent Services Department of the Royal Blind Society of N.S.W. is discussed.

The developing low vision child often establishes strategies around his visual impairment to compensate for his visual loss. The importance of clinical observation, and of spending time with these children, indoors and outdoors, is discussed. In addition to this, emphasis is placed on obtaining information from the parents, who can provide details concerning the child's visual performance in the home and a history of any deterioration or improvement in his vision.

Key words: *Visual impairment, strategies, clinical observation, visual functioning, residual vision, parent observation.*

INTRODUCTION

While much emphasis is placed on obtaining a visual acuity recording of the pre-school child in an orthoptic assessment, straightforward clinical observations and a discussion with the parents of the child can be easily forgotten. This, of course, involves a great deal of time which is not usually available in the busy ophthalmological practice or hospital eye clinic.

At the Child and Adolescent Services Department (C.A.S.D.) of the Royal Blind Society, this time is available which proves to be invaluable when attempting to fully understand a child's visual function. Experience in knowing what to look for and a keen eye for a child's every move is essential.

The aim of this paper is to briefly outline the various methods and situations used in the C.A.S.D. when gathering information regarding a child's visual performance in general, with special attention to some of the many strategies employed by the children to compensate for their visual loss.

POPULATION

A vast range of children with varying degrees of visual impairment and delayed development are seen in the C.A.S.D. For the purpose of this study, children whose cognitive functioning was in the range normal to mild intellectual ability were considered. The various eye conditions represented can be seen in Table 1.

UNDERSTANDING THE CHILD'S DEVELOPMENT

Before observing the visual performance of children with low vision, it is important to fully understand the nature of their eye condition and any other disabilities they may have. As 45% of children seen at the C.A.S.D. do have other disorders that may affect their development, it is important to have a comprehensive medical history of the child and the family situation to help determine possible reasons for any developmental delay that may be present.

In the C.A.S.D. this information is obtained by case discussions with professionals working

Reprint request: Child and Adolescent Services Department, Royal Blind Society of New South Wales, 4 Mitchell Street, Enfield, New South Wales 2136.

TABLE 1
Patterns of Referral to the C.A.S.D.
April 1st 1979 — March 31st 1985

Total No. 595	Incidence	%
Cortical Blindness	91	15.3
Optic Nerve Atrophy	76	12.8
Congenital Cataract (except Rubella)	55	9.2
Retrolental Fibroplasia	48	8.1
Albinism	45	7.6
Congenital Nystagmus	44	7.4
Genetical Retinal Dystrophy	34	5.7
Optic Nerve Hypoplasia	27	4.5
Leber's Tapeto Retinal Degeneration	16	2.7
Coloboma of Retina/Choroid	16	2.7
Congenital High Myopia	14	2.4
Congenital Glaucoma	14	2.4
Aniridia	11	1.8
Bilateral Retinoblastoma	8	1.3
Rod Monochromatism	8	1.3
Microphthalmus	8	1.3
Retinitis Pigmentosa	7	1.2
Optic Nerve Dysplasia	7	1.2
Optic Nerve Glioma	6	1.0
Septo-Optic Dysplasia	6	1.0
Miscellaneous	54	9.1

with the children (including occupational therapists and pre-school teachers) and other members of the assessment team (which includes an ophthalmologist, neuro-pyschologist and a physiotherapist).

Those working with the children, to stimulate vision and development, can provide the orthoptist with valuable information regarding how the child utilizes his residual vision in the home and at pre-school.

Up to date ophthalmological reports concerning the ocular diagnosis are also essential to have.

TESTING ENVIRONMENT

It is extremely important to allow children to be fully relaxed when assessing their visual functioning.

The layout of the indoor assessment area of the C.A.S.D. is similar to a lounge/rumpus room in the average Australian home. It consists of a beige coloured carpeted floor, to provide a good contrasting background, and many toys, including a cubby house and rocking horse, which help to stimulate and maintain the child's interest. Similarly, in the outdoor assessment area, there is much play equipment, including tricycles, a small trampoline and a sandpit complete with buckets and spades.

An outdoor assessment area is vital when assessing low vision children as there can be a marked difference in their visual behaviour when the boundaries of the environment are harder to realize. Children affected by glare, such as those with albinism, rod monochromatism and cataracts, can be "blinded" outdoors and yet function visually once they are inside. It must be pointed out that glare is often worse for the low vision child on a light overcast day than on a fine sunny day.

Indoor lighting should also be considered in all vision testing. Dimmer switches for internally illuminated charts and general ceiling lights enable vision to be tested under many different lighting conditions.

To achieve optimum results children also need to feel at ease with the examiner. At least 10 minutes are allowed for the child to develop confidence in the orthoptist.

As many children may have experienced many visits to a hospital the orthoptist is careful about using a clinical torch in this period.

ASSESSMENT OF VISUAL PERFORMANCE

(1) Standardised Tests

When assessing pre-verbal children, the familiar Stycar series of toys and balls and 100's and 1000's are used. These, however, are often modified according to the degree of visual impairment present and/or the child's level of interest. For example, some Stycar balls have been painted various colours to vary the contrast against the assessment area's beige carpeted floor or conversely, the white balls are rolled across the floor without the black felt to assess the child's visual discrimination of low contrast objects within a room. Care is taken when setting the balls in motion by "flicking" them rather than moving a hand or arm which gives the child clues as to which direction the ball is travelling. The standard visual acuity notations are not used as it is the opinion of the author that they are inaccurate. For example, when the test was administered on a six year old normally developing co-operative child who had Leber's

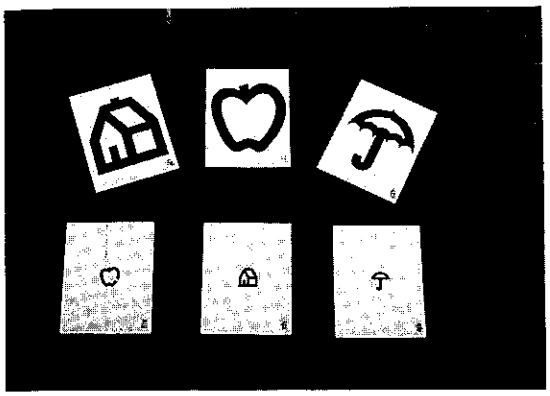

Figure 1: Examples of Lighthouse Symbols.

Figure 3: Logmar Linear Chart.

optic atrophy and a binocular visual acuity of 3/60 (as measured on a Logmar Linear Chart), it was found that she could accurately locate a 6/5 (9 mm) ball from a distance of three metres.

Once a child begins to develop some speech or can match symbols and letters it becomes possible to employ more of a range of tests and, thereby, begin to obtain the much sought after visual acuity recording.

Sheridan Gardiner Single Letters are used extensively and modified at times by alternating the testing distance and the number of letters on the child's card. Sometimes the letters are referred to as various objects depending on the child's language ability. For example, the 6/60 letter "O" is often referred to as a "ball".

Other tests used that are not often seen in the regular eye clinic include Lighthouse symbols (Figure 1) for distance acuity testing and Bust symbols for near acuity testing.

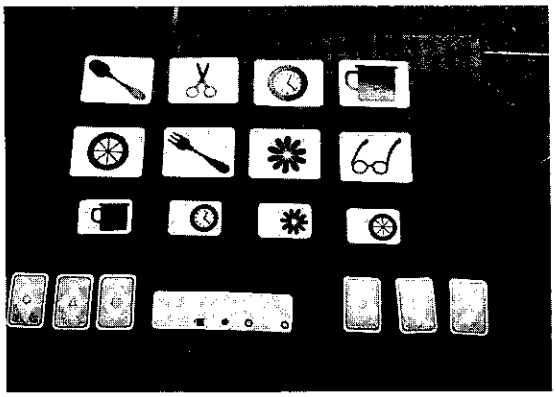

Figure 2: Examples of Bust Symbols.

Bust (see Figure 2) is the name of a new test for children at early mental ages and is a combined test of form perception and visual acuity. It was devised by Eva Lindstedt, Ophthalmologist, Sweden, and is designed in such a way that the demands on the child's understanding of the image and also the demands made on his visual acuity can be varied. The test can be used as a card game, played with in various ways and finally used directly for testing visual acuity.[1] Each card is designed in such a way that the person leading the game can see from the back of them which series each card belongs to and the size of its symbol. The pictures come in nine sizes with the largest of them equivalent to N144 and the smallest to N8-10.[2]

When a child has mastered the art of matching, or knows the letters of the alphabet, a Logmar Linear Chart is used. (Figure 3)

This chart is much more comprehensive than the Snellens Chart in that it gradually reduces the letters by the addition of a 6/48, 6/38, 6/30, 6/19 and 6/15 line, with each line consisting of five letters.

(2) Clinical Observations

Simple observations of a child's spontaneous behaviour can provide an examiner with a wealth of information about a child's level of vision. When we look at the practical purpose of vision

testing we find that perhaps it is not always so very important to quantify the child's visual acuity. Explanation of a child's visual impairment, in the context of daily activities, is often more meaningful to a parent or teacher than an interpretation of visual acuity notations.

(a) Near Tasks

Low vision children often hold objects very close to their eyes and adopt an abnormal head posture to reduce their nystagmus. The latter can sometimes be misleading when determining which is the better seeing eye. For example, a child who is adopting a face turn to the left may appear to be favouring his right eye when in actual fact he is viewing with his left eye which is in the adducted null point position for his nystagmus.

Stycar balls and toys or anything held in front of a child for him to reach for are placed on a thin black rod as well as held in the orthoptist's hand to compare each response. When the child reaches for the object, the way in which he directs his hand towards it is noted. Such questions as the following are considered.

Does he under or overshoot? Does he grasp the orthoptist's hand first, then feel his way up to the object? or is his reaching out poorly directed? Once having grasped the object it is also interesting to note how the child handles it. Is he inspecting the object visually, with his tactile sense, or with both?

There are some children, particularly in their first two years of life, whose vision improves from light perception to that of reaching out on sight and spotting objects within a 2-3 metre range. This has been observed in conditions such as cortical blindness and optic nerve hypoplasia. The improvement in the visual system can occur within a matter of weeks but the child's ability to exercise his newfound vision effectively can take longer. Blind mannerisms such as utilization of tactile and auditory senses, or the child simply not looking at the task at hand, are often retained for some time.

(b) Distance Spotting

When encouraging a child to use his vision to find a particular object care is taken to avoid any auditory cues or familiarity as to where it is or should be. Any object can be used when assessing distance vision as long as it's size, description and distance it was spotted at, are accurately recorded. Although some children may only respond to well known objects, e.g. a dummy or a bottle, this can be an advantage as it becomes more meaningful to the parent when explained in terms familiar to them.

When a child is encouraged to retrieve an object, the way he approaches the task is closely observed. Such questions as the following are considered:

Is he effectively scanning his environment to find the object?

Has he spotted it before venturing out to retrieve it? or does he move forward in the hope of seeing the object when it comes into his range of vision?

The child is encouraged to point to the object before retrieving it, which gives a truer indication as to whether or not he has seen it. If he will not point, he is closely watched as he moves forward to see if he "alters his course" as he approaches the object. Often he will break into a run or a quick trot and get excited when he has spotted it.

(c) Recognising People's Faces

A simple test has been devised in the C.A.S.D. to help determine the exact distance a child needs to be from people before being able to recognise their faces. Three participants, including one parent, are used and seated within a chair space of each other in the corner of the assessment room while the child is in another room. The participants are carefully selected, so that hair colouring and general outline are similar, and are instructed to wear thin lemon over-coats to obscure any item of clothing that may be a clue for the child. Each participant remains silent and does not make any hand gestures or facial expressions.

The child is encouraged to "find daddy or mummy" by having him give something to his parent or get something from them. After he has become familiar with where the parent is seated the test is repeated with the participants exchanging places.

It is often quite striking to the parents how close their child needs to be before being able to recognise their face. The test highlights his reliance on other clues such as colour, contrast and memory.

This test has been titled the "Kimino" test.

(d) Hand Gesture and Facial Expressions

Communication between one another does not only occur through speech but can be just as effective through body language. As Scott et al. point out, in any family, there is a tremendous amount of nonverbal communication going on all the time.[3] By a smile, a frown, or a tender look a clear message can be sent across the room. A wave of the hand, a pointing of the finger and shaking or nodding a head all convey messages. As a visually impaired child can often miss or misunderstand these gestures, the distance at which they can see these non-verbal clues needs to be ascertained.

In view of this, gross and fine hand gestures, e.g. hands on head and pointing to facial features respectively, and various facial expressions are performed by the orthoptist at various distances. Eye contact is also noted during this test.

Gross hand gestures can usually be seen by children with 3/60 vision or better at six metres but fine hand gestures with children whose visual acuity is between 3/60 and 6/24, are often confused at this distance.

This information is not only valuable to the parent, but also the teacher, as it helps to correctly position the child in the classroom.

(e) Orientation and Mobility

Observation of the low vision child moving around his environment, indoors and outdoors, can not only provide the orthoptist with information about the degree of visual impairment present and whether any residual vision is used in an effective manner, but can also give a general idea of any gross field defects present.

The orthoptist pays particular attention to how the child approaches unfamiliar territory and how he scans his environment. The latter can be assessed by encouraging him to walk or run through an obstacle course.

A common feature seen during assessments in the C.A.S.D. is the child hesitating or putting his foot forward to feel any changes in contrast of the terrain, before negotiating it. Some children move across the change with a high stepping action or bend down to crawl over it, thinking the new surface is at a different level.

A similar action can be seen when the child passes over shadows or cracks in the pavement.

The orthoptist also notes if the child walks with slightly outstretched hands or with a tentative gait. Some children consistently lead with one foot, walking slightly sideways and adopting an abnormal head posture to centralise their field of vision.

As previously mentioned, glare can have an adverse affect on visual functioning. Mobility can be grossly affected, particularly outdoors, in that the child becomes cautious in his surroundings and relies on his other senses more.

(c) Parents' Observations

The parents are often the best assessors of their child's visual functioning as they are with him in many different situations and know his every mood. More often than not an orthoptist or ophthalmologist is only confirming what the parents already know, as far as the child's visual capabilities are concerned.

During an assessment at the C.A.S.D. a good half an hour or more is spent with the parents to gather information about the child's visual functioning in the home or places he is likely to visit. The following are a few examples of questions that are asked:—

1. How close to the television does he sit?
2. At what distance does he hold objects to inspect them?
3. What does he indicate he can see while travelling in a car, e.g. can he spot animals in nearby paddocks?
4. Can he see the moon or stars at night?
5. Does he see better during the day, at dusk or at night?
6. Does he become "clingy" in unfamiliar environments?

Answers to these questions help to form a profile of the child's visual capabilities for

practical purposes. When documented, this helps the lay person to fully understand the needs of the visually impaired child.

The parent can also provide a detailed history of any changes in the vision. The child may become more adept at utilizing his residual vision or may actually become more visual due to such things as the maturation of his visual system, cataracts being removed or appropriate spectacles being prescribed. Conversely he may begin to stumble over things, hold objects close to his eyes or become more irritable or frustrated as his vision deteriorates. Usually the parents can give the most accurate information concerning these observations.

CONCLUSION

Through experience in clinical observation the orthoptist can become more adept at placing a child's visual functioning in the following categories of visual acuity.

Light perception	→	1/60
1/60	→	3/60
3/60	→	6/60
6/60	→	6/18
6/18		or better

One must always be on the lookout for any signs of visual impairment as many children may present extremely visually alert and perform very much like a normally sighted child. Pre-school children seen in the C.A.S.D. who have as little vision as 3/60 often have adequate vision for their present needs. The visual impairment of these children is more obvious when greater demands are placed on their vision at school, e.g. when they commence reading or blackboard work.

In summary, clinical and parental observations of the low vision child's visual functioning, a good understanding of his physical and psychological development, and time with the child are all important factors when making recommendations to parents. The orthoptist can play a key role in the assessment of the low vision child by bringing the parents, and other professionals involved, to a fuller understanding of the child's present condition and its implications in the present environment and the future.

ACKNOWLEDGEMENTS

I would like to thank Dr J. E. Rowlands who compiled the statistics in Table 1 and was instrumental in developing the Kimino Test. I would also like to thank the staff at the C.A.S.D. of the Royal Blind Society for their invaluable assistance when working with the families.

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THE SPECTRUM OF CONGENITAL RUBELLA

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Abstract

Twenty four children, all students at the deaf and blind children's centre, North Rocks, are reviewed. All have multiple defects as a result of congenital rubella. The types of defects are discussed with the prevalence of bilateral cataracts, nystagmus, psychomotor retardation and hearing loss being found to be significant. Emphasis is placed on the importance of immunisation against maternal rubella.

Key words: *Cataracts, nystagmus, retardation, hearing loss, immunisation.*

INTRODUCTION

The Congenital rubella syndrome in its most severe and complete form is characterised by signs of disseminated virus infection. Consequences of infection are determined primarily by the timing of the viral insult. Infection at any time during the first four or five months of gestation may result in widespread or multifocal inflammatory disease of varying severity. Infection in the first six to eight months presents the greatest hazard to organogenesis and life as a result of cell damage and necrosis.

The effects are widespread and severe including microcephaly, psychomotor retardation of varying severity, spastic quadriparesis and sensorineural hearing loss. Ocular abnormalities are cardinal manifestations of congenital rubella. They include unilateral or bilateral pearly nuclear cataract, frequently associated with microphthalmia, iris hypoplasia, atrophy synechiae and vascularization are common. Congenital glaucoma due to incomplete differentiation of the chamber also occurs and pigmentary retinopathy caused by a disturbance of pigmentation in the retinal epithelium. Optic nerve fibre damage and high refractive error are also common and these cause vision deficits, pendular ocular nystagmus and strabismus.

Sir Norman Gregg[1] first drew a correlation between maternal rubella and congenital cataracts in 1941 following an epidemic of german measles. In 1944 he reported further[2] on other defects which were also associated with maternal rubella including cardiac defects, deafness and mental deficiency.

The purpose of this review, of subjects attending the Deaf and Blind Children's Centre, North Rocks, was to assess the incidence of these associated defects in those diagnosed as having congenital rubella.

Information was gathered from the medical files of 24 subjects and by observation, since all are residents of the Centre. Age was considered, as well as the following eye signs — the incidence of cataracts, bilateral and unilateral, microphthalmia, vascularization of the cornea, congenital glaucoma, pigmentary retinopathy, optic atrophy, nystagmus, high refractive error. Other effects considered were microcephaly, psychomotor retardation, cerebral palsy, hearing

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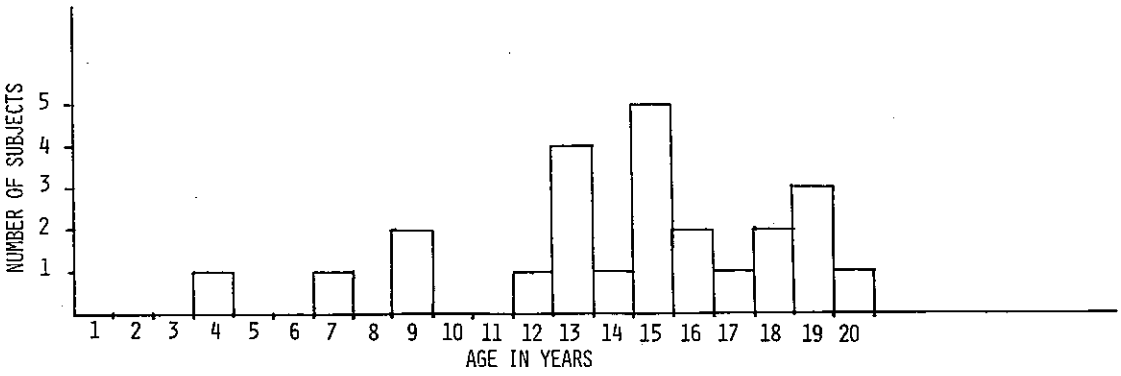

Figure 1: Age of subjects.

loss and congenital heart defects. Note was also taken of how many of the subjects were totally blind and if they had had one or both eyes enucleated.

RESULTS

The subjects' ages ranged between four and twenty years, with the mean age being 14.3. (Fig. 1).

Nystagmus, bilateral cataracts, psychomotor retardation and hearing loss were all found to be most frequently associated with congenital rubella.

One hundred per cent of the subjects in the study had psychomotor retardation ranging from moderate to severe. Eighty seven per cent had a hearing loss with some achieving good hearing with the help of hearing aids and others being classed as profoundly deaf. Seventy one per cent had bilateral congenital cataracts and 8% had unilateral cataracts. The cataracts associated with the congenital rubella syndrome are pearly white, involving all but the outermost layers of the lens, thus indicating that they had begun to develop early in the life of the embryo. Visual acuity was low in all subjects who had cataracts removed with the best assessable vision being 6/24. Twenty nine per cent of subjects were totally blind and nystagmus was present in the remaining 71%, the nystagmus varied between fine horizontal to the fairly gross wandering nystagmus associated with low vision. Thirty seven per cent had microphthalmia in either one or both eyes and glaucoma was present in 21%. Glaucoma is usually difficult to control in these children, two subjects had buphthalmos in one eye with one suffering from a chronic infection in that eye. One subject had a total anterior chamber hyphaema as a result of the glaucoma and another has had both eyes enucleated.

Pigmentary retinopathy was present in 25% of subjects. This pigmentary retinopathy is caused by a disturbance of pigmentation in the retinal epithelium with discrete areas of pigment clumping and focal areas of atrophy and depigmentation of varying size and distribution. The pigment mottling and coarse "salt and pepper" changes are often most pronounced in the macular area and just posterior to the equator and the foveal reflex may be distorted. The retinopathy has minimal, if any, effect on visual functions; it is non-progressive and the ERG responses are generally normal. Vision deficits, pendular ocular nystagmus and strabismus are more commonly due to the cataracts, glaucoma, optic nerve fibre damage, or high refractive error.[3]

Optic atrophy had only been noted in 8%, and 4% of subjects had a high refractive error, e.g., — 10.00 DS. Congenital heart defects were present in 29% with the majority of these being cardiac murmurs. Twenty five per cent had cerebral palsy with 66% of those having spastic quadriplegia and the remaining 33% having hemiplegia. Twenty one per cent were diagnosed as having microcephaly and 17% had vasculari-

sation of the cornea with 75% of these being bilateral and associated with total blindness. Twenty five per cent were affected in one eye only with this eye being buphthalmic.

Other defects noted were hypertension, syndactyly of hands and feet, hare lip and cleft palate. Growth failure was also evident and most subjects had behaviour problems including hyperactivity and difficulty establishing normal sleep patterns.

DISCUSSION

The congenital rubella syndrome has been shown to cause many and varied developmental defects. Generally those affected tend to have several of the associated defects. The group used for this study would be expected to be more severely affected because they are residents in a school and activity therapy centre for multi-handicapped blind students. Therefore, it would be expected that the congenital defects would be found in higher percentages than groups reported by Sir Norman Gregg.[1,2] These two groups are used for a comparison, one with 99 cases and the other with 7. Deafness was present in 55% of the first group and 3% of the second, whereas the North Rocks group had 87%. Cataracts were not included in the first study; in the second they were present in 86% of the group with 57% being bilateral and 29% being unilateral. The North Rocks group had 79% with 71% being bilateral and 8% being unilateral.

Through this comparison it was found that the North Rocks group had a much higher percentage of deafness than Sir Norman Gregg's Group. This may be due to the fact that North Rocks caters for multi-handicapped blind children, so would therefore have a higher percentage of subjects with deafness or any other handicap. The prevalence of cataracts in both groups was in similar percentages indicating that it would be found to be significant in other studies. It is interesting to note that the North Rocks group had a much lower percentage of unilateral cataracts than Sir Norman Gregg's group. The majority of the North Rocks group, i.e., 90% having bilateral cataracts, whereas they were present in 66% of the group used for comparison.

The sample used for this study is small and limited in that all subjects were students in a special school for multihandicapped blind children, or an activity therapy centre which is a progression from the school. It must be assumed that not all babies born with the congenital rubella syndrome will be as severely affected as this sample. However, the risk involved is extremely high. Swan, Tostevin, Mayo and Black[4,5] found that in every instance in which the mother contracted rubella in the first two months of pregnancy she later gave birth to an infant with congenital abnormalities.

Of the subjects included in this study, several are state wards and others attend the centre on a full-time basis with occasional outings or weekends with their families. This is indicative of the difficulties families face in trying to care for these children. Behaviour problems include tantrums, scratching and biting and difficulty establishing normal sleep patterns. Many require full-time care with some being almost fully dependant until they are at least ten years of age. Most will always require some form of institutionalised care and if possible a sheltered workshop situation.

At this point, the advisability of vaccination against rubella cannot be overemphasised. The N.S.W. Department of Health visits all primary and secondary schools once a year and vaccinates all girls who have not yet been vaccinated, provided they have not reached the age of 15 years. Vaccination is still recommended if the child has previously had an attack of rubella. The Department of Health also recommends that all women, before starting a pregnancy, should check whether they are immune to rubella by having an H.A.I. blood test. This is advisable even if they were vaccinated at school.

CONCLUSION

This study has documented the vast range of disabilities that can result from the congenital rubella syndrome. It has shown that children are still being born with this syndrome despite the publicity and Department of Health warnings.

Therefore, a responsibility lies with all health professionals to educate the public as to the necessity for vaccination against rubella, even if it is only by having pamphlets available in their waiting rooms.

ACKNOWLEDGEMENTS

Thanks must be extended firstly to Dr. John Gregory-Roberts for suggesting the topic and title of this paper, and also to Mr Reg. Mitchell.

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VISUAL RESPONSES OF PATIENTS WITH ECCENTRIC VIEWING

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Abstract

Three case studies are presented as examples of unusual visual response during eccentric viewing training. Two cases present problems with localisation, the third with ocular dominance. None of these problems proved to be a barrier to eccentric viewing.

Key words: *Eccentric viewing, retinal projection, visual localisation, ocular dominance.*

The relative localisation of the observer in relation to the environment is a function of normal binocular single vision. The fovea has straight ahead visual direction and is the primary point of reference. The remaining retina consists of a series of points which have functional correspondence with points of the contralateral retina. When pathology prevents the use of the fovea for fixation forcing the use of a peripheral retinal point, changes in retinal projection and or localisation may reasonably be expected.[1]

Ocular dominance is a visual phenomena which has been studied in relation to dyslexia,[2] occlusion therapy[3] and as an aetiological factor in some orthoptic problems. Fowler and Stein[4] discuss the barrier created by ocular dominance when a child is forced to use the non-dominant eye or where ocular dominance is not established. Amphlett's research indicates occlusion of the dominant eye does not prevent improvement in the visual acuity of the non-dominant eye when treating amblyopia. Does ocular dominance effect eccentric viewing when visual acuity is not equal?

The effect of eccentric viewing training on retinal projection or visual localisation has not been widely studied. The effect of ocular dominance on eccentric viewing is equally sparse in report.

Two cases of localisation in relation to eccentric viewing and one case of a problem of ocular dominance and eccentric viewing will be discussed.

CASES

Case I

35 year old male

Diagnosis: probably a form of Bull's eye dystrophy.

V.A R 1/12 L 2/24

BEO N32

FIELD: Bilateral central scotoma

Bilateral viewing position, dextro depression.

To gain maximum benefit from his residual vision Case 1 needs to establish an eccentric viewing point on his lower temporal retina of the right eye and a lower nasal point with the left eye. This is achieved by placing the eyes in a position of dextrodepression. If normal projection is present, the stimulation of these two

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retinal points will result in a localisation of the object in the upper field. The print used as a stimulus in the eccentric viewing training will therefore appear above and to the left of their actual position in space. This sensation of indirect localisation was observed by the patient and found to be most distracting. The problem was not overcome but once the patient understood the reason for his confusion in localisation, he was able to concentrate on eccentric viewing and ignore the indirect localisation.

This case illustrates the importance of understanding the underlying condition and developing communication in the successful management of these patients.

Case II
21 year old female
V.A R 3/18 L 3/18
BEO N36
FIELD: Bilateral central scotoma
SYNOPT.: Obj. FR & L — 10° (Obj. & Subj.)
After image: 
Eccentric viewing position dextroversion.

This young patient had already established an eccentric viewing point prior to training. However, she was unable to determine when her eyes were in the primary position. The aim of training was to modify the currently used eccentric viewing point to improve visual acuity and to assist the patient to reach primary position at will. Whilst viewing eccentrically in dextroversion, the patient thought her eyes to be in primary position. The eccentric viewing training required her to place her eyes in primary position then look into the selected position of gaze. However, this patient was unable to place her eyes into primary position without specific directional instructions. The question arose, was this due to a change in retinal projection?

To determine the state of retinal correspondence, the patient underwent a Bagolini lens test. There was no response as the light did not provide sufficient stimulus. The synoptophore was tried, the results indicated normal retinal correspondence. The after image test was performed as the cross provided a stimulus which could be localised despite the central field loss. This test also indicated normal retinal correspondence. (However, the unreliability of this test is noted.) The patient was able to correctly localise objects in the visual field. It would appear that retinal correspondence and localisation had not been altered. The patient was therefore instructed to move her eyes into laevoversion and hence attain primary position.

Case III
66 year old male
Diagnosis: Angioid streaks with associated macular and peripheral scarring. Glaucoma, which is controlled.
No evidence of glaucomatous field defect.
V.A R 1/6 L 1/6
BEO 6/36 pt.
BEO N 80.
FIELD: Bilateral central scotoma
Eccentric viewing dextroversion RE.
Laevoversion LE
Laevoversion provided the better prognosis for V.A
Ocular dominance of RE

This patient faced a dilemma of eccentric viewing. The patient demonstrated right dominance in right handedness and preferred the right eye for uniocular viewing. Macular lesions were more extensive in the right eye leaving the left eye with the better visual prognosis.

The most viable area of retina for eccentric viewing in the right eye is dextroversion, whereas laevoversion is preferable for the left eye. Therefore, to obtain maximum vision, the patient should use the left eye in laevoversion. Initially the patient was unable to do this and persisted in using the right eye in dextroversion. Occlusion of the right eye was instituted during training. Over a period of four to five weeks this technique proved to be successful and the patient was able to use his left eye in laevoversion. Reading accuracy improved with the change in eccentric viewing position.

CONCLUSION

Three cases are presented.

Case I, where eccentric viewing with the retention of normal retinal projection resulted in the sensation of indirect localisation when viewing a printed stimulus. This was a distraction to eccentric viewing but was not an insurmountable problem.

Case II, where an eccentric viewing point appeared to have taken on the straight ahead visual projection. Further testing indicated normal retinal correspondence and the patient was able to accurately localise objects in space. This appears to be an acceptance of ocular position without change to retinal function.

Case III. Ocular dominance initially prevented the use of the better eye for eccentric viewing. Total occlusion of the dominant eye in the early stages of training allowed the use of the better eye. Once eccentric viewing was established with this eye there was no further problem.

The most effective use of residual vision can be influenced by many factors. The above examples indicate the need for further study in the area of retinal physiology to gain more complete understanding of visual retraining.

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TREATMENT OF CONVERGENCE INSUFFICIENCY IN AUSTRALIA

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Abstract

Data on 190 cases of convergence insufficiency who received treatment from a variety of orthoptists during 1984 and 1985 is analysed. Results show that patients are typically young adults, have no refractive error and complete treatment in approximately eight weeks, requiring about three office visits. Significant improvements are found in measures of convergence, accommodation, near deviation, and fusional convergence. No significant change is found in the distance deviation or fusional divergence.

Key words: *Convergence insufficiency, orthoptic treatment.*

Convergence insufficiency is probably one of the major conditions for which orthoptic treatment is given in Australia. It is considered[1-3] as a relatively simple and efficient method of overcoming asthenopic symptoms for near, yet little data is available on it's effectivity, or on other factors such as the types of patients treated, the most common forms of treatment given, or the usual length of a course of treatment, in Australia. Without such information, justification of public or private spending for orthoptic services cannot be made and standards are not available against which one can measure individual patient characteristics.

In an attempt to overcome these perceived shortcomings, the Scientific Committee of the Orthoptic Association of Australia initiated an Australia wide survey to establish data on some of these issues. Members were requested to record information pre and post treatment on patients referred for convergence training. Patients were excluded from the study if they were on medication, had an history of head trauma or systemic disease, had a manifest ocular deviation or a distance heterophoria of greater than -10Δ, $+6\Delta$ or 2Δ of hyperphoria. Visual acuity had to be at least 6/9 in each eye. Information was recorded on the following issues:

1. *Patient Characteristics:*
   Age, refractive error, symptoms.

2. *Treatment:*
   What was the main reason for treatment, how long did it last, how many office visits were required, what forms of treatment were given?

3. *Effect of Treatment:*
   Change in symptoms, near and distance heterophoria, convergence near point, accommodation, fusion reserves, stereoacuity.

To gain the maximum amount of information, respondents were encouraged to return information whether or not all the variables were recorded.

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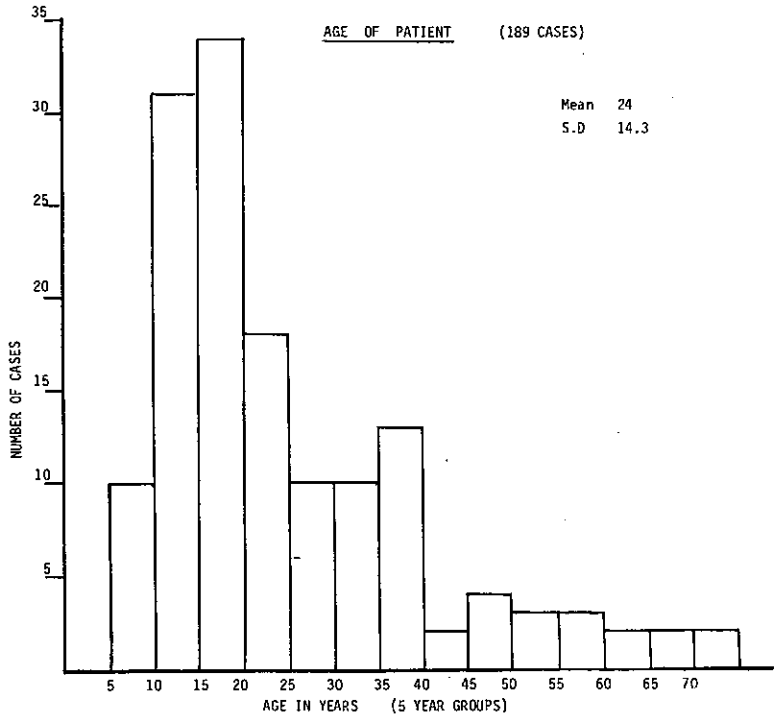

Figure 1

RESULTS

Data on 203 patients were returned, of which 190 fulfilled the required criteria. However, information from all 190 cases was not necessarily available on each specific issue studied.

PATIENT CHARACTERISTICS

1. *Age* (See Figure 1)

   It can be seen that the patients are predominantly young adults, most commonly between the ages of 15-25 years. Nevertheless, older patients are still treated. It is interesting to note that the least common age group is between 40-45 years, when near symptoms would most likely be treated by reading glasses.

2. *Refractive Error*

   Of 151 cases, most (93 or 61.5%) were emmetropic; 19 (12.5%) were hypermetropic, 16 (11%) were myopic and 12 (8%) were presbyopic. The remaining 7% had varying types of astigmatism.

3. *Symptoms*

   The most common recorded symptom (in 114 cases) was asthenopia — that convenient name for a mixture of symptoms related to general ocular discomfort. 94 complained specifically of blurred vision, 89 of headaches, 28 of diplopia and 19 of other symptoms (many patients had more than one symptom). Only six were said to be asymptomatic, of these five were aged 14 or younger, probably reflecting the perception that it is worthwhile to treat convergence defects in children to prevent future symptoms.

4. *Reasons for Treatment*

   188 responses were available for this question. Treatment was given based on symptoms alone in 46% (87), on clinical signs alone in 6% (11 cases) and for both reasons in 48% (90 cases). Therefore, in 83.5% (157) the patient's symptoms were significant in the decision to carry out treatment.

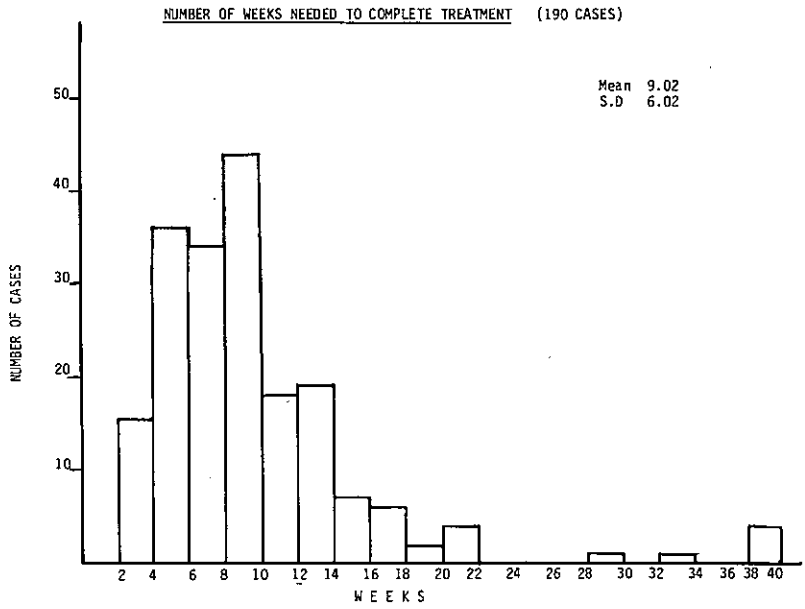

Figure 2

TREATMENT

1. *Length of Treatment and Visits Required* (See Figures 2 and 3)

   The treatment period itself was relatively short, most commonly 8-10 weeks, with usually only 2-3 visits required. At the most (two cases only), eight visits were required.

2. *Specific Exercises*

   Of course, for each patient, several forms of treatment may have been given. Information was available on all 190 cases. The most common exercise given was simple convergence (140 cases) followed by training on stereogram cards (125 cases). Training in voluntary convergence, development of fusional reserves (using prisms or the synoptophore), physiological diplopia and relaxation exercises were given to between 75 and 85 cases. Anti-suppression treatment was given to only 23 cases. "Other" treatment (in some cases named as "Union Jack" and "red work"), was given to 40 cases.

EFFECTS OF TREATMENT

1. *Symptoms*

   Information was available on 65 cases, of whom 97 (63%) were recorded as being asymptomatic after treatment. Of the remaining two, one still had headaches, the other ocular discomfort. It was disappointing that more respondents did not answer this question, due probably to the design of the form which may have appeared to relate symptoms only to the main reason for treatment. (A lesson for next time!). However, of the responses available, the results are certainly good.

2. *Deviation Size* (See Table 1)

   The distance deviation did not change significantly with treatment, however, in 92 cases there was a significant change in the size of the near deviation of the whole group with 41 (38%) showing a reduction in exophoria, at times much as 12Δ (see Figure 4). It is worth considering the reasons for this change. Tradi-

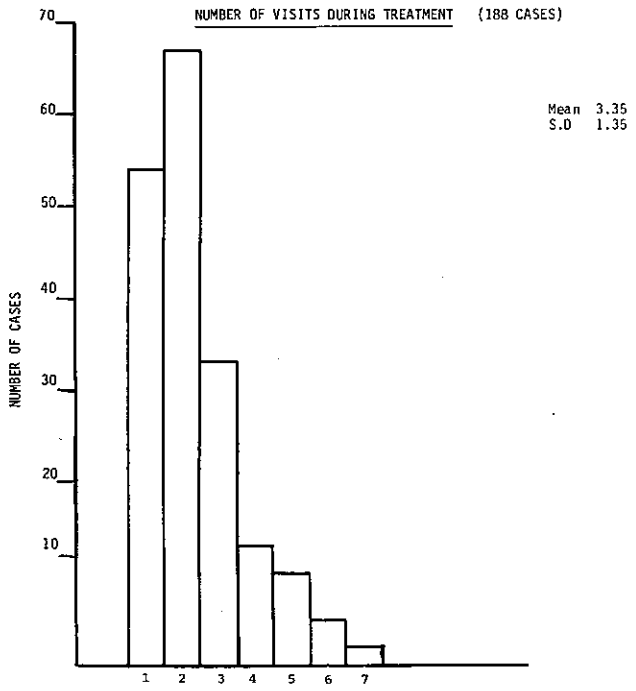

Figure 3

tionally, convergence treatment is said to develop fusional control over a near exophoria.[2,4] However, this should not, of course, affect the size of this exophoria. Therefore, it is likely that other factors such as proximal convergence[5] or accommodation (leading to improved accommodative convergence) have also been affected by this treatment. Some criticisms[6] have been made of the logic in developing the near point of convergence in the treatment of convergence insufficiency. However, it is quite likely that this exercise would stimulate both accommodation and proximal convergence and could thus explain the above reduction in the near deviation.

3. *Convergence Near Point*
(CNP, as measured on the RAF rule).
The improvement in the CNP in virtually all cases can be clearly seen in Figure 5. Before treatment, the mean value was 15.5 cm (S.D. 17.4 cm) and after treatment 6 cm (S.D. 7.6 cm). This was found on the t test to be significant at the .001 level. The possible reasons for the effectivity of this form of treatment have already been discussed.

4. *Accommodation*
Because of the normal variations in the range of accommodation which occur with age, respondents were asked to record whether the near point of accommodation was normal or

TABLE 1
Change in Deviation Size with Treatment

Distance	No. of Cases	Deviation (Δ) Before Treatment	Deviation (Δ) After Treatment	Significance ('t' test)
33 cms	92	−4.8 (S.D. 4.9)	−3 (S.D. 3.4)	P<.001 (Highly Significant)
6 m	87	−0.8 (S.D. 2.5)	−0.7 (S.D. 3)	(Not Significant)

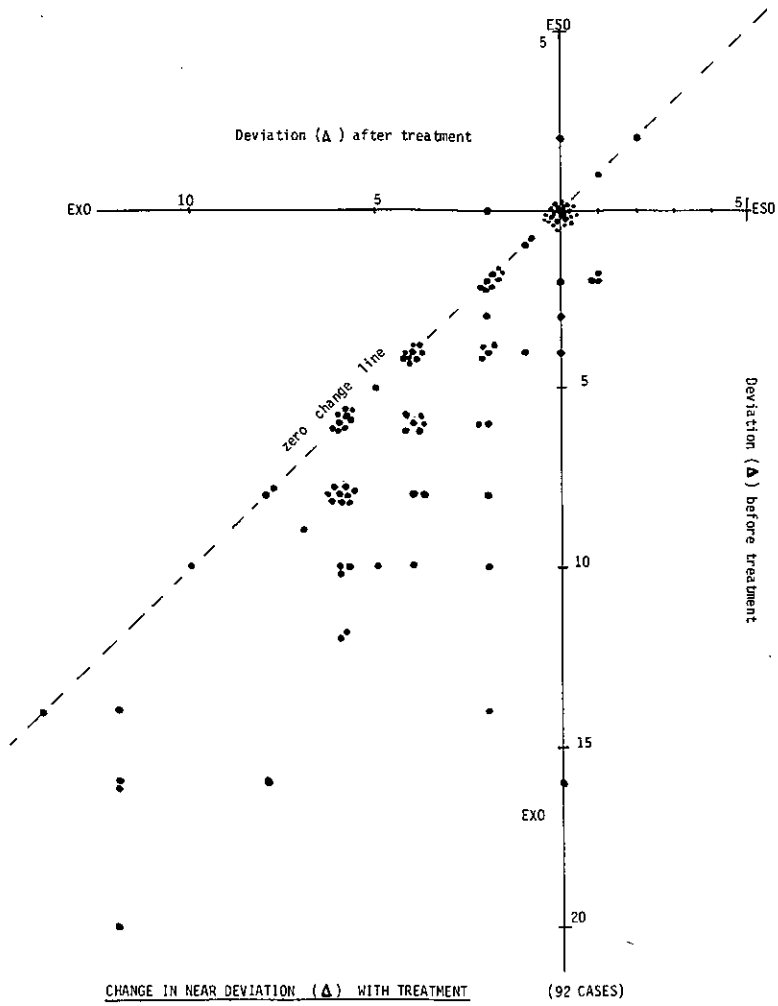

Figure 4

abnormal for the patient's age, allowing for any uncorrected refractive error. Of 109 cases, accommodation was recorded as normal before and after treatment in 45% (49 cases) and abnormal, remaining so, in 2.7% (3 cases). However, for the remaining 52.3% (57 cases) accommodation was reduced before treatment and improved to normal with treatment. It is unlikely that these were cases of true accommodation insufficiency as described by Hitch,[7] but were probably secondary to reduced convergence.

Nevertheless, improvement in accommodation doubtless leads to improved accommodative convergence and, as discussed, may well have contributed to the reduced exophoria for near shown in Figure 3.

5. *Fusion Amplitudes* (See Table 2)
Whether fusional amplitudes were evaluated using prisms or the synoptophore, it can be seen that, whereas there was no significant change to divergence, there was a highly significant improvement in convergence amplitudes. The apparently better ranges achieved at the synoptophore were no doubt due to the fact that the prism bar "runs out" at 40△, and most orthoptists accepted this as

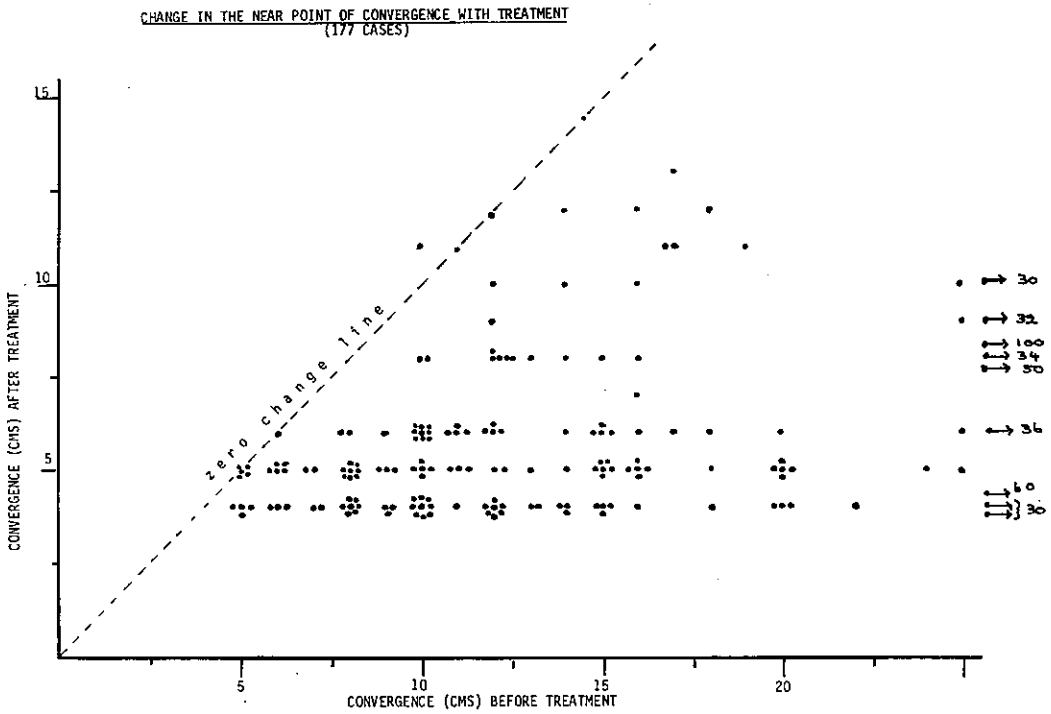

Figure 5

satisfactory. The synoptophore, of course, does not have this limitation.

6. *Stereoacuity*

Stereoacuity, was measured before and after treatment in 82 cases. Of these, 65% (53) showed no change in acuity which was usually high (40'') before and after treatment. However, 35% (29) did show improvement, 9 by 10'', 10 by 30''-60'' and the remaining 10 by 100'' or better. The mean value before treatment was 83.5'' (SD 109.5'') and after treatment 53.6'' (SD 46.6''), an improvement significant on the t test at the 0.01 level.

As all of these cases had good distance visual acuity before and after treatment this improvement could be attributable to either

TABLE 2
Change in Fusional Amplitudes with Treatment

	Range Before Treatment (Δ)	Range After Treatment (Δ)	Significance ('t' test)
Fusional Convergence			
1. Measured with prisms at 6 m (59 cases)	12.8 (S.D. 8.3)	27.3 (S.D. 11)	P<.001 (Highly Significant)
2. Measured at the synoptophore (92 cases)	15.3 (S.D. 10.4)	36.9 (S.D. 14.4)	P<.001 (Highly Significant)
Fusional Divergence			
1. Measured with prisms at 6 m (59 cases)	3.9 (S.D. 1.3)	4.1 (S.D. 1.2)	Not Significant
2. Measured at the synoptophore (92 cases)	4.5 (S.D. 1.9)	4.4 (S.D. 1.7)	Not Significant

improved accommodation, or more precise alignment of the visual axes. This is an area which warrants further research.

SUMMARY

It is readily acknowledged that this study is by no means a comprehensive assessment of all variables influencing convergence insufficiency treatment or its results. It is also possible that some examiner bias may unintentionally have influenced some of the data. Certainly, the ability to converge to within a few centimetres from the eyes is not a function that is required in normal situations, yet it has been shown that improving this function is accompanied, in many cases, by a reduction in the near deviation, improved accommodation and improved fusional reserves, all of which are required for comfortable reading. Most importantly, such treatment can be carried out efficiently, usually only requiring a few visits spaced 2-3 weeks apart. Since most patients are young adults, either students or in their early working life, ocular comfort for near is of obvious importance.

This study can be considered as a preliminary one only, as it has raised several areas where further evaluation is warranted. These include as a more detailed study of the effect of convergence treatment in accommodation, the relationship between measures of improvement and issues such as age, refractive error and amount of near exophoria. However, this type of evaluation of orthoptic treatment is overdue, and hopefully has provided incentive for similar future studies.

ACKNOWLEDGEMENTS

Most of the credit for this research should be given to the orthoptists who participated and returned completed forms. In addition, the other members of the Scientific Committee, Keren Corby, Julie Greene, Alison Terrell, Cindy Dykes, Barbara Walch and Robyn Hill contributed significantly to this study.

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ANALYSIS OF THE AC/A RATIO IN A SAMPLE OF INTERMITTENT EXOTROPES OF DIVERGENCE EXCESS TYPE

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Abstract

Previous authors have suggested that many of the patients diagnosed as intermittent exotropes of divergence excess are, in fact, simulated forms of this condition, ie., that some of these patients control an equally large near deviation by excessive accommodative convergence and possess a high AC/A ratio. In order to investigate the type of AC/A ratio present amongst a sample of seventeen intermittent exotropes of divergence excess type the AC/A was calculated by the gradient method at ⅓m using +3.00 DS lenses. An equivalent normal sample was also investigated. When comparing the two groups the intermittent exotropic sample had a significantly greater proportion of high AC/A subjects. Disclosure of a high AC/A ratio is valuable when considering orthoptic and surgical management of the intermittent exotrope of divergence excess.

Key words: *AC/A ratio, intermittent exotropia of divergence excess type.*

Intermittent squints generally are an example of a dichotomy in the laws of strabismus, by presenting as a controlled heterophoria and a manifest deviation under different circumstances. The nature of an 'intermittent' deviation provides excellent potential to achieve good control of the manifest component and a high standard of binocular vision.

Windsor[1] suggests that intermittent exotropes of divergence excess are rare. These patients are believed to have a basic mechanical or anatomical abnormality which creates a significantly greater divergent deviation in the distance. Recent research implicates the existence of an active divergence centre, Tamler and Jampolsky[2] and Seaber,[3] proposed an innervational aetiology for divergence excess.

Many of the patients who present clinically with heterophoria at near fixation and a manifest squint in the distance are said to be a simulated form of divergence excess. Duke-Elder[4] states simulated or pseudo-divergence excess intermittent exotropes control an equally large near deviation by means of excessive fusional or accommodative convergence.

Burian[5] has outlined an investigative procedure which reveals this larger near angle when the influences of accommodation and fusion are suspended. The method involves preventing fusion by a period of diagnostic occlusion. The influence of accommodation may be eliminated by the use of +3.00 DS lenses at ⅓m.

Burian classified those patients whose near deviation increased to equal, or exceed the distance angle as simulated forms of divergence excess. Those patients whose near deviation could not be significantly altered by either procedure were considered to be examples of true divergence excess.

Various clinical studies adopting Burian's criteria,[6,7,8] support the theory that the majority

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TABLE 1
AC/A Scores in each Group

	AC/A Ratio (to nearest whole number)								
	9	8	7	6	5	4	3	2	1
Divergence Excess group	1	4	1	2	1	—	4	4	—
Normal group	—	—	—	—	2	9	1	5	—

of intermittent exotropes are simulated forms of divergence excess. Windsor[1], indicates that failure to expose simulated forms of divergence excess will result in inappropriate surgical correction, reduced potential to achieve and maintain an adequate standard of binocular vision, and increase the necessity for additional surgery.

Duke-Elder[4] and Brown[9] propose that patients whose near deviation increases significantly under the influence of +3.00 DS lenses possess a high AC/A ratio. This study was designed to investigate the type of AC/A ratio amongst a sample of intermittent exotropes of divergence excess type.

METHOD

A sample of seventeen intermittent exotropes of divergence excess type were selected from a variety of clinical encounters. The subjects were required to have a distance deviation measured by prism bar cover test 15 diopters greater than the angle at near fixation and have no significant refractive error. Windsor[1] stipulates that the accommodative convergence induced by uncorrected hypermetropia will reduce the maximal divergent deviation measurable in exotropes. In order to maintain accommodative consistency the same detailed target was used when the near deviation was calculated, and then recalculated with +3.00 DS lenses in place. Calculation of the AC/A ratio at a fixation distance of 33 cm was chosen principally because the subjects selected for this study displayed greater abilities to relax accommodation at near fixation. Difficulties were encountered when these patients tried to clear an accommodative target at 6 m through −3.00 DS lenses, possibly due to cognitive ability, unfamiliarity with accommodating in the distance and the sensory adaptations present. Since the near deviation was measured with and without +3.00 DS lenses by the same examiner, using the same accommodative target, the influence of proximal and accommodative convergence were considered standardized. The AC/A was calculated by the gradient method. In order to validate this method an orthoptically screened sample of seventeen normal subjects were also investigated.

RESULTS

The AC/A ratio is considered normal by Lyle and Wybar[10] when its value falls between 3 and 5. The AC/A ratio mean of the normal sample was 3.72 with a standard deviation 0.96. The individual scores for the normal sample are presented in Table 1, where it can be seen that 58.8% of the sample were found to possess an AC/A ratio within normal limits, 29.4% had a low AC/A, while 11.8% had a high AC/A.

The divergence excess group had a higher average AC/A ratio of 5.21 with a standard deviation of 2.6. It is evident that a greater proportion of intermittent exotropes possess a high AC/A ratio with 47.1% of the sample displaying an AC/A ratio greater than 5. Only 29.4% of this group have an AC/A ratio within Lyle and Wybar's normal range, considerably less than the control group. Subjects with an AC/A ratio less than 3 account for 23.5% of the divergence excess sample, a figure quite comparable with the control group.

Using the "t" test to compare the two groups, a significant "t", ($+ = 3.26$ $P < 0.05$) was obtained. It may be concluded that a significant difference exists between the two groups.

The two groups are represented graphically in Fig. 1, where the divergence excess sample is seen to deviate markedly from the normal curve shown by the control group.

DISCUSSION

It would appear from the results of the study that a high AC/A ratio occurs more frequently amongst intermittent exotropes of divergence excess than amongst normal subjects. These results would support those who propose that

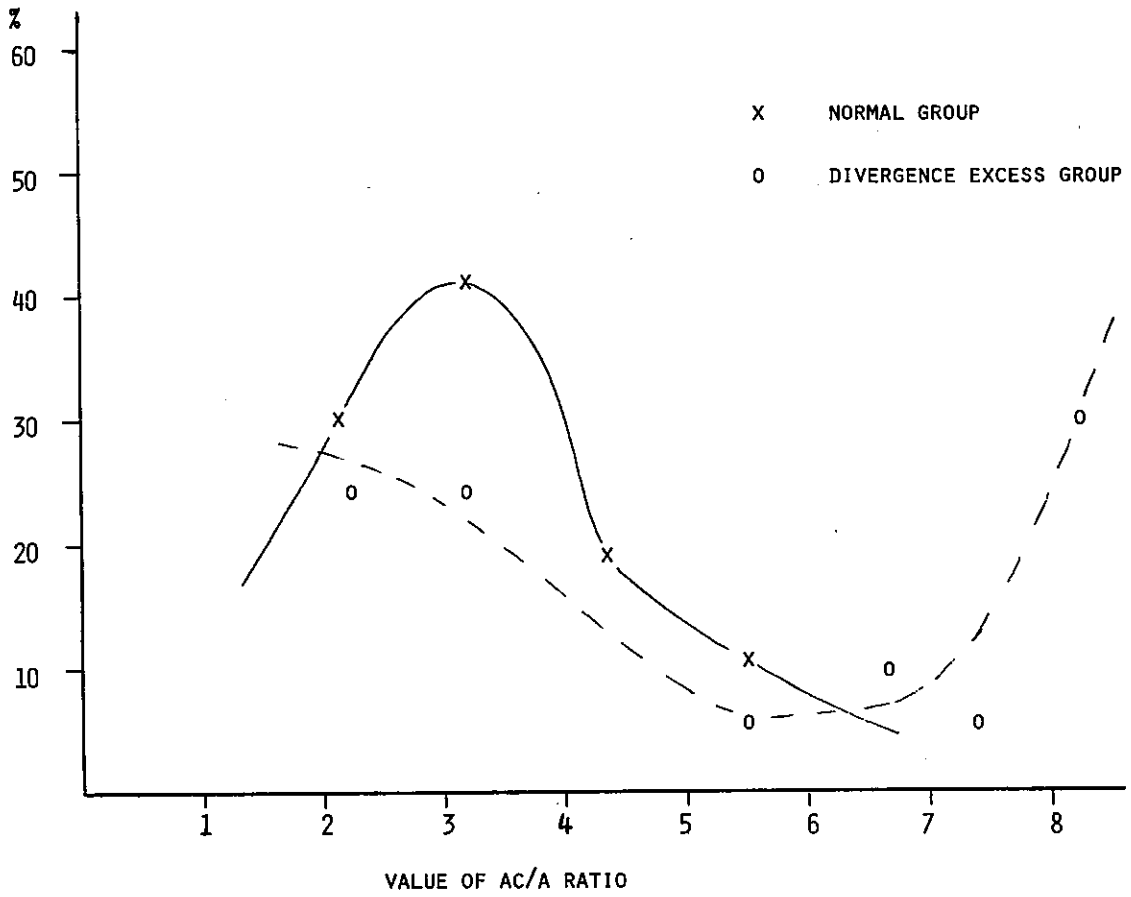

Figure 1: Distribution of AC/A values for each group.

some intermittent exotropes of divergence excess control a large near angle with accommodative convergence.

It is obvious, however, that not all intermittent squints of this type have a high AC/A ratio, and therefore achieve control at near fixation by other means. It is likely that fusional convergence must be another controlling factor, but discussion of this would require further research.

The existence of a low AC/A ratio is not necessarily inconsistent with an intermittent exotropia of divergence excess as often a reasonably large exophoria may be noticed in these cases when cover testing at ⅓ m. It is likely that this angle is controlled by strong fusional and proximal convergence mechanisms.

Knowledge of a high AC/A ratio is valuable when considering orthoptic management by minus overcorrection in intermittent exotropia of divergence excess. Accurate assessment of the AC/A may aid in the selection of suitable candidates for minus lenses, ensuring that those patients who cannot possibly benefit from this type of treatment are managed surgically and not subjected to an unproductive treatment period, where vision may be blurred and sensory adaptations reinforced, before eventual surgery is performed.

The AC/A is also an important consideration when surgery is contemplated. Failure to expose the masked near deviation may result in inadequate surgical correction, reducing the functional

result and increasing the possibility of post surgical redivergence, particularly with the onset of presbyopia when accommodative convergence is no longer functioning to maintain binocularity. Often the usual bilateral lateral rectus recession is insufficient for the intermittent exotrope of divergence excess with a high AC/A and a recess-resect procedure is indicated.

Despite the importance of the accommodative-convergence mechanism in many forms of strabismus, the AC/A ratio remains a useful piece of clinical information seldom obtained. Clearly a high AC/A ratio has a great significance with regard to aetiology and management of some intermittent exotropes of divergence excess.

ACKNOWLEDGEMENTS

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HUMAN COLOUR VISION: ITS BASIS AND CLINICAL SIGNIFICANCE

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Abstract

To understand abnormalities in colour vision a full knowledge of the properties of light, the eye and the brain, all of which are responsible for colour vision is necessary.

This paper presents a review of the physiology of colour vision and gives a detailed explanation of colour vision abnormalities.

Key words: *Trichromatic colour vision, dichromatic colour vision, monochromatic colour vision, deutan, protan, tritan.*

INTRODUCTION

In order to understand any abnormality in colour vision it is necessary to have a full knowledge of the properties of light, the eye and the brain all of which are responsible for colour perception.

This paper explains normal colour vision and gives a detailed explanation of colour vision abnormalities.

Light is the physical stimulus that initiates the visual process. It is a form of electromagnetic energy which is radiated (or emitted) from a source such as the sun or an electric light. The source releases particles of matter (photons) as an electromagnetic vibration. These particles vibrate at various different frequencies and the reciprocal of the frequencies occur is refered to as the 'wavelength'. The wavelengths of light that are visible to the human eye range from 380nm to 750nm (1 nanometer, nm = 1 millionth of a millimeter or 10^{-9} meter) and form the visual spectrum. Wavelengths shorter than 380nm (ie particles that vibrate too quickly to stimulate cone receptors and thus be seen by the eye) include ultraviolet rays and X-rays. Those wavelengths longer than 750 nm include infra-red rays and radiowaves. (These particles vibrate too slowly to stimulate cones and be seen by the human eye).

In the normal eye light within the visual spectrum excites rod and cone photoreceptors. These in turn excite colour coding cells in the retinal ganglion cell layer, the lateral geniculate nucleus (LGN) and the occipital cortex allowing the person to experience colour vision.

It is commonly believed that the colour of an object depends on the pigment and texture of its surface. This is untrue. The colour of an object depends on the following:

(1) the light source (eg. the sun or an electric light). As slightly different wavelength light is emitted from different sources the exact colour of an object will vary under different light sources.
(2) the dominant wavelength of light emitted from the source that bounces off the object. When light emitted from any source hits an

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object some wavelengths are absorbed by the object and others are reflected from (bounce off) it into the eye. One dominant wavelength is always reflected from an object.

Objects which absorb most of the light which strike them appear to be black. Those which reflect all the light appear to be white. Grey objects partially absorb the same proportion of light at each wavelength. Coloured objects selectively absorb various wavelengths and transmit others hence colour depends on the dominant wavelengths of light transmitted or reflected from the object. For example, a green chair absorbs all wavelengths of light except green light. Green wavelengths are reflected from the chair into the eye. The mixture of pigments which gives green is called a SUBTRACTIVE colour mixture. When white light hits the chair both the very short and the very long wavelengths are absorbed. By absorbing these wavelengths the pigment on the chair is virtually subtracting the short and long wavelengths from the white light. Thus the only wavelengths left to be reflected are the mid wavelengths so the chair appears green.

The other type of colour mixture is known as ADDITIVE colour mixture. Physical superimposition of different wavelengths of light on the same points on the retina is known as additive colour mixing. This affect can be achieved by projecting blue, red and green (ie the primary colours) from three projectors onto a white screen. By varying the brightness of each colour (using neutral density filters) all the different hues that can be perceived by the human eye may be produced.

The light source and the dominant wavelength of light which bounces off the object are the only objective factors affecting its colour. The rest of the colour interpretation depends on physical factors (the eye, the brain etc) and the individuals past perceptual experience.

The colour that the human brain perceives an object to be depends on the following[1]:

(1) the eye itself. The media must be normal allowing a clear passageway for wavelengths of light to reach the retina.

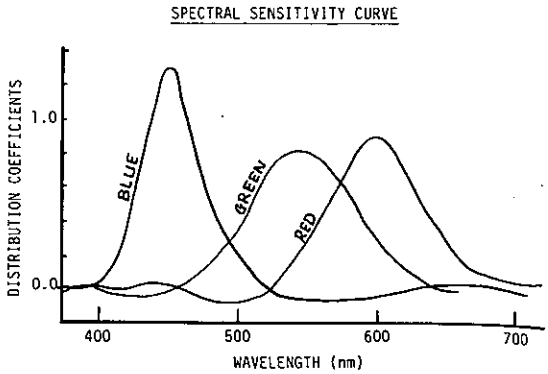

Figure 1: Spectral sensitivity curve.

(2) all retinal receptor cells and neural cells must be intact and functioning normally.
(3) neural pathways from the eye to the brain must be functioning normally.
(4) the individual must have past perceptual experience on which to base their interpretation of the stimulus.

Although the eye must be functioning normally in order to perceive colour, the eye itself does NOT recognise colour. Colour interpretation occurs in the brain. However, the eye must recognise the different wavelengths of light that enter it. Just as various wavelengths of sound stimulate the human ear differently enabling us to hear different noises, various wavelengths of light entering the eye stimulate cone photoreceptors differently. This ultimately enables us to see different colours.

When white light (ie a combination of all wavelengths of light from 380nm to 750nm) passes through a medium that is denser than air it is refracted or bent by that medium. As short wavelength (quickly vibrating) particles are bent more than slowly vibrating long wavelength particles white light passing through a prism is dispersed into the colours of the spectrum. This phenomenon was first reported by Newton[2].

The six colours (red, orange, yellow, green, blue and violet) that a prism breaks white light up into are known as SPECTRAL colours. All other colours such as pinks and browns are known as NON SPECTRAL colours.

ANATOMY

Cone receptor cells

When entering the eye light passes through the media and the outer retinal layers to the layer of rods and cones (ie the photoreceptors). Light is absorbed by the photoreceptors and this is the first stage in the perception of colour.

There are estimated to be 7 million cones in the human retina.[3] Light is absorbed by the pigment within each cone. Although little is known about cone pigments, using microspectrophotometry, researchers[4,5] have established that there are three varieties of photopigment. Each individual cone responds to all wavelengths of visual spectrum light however the response differs as each cone contains one of the three different photopigments. Thus the individual cone is maximally stimulated by (or maximally absorbs) either short wavelength 450nm blue light (ie 'blue' cones), mid wavelength 540nm green light ('green' cones) or long wavelength 590nm red light ('red' cones). Therefore a cone is named after the wavelength of light which the cone pigment it contains maximally absorbs.

The sensitivity of an individual cone to a particular wavelength of light is known as spectral sensitivity. The spectral sensitivity of the individual cone pigments to the various wavelengths of light is clearly shown on the spectral sensitivity or luminosity function curve (see Figure 1).[6] This curve represents the absorption function of the three cone pigments.

The difference in spectral sensitivities of the individual cone pigments is essential for normal colour perception. Without it all wavelengths of light would be absorbed equally by cones thus different wavelengths reflected from objects would produce only differences in brightness and the world would look like the picture on a black and white television set.

RETINAL GANGLION CELLS

As the cone receptors are not colour specific there must be neural mechanisms that can contrast the relative amounts of absorption of the three cone pigments in order to colour match. When light maximally stimulates red cones for example it causes these cones to send an excitatory input to the retinal ganglion cells. The same wavelength of light causes an inhibitory response to be sent from the green and blue cones to the retinal ganglion cells. In the retina the mechanism for colour coding consists of two groups of ganglion cells:

(1) spectrally opponent cells[7]
(2) spatially opponent cells[8]

Red and green are mutually exclusive colours. In daily life we see red-yellows, red-blues, greeny yellows and greeny blues however we never see red-greens or blue-yellows. This is because red and green are mutually exclusive colours. Blue and yellow are the only other pair of mutually exclusive colours. In the literature these colours are described as 'opponent' colours.[7,8]

(I) Spectrally opponent ganglion cells

After staring at a red spot on a white background for a number of minutes then looking away, the after image of the red spot is a green spot. This occurs because red and green are SPECTRALLY OPPONENT colours.

There are two sets of spectrally opponent colours in the human visual system. They are red — green and blue — yellow. Each individual spectrally opponent ganglion cells is thought to have both an excitatory and inhibitory field thus each individual ganglion cell is excited by stimulation of one type of cone photoreceptor and inhibited by another.

In 1975 De Valois and De Valois[7] reported that there were 4 types of spectrally opponent retinal ganglion cells. These cells are:

(1) red (long wavelength) excitatory — green (mid wavelength) inhibitory (R+ and G−)
(2) green excitatory — red inhibitory (G+ and R−)
(3) blue excitatory — yellow inhibitory (B+ and Y−)
(4) yellow excitatory — blue inhibitory (Y+ and B−)

Red — green cells would for example be excited by input from red cones that were stimulated by long wavelength light and inhibited by input from green cones that were stimulated by mid wavelength green light (see Figure 2).

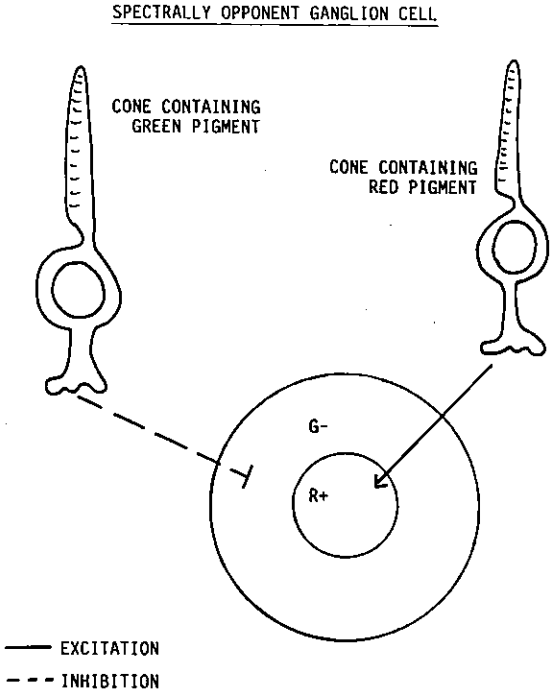

Figure 2: Spectrally opponent retinal ganglion cell.

(II) Spatially opponent retinal ganglion cells
These ganglion cells are also thought to have an excitatory and an inhibitory field. However, they respond to increases and decreases in surrounding light.

There are two types of spatially opponent retinal ganglion cells. They are:

(1) black excitatory — white inhibitory (Bl+ and Wh−)
(2) white excitatory — black inhibitory (Wh+ and Bl−)

These cells enable us to perceive objects clearly in the absence of colour stimulus; for example when watching black and white television, objects are perceived individually.

As the receptive field organization of the retinal ganglion cells is similar to that of the LGN neural coding of colours occurs in much the same way in the LGN.[6] Little is known about the mechanisms involved with colour vision in the visual cortex, however it is suspected on the basis of work done on the Rhesus monkey cortex[9] that neural coding occurs in much the same manner in the visual cortex as it does in the retinal ganglion cells.

NORMAL COLOUR VISION

Colour perception in individuals who use all three cone photopigments to colour match is called normal TRICHROMATIC colour vision (i.e. Young-Helmholtz trichromatic theory). Such individuals mix the three primary coloured lights in various proportions to perceive all the colours in the spectrum.

ABNORMAL COLOUR VISION

There are two major subgroups of colour vision abnormalities (excluding colour blindness). These are:

(1) anomalous trichromatic colour vision (deuteranomaly, protanomoly and tritanomaly).
(2) dichromatic colour vision (deuteranopia, protanopia and tritanopia).

(The prefix deut- means green, prot- means red and trit- means blue. The suffix -anomaly means there is an anomaly of pigment whereas -anopia refers to an absence of pigment).

(I) Anomalous trichromatic colour vision
These patients have cones containing the three photopigments, however they use abnormal proportions of these to colour match. This is a congenital condition which is associated with normal visual acuity and no other ocular problems.[10] Usually the patients are unaware of any colour vision defect.

These patients are labelled 'colour blind' and this term is extremely confusing. The patients do see colours, they just perceive them slightly differently. For example, if an orange block and a yellow block were placed on the table in front of an anomalous trichromat (protanope — see below) and the patient was asked to pick up the orange block, he would most probably select the yellow block stating that it was orange.

Most patients with anomalous trichromatic colour vision have red-green colour abnormalities. This abnormality occurs in 8% of the male

population.[10] Congenital blue-yellow colour abnormalities are extremely rare.[10]

Red-green 'colour blindness' can be further subdivided into:

(a) deuteranomalous 'colour blindness' (green deficiency)
(b) protanomalous 'colour blindness' (red deficiency)

Patients with blue-yellow defects are said to have a tritanomalous defect.

DEUTERANOMALOUS COLOUR VISION

These patients are labelled as 'green blind'. Despite the fact that they have a red-green deficiency, they CAN DISTINGUISH pure greens and pure reds. On colour vision testing these patients perform as though they are using more green to colour match. This can be explained in terms of a shift in the spectral sensitivity curve towards longer wavelengths (i.e. to the right).

As a result deuteranomalous patients have most trouble distinguishing colours in the yellow, orange and blue sections of the colour circle, that is the colours that are a mixture of red and green.

PROTANOMALOUS COLOUR VISION

Society calls these patients 'red blind'. Once again they have a red-green defect but have no problem distinguishing pure reds and pure greens. On colour vision testing, these patients perform as though they are using more red to colour match. This can be explained in terms of a shift in spectral sensitivity towards the shorter wavelengths. In performing the same test with colour blocks, if asked to select the YELLOW block the protanomalous subject would pick the ORANGE block. Protanomalous subjects have most problems distinguishing between yellows, oranges and blue colours. In dim lights these subjects may have problems distinguishing long wavelength reds.

TRITANOMALOUS COLOUR VISION

This category of colour vision defect is extremely rare.[10] They are called 'blue blind' patients yet they have no trouble with either pure blue or pure yellow. Their problems occur with greens and reds.

Aetiology of Anomalous Trichromatic Vision

The hypothesis which explains the aetiology of anomalous trichromatic colour vision suggests that these patients have a slight abnormality in the cone pigments causing them to maximally absorb slightly shorter (protanomaly) or slightly longer (deuteranomaly and tritanomaly) wavelength light.[10]

Dichromatic Colour Vision

This condition may be congenital or acquired. When it is congenital the patients usually have normal vision and no other ocular problems. Once again these patients are said to be colour blind but in fact they are not. Patients with dichromatic colour vision abnormalities can be subdivided into three groups:

(a) deuteranopia — green 'blind'
(b) protanopia — red 'blind'
(c) tritanopia — blue 'blind'

DEUTERANOPIA AND PROTANOPIA

The nomenclature of these abnormalities is more appropriate because the patients do have problems with red-green discrimination and they cannot distinguish green from reds yet show no confusion between yellow and blues. All colours appear as various saturations of blue and yellow or as grey or black. The deuteranopic patient frequently describes the green object as black and the protanopic patient may describe a red object as black. In either group red and green may be confused with yellow when tested on the anomaloscope.

TRITANOPIA

These patients cannot distinguish blues from yellows yet they show no confusion between reds and greens. Sometimes they may confuse blue with grey when tested on the anomaloscope.

Aetiology of Dichromatic Colour Vision

There are two hypotheses as to the cause of dichromatic colour vision. The first, and by far the most popular is that the patients have a

normal population of cones all of which contain only two of the three cone pigment[10,11] (i.e. either the red, green or blue cone pigment is missing). As a result the patient colour matches using only two rather than the three cone pigments. This hypothesis fails to explain acquired dichromatic colour vision resulting from disease or injury to the higher neural centres not affecting the cones. Some authors suggest acquired dichromatic colour vision is thought to be due to post receptoral loss of colour discrimination in one of the two pairs of opponent neural colour coding cells either in the ganglion cell layer, the LGN or the visual cortex.[12] Protanopic and deuteranopic individuals would have loss of red-green opponent neural colour coding cells and tritanopic individuals would have loss of blue- yellow opponent colour coding cells.

COLOUR BLINDNESS

Monochromatic Vision

Individuals with monochromatic vision are the only group of so called 'colour blind' patients who ARE actually colour blind. They see the world in black, white and shades of grey.

There are several subgroups within the monochromatic group. They include;

(1) Cone monochromatism:
    simple cone monochromats
    blue cone monochromats
(2) Rod monochromatism:
    partial rod monochromats
    total rod monochromats

CONE MONOCHROMATISM

(a) Simple cone monochromatism: Patients have good foveal vision, no nystagmus and no photophobia. Their only visual abnormality is their total colour blindness[10].

Simple cone monochromatism is thought to occur because of a loss of both red-green and blue-yellow opponent ganglion cells.

(b) Blue cone monochromatism: This is an extremely rare condition in which there is decreased visual acuity.[10] The decreased visual acuity occurs because foveal cones do not respond to short wavelength light (i.e. blue light. It is suggested by the author that this situation arises because the macular pigment, Xyanthyll, absorbs all the short wavelength light before it reaches the layer of foveal cones). Patients with blue cone monochromatism have nystagmus and a central scotoma. This condition is thought to occur due to a congenital absence of both green and red cone pigments.

ROD MONOCHROMATISM

(a) Partial rod monochromatism: This is a rare condition in which the patient presents with extreme photophobia, nystagmus (which improves with age), very decreased visual acuity for distance, slightly reduced visual acuity for near (N8, N12) and total colour blindness.[10]

Partial rod monochromatism is thought to be due to either a congenital decrease in the number of functioning cones in the retina.[13,14] Some authors suggest that there may be a number of rods in the foveal area.[15,16,17] The author suggests that foveal cones may actually contain rhodopsin (the rod pigment), in which case it would be interesting to speculate on the performance of receptors containing rhodopsin connected to ganglion cells in a one to one relationship.

(b) Total rod monochromatism: This is an extremely rare condition. Signs and symptoms are similar to but more severe than those for partial rod monochromatism. Patients with total rod monochromatism are believed to have no functioning cones in their retina.

DISCUSSION

An understanding of the basis of colour vision is vital if clinical colour vision tests are going to be applied correctly. Also, correct interpretation of the results of such tests cannot be made without a complete understanding of the basics.

It is also important that the clinician is able to explain any abnormality in colour vision to the patient or parent. For example the parent should understand why their child who has been called red-green colour blind can actually see reds and greens. Also parents should be aware of some employment opportunities which may be limited due to defective colour vision.

CONCLUSIONS

There are many fallacies commonly associated with colour vision abnormalities. The term 'colour blindness' itself is extremely misleading as only a very small percentage of the population (approximately 0.009%[10]) have no perception of colour. Up to 8% or 9% of the population are described as being 'colour blind' yet they do see all the colours of the visual spectrum. These patients would be better described as having an alteration in their perception of colour.

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EVALUATION OF THE CITY UNIVERSITY COLOUR VISION TEST

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Abstract

The use of the City University Colour Vision Test for routine screening for colour vision abnormalities is evaluated. The results of screening for colour vision abnormalities using the City University Test are compared to results obtained when the patients are tested with the Ishihara test and Farnsworth Munsell Panel D 15 test. All patients in the study were also tested on the Farnsworth Munsell 100 Hue Test to confirm the presence and severity of the color vision defect. The City University Test was found to be the most reliable of the three screening tests used in this study for detecting congenital and acquired red green and/or blue yellow defects however it appears to be of limited value in testing patients with optic nerve disease.

Key words: *City University Test, colour vision, anomalous trichromat, congenital/acquired colour vision abnormality, Farnsworth Munsell 100 Hue/D-15, Ishihara.*

INTRODUCTION

The most common colour vision abnormality is congenital red green colour blindness (anomalous trichromatism).[1] As a result most simple colour vision tests have been designed to primarily test for this defect ignoring the other defects, especially the acquired colour vision anomalies. Common examples of tests designed to detect congenital red green anomalies only include the Ishihara, the Matsubara Children's Colour Vision Test and the Standard Pseudoisochromatic Test. The advantages of these tests are that they are very quick and easy to perform for both the patient and the examiner. The major disadvantage, which far outweighs the advantages, is the fact that patients with acquired colour vision abnormalities or congenital blue yellow defects pass these tests and thus appear to have no colour vision abnormality.

To date, in our department the Farnsworth-Munsell 100 Hue (FM 100 Hue) Test has been used to investigate patients with suspected colour vision abnormalities other than congenital red green defects. This test is one of the most thorough colour vision tests available as it will detect any congenital or acquired colour vision anomaly. The major disadvantage of the test is that it takes up to 45 minutes to test, score and print out the results from one patient.

As a result of the time involved in using the FM 100 Hue Test and the relative inaccuracy of the other vision tests mentioned, we decided to use the City University Colour Vision Test. The City University Test is quick and easy to perform and it is designed to test for all types of colour vision abnormalities. To date eighty patients have been tested in our department using the City University Test.

METHOD

City University Test

The test contains 11 plates including one control plate. Each plate consists of one central circle surrounded by four other circles — (see figure

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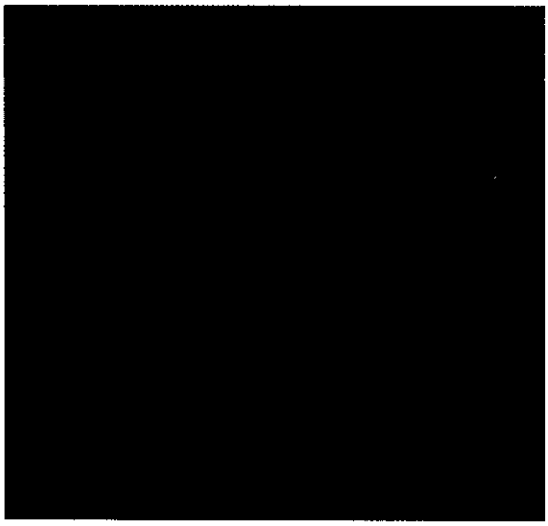

Figure 1: City University test plate 1. (Reproduced from the City University Colour Vision Test.)

1). Plates one to six have coloured circles 8 mm in diameter and plates seven to ten have 4 mm diameter coloured circles. On each plate one of the surrounding circles is very similar in colour to the central circle and the other three are different colours. The patient is simply asked to look at the central circle and then to tell the examiner which one of the four surrounding circles is MOST SIMILAR in colour to the central one (i.e. the circle to the top, bottom, right or left of the centre circle).

Initially the patient is tested with the control plate. This plate is designed so that achromatic (totally colour blind) observers can respond correctly. If, even after re-explanation, the patient responds incorrectly, there is nothing to be gained by continuing the test. Once the patient has responded to the control plate the examiner continues with plates one to ten. The patient's responses are recorded on the score sheet (see figure 2). To score the test the examiner simply circles the response the patient gives for each plate. For example, in figure 2 the patient gave a normal response. In figure 3 the results clearly indicate that the patient has the red green defect, protanomaly.

Plates one to six (called 'chroma four' plates) are not excessively difficult, hence patients with

CITY UNIVERSITY COLOUR VISION TEST (2nd Ed. 1980)

Address DESJ1 Patient 9003675

Examiner AF Male/~~Female~~ Date 17/7/1985

Spectacles worn? YES/(NO) (RE)/LE/BE

Illumination ("Daylight") Type Daylight Globe level

FORMULA: Here are 4 colour spots surrounding one in the centre. Tell me which spot looks most near in *colour* to the one in the centre. Use the words "TOP", "BOTTOM", "RIGHT" or "LEFT". Please do not touch the pages.

	PAGE (A is for demonstration)	SUBJECT'S CHOICE OF MATCH R / L / Both	NORMAL	DIAGNOSIS PROTAN	DEUTAN	TRITAN
"CHROMA FOUR"	1		(B) ◇	R	L	T
	2		(R) ◇	B	L	T
	3		(L) ◇	R	T	B
	4		(R) ◇	L	B	T
	5		(L) ◇	T	B	R
	6		(B) ◇	L	T	R
"CHROMA TWO"	7		(L) ◇	T	R	B
	8		(R) ◇	L	B	T
	9		(B) ◇	L	T	R
	10		(T) ◇	B	L	R
SCORE	AT CHROMA FOUR		6/6	/6	/6	/6
	AT CHROMA TWO		4/4	/4	/4	/4
	OVERALL		10/10	/10	/10	/10

Probable type of Daltonism P; PA, EPA MIXED D, DA, EDA TRITAN

Figure 2: Normal result from City University Test.

CITY UNIVERSITY COLOUR VISION TEST (2nd Ed. 1980)

Address SPA G1 Patient 9003456

Examiner ~~Male/Female~~ Date 10/4/1985

Spectacles worn? YES/(NO) (RE)/LE/BE

Illumination ("Daylight") Type Daylight Globe level

FORMULA: Here are 4 colour spots surrounding one in the centre. Tell me which spot looks most near in *colour* to the one in the centre. Use the words "TOP", "BOTTOM", "RIGHT" or "LEFT". Please do not touch the pages.

	PAGE (A is for demonstration)	SUBJECT'S CHOICE OF MATCH R / L / Both	NORMAL	DIAGNOSIS PROTAN	DEUTAN	TRITAN
"CHROMA FOUR"	1		(B) ◇	R	L	T
	2		R ◇	(B)	L	T
	3		L ◇	(R)	T	B
	4		R ◇	(L)	B	T
	5		(L) ◇	T	B	R
	6		B ◇	(L)	T	R
"CHROMA TWO"	7		L ◇	(T)	R	B
	8		R ◇	(L)	B	T
	9		B ◇	(L)	T	R
	10		(T) ◇	B	L	R
SCORE	AT CHROMA FOUR		2/6	4/6	/6	/6
	AT CHROMA TWO		1/4	3/4	/4	/4
	OVERALL		3/10	7/10	/10	/10

Probable type of Daltonism P; PA, EPA MIXED D, DA, EDA TRITAN

Figure 3: Protan result from City University Test.

only mild colour vision defects may get most of these plates correct. In plates seven to ten ('chroma two' plates) it is more difficult to differentiate the colours thus it is more difficult to respond correctly. Hence, if a patient makes two or more errors on any of these 4 plates, they are said to have a colour vision abnormality.

This test may be performed monocularly or binocularly depending on the type of colour vision defect suspected (congenital defects are usually bilateral and symmetrical, hence the test may be performed binocularly). If possible the test should be performed under a daylight globe (6740° Kelvin). According to the instruction booklet patients should be given three seconds per plate, thus to perform the test monocularly, score the test and interpret the results takes approximately three to four minutes per patient.

Fifteen patients with colour vision defects included in the results of this paper were tested using the City University test together with the Ishihara test, the Farnsworth Munsell Panel D-15 Test (FM D-15) and the FM 100 Hue. Details of the testing procedures used in these three other tests have been outlined in a previous publication by the author.[2]

Patients reported in this study have defects including anomalous trichromatic colour vision (5 cases), cone and/or rod dystrophy (5 cases), optic nerve anomaly (4 cases), and macular dystrophy (1 case).

RESULTS

Anomalous trichromats: (cases 1, 2, 3, 4, 5)

Two cases of anomalous trichromatic colour vision (cases 1 and 5) demonstrated their abnormality on all four colour vision tests. Cases 2 and 4 showed anomalous trichromatic colour vision on all the colour vision tests except the FM D-15.

Case 3, a mild anomalous trichromatic patient appeared to have normal colour vision when tested on the Ishihara, and showed only one error on the FM D-15, a normal result. However, when tested with the City University Test, a red green anomaly (deuteranomaly) was apparent. (Responses to plates one to six were normal, however plates seven to ten all demonstrated deuteranomalous defect). The patient also demonstrated mild deuteranomaly on the FM 100 Hue Test (with a score of 320).

Retinal Dystrophies: (cases 6, 7, 8, 9 and 10)

Cases 8 and 10 showed achromatopsia (total colour blindness) on all tests. Cases 6 and 9 showed a blue yellow defect on all tests except the Ishihara. This was expected as the Ishihara does not test for blue yellow defects.

Case 7 passed the Ishihara Test, however showed a red green defect on the other tests.

Optic Nerve Abnormalities: (cases 11, 12, 13 and 14)

In all cases of optic nerve anomalies, the patients performed as normals on both the Ishihara and the City University tests. The colour vision abnormalities were apparent on both the FM D-15 and the FM 100 Hue in all cases. (It must be noted that this is only a small sample of patients with optic nerve dysfunction.)

Macular Dystrophy: (case 15)

In this case only the FM 100 Hue response was abnormal. The results of the other tests were normal.

DISCUSSION

The results of this paper have indicated that the FM 100 Hue was the only one of the four tests to demonstrate the colour vision abnormality in all cases. The reliability of the other three tests will now be compared.

As expected, most patients with acquired colour vision anomalies showed normal results when tested with the Ishihara Test. (The Ishihara is only designed to detect congenital red green colour vision anomalies).

Four of the five (80%) cases of congenital anomalous trichromatic colour vision showed a red green defect on the Ishihara Test. The defect was also apparent in all cases on the City University Test and the FM 100 Hue. It was interesting to note that three (60%) of the 5 cases (cases 2, 3 and 4) performed normally on the FM D-15. (These three cases were only mild anomalous trichromats when tested on the other tests). Thus the City University Test and the

TABLE 1: SUMMARY OF RESULTS (R/G = red green; B/Y = blue yellow)

CASE NO & DIAGNOSIS	COLOUR VISION TEST RESULTS			
	CITY UNI	ISHIHARA	FM D-15	FM 100 HUE
1 ANOMALOUS TRICHROMAT	R/G defect	R/G defect	R/G defect	R/G defect
2 ANOMALOUS TRICHROMAT	R/G defect	R/G defect	normal	R/G defect
3 ANOMALOUS TRICHROMAT	R/G defect	normal	normal	R/G defect
4 ANOMALOUS TRICHROMAT	R/G defect	R/G defect	normal	R/G defect
5 ANOMALOUS TRICHROMAT	R/G defect	R/G defect	R/G defect	R/G defect
6 ROD/CONE DYSTROPHY	B/Y defect	normal	B/Y defect	mild general loss
7 RETINITIS PIGMENTOSA	R/G defect	normal	R/G defect	R/G defect
8 CONE DYSTROPHY	achrom-atopsia	achrom-atopsia	achrom-atopsia	achrom-atopsia
9 RETINAL DYSTROPHY	B/Y defect	normal	B/Y defect	B/Y defect
10 ROD MONOCHROMAT	achrom-atopsia	achrom-atopsia	achrom-atopsia	achrom-atopsia
11 OPTIC NEURITIS	normal	normal	B/Y defect	B/Y defect
12 OPTIC N HYPOPLASIA	normal	normal	mild general loss	mild general loss
13 OPTIC ATROPHY	normal	normal	mild general loss	mild general loss
14 OPTIC ATROPHY	normal	normal	mild general loss	mild general loss
15 MACULAR	normal	normal	normal	mild general loss

Ishihara are more reliable than the FM D-15 for screening for congenital anomalous trichromatic colour vision.

In the cases of retinal dystrophy the results obtained on the City University, FM D-15 and FM 100 Hue Tests were all compatible. The abnormality was only demonstrated using the Ishihara in two of the five cases. Thus the City University Test and the FM D-15 are the more reliable tests for screening for colour vision anomalies in cases of retinal dystrophy.

The City University Test was not found to be reliable for screening for colour vision anomalies in cases of optic nerve anomaly. All patients with

optic nerve anomaly showed normal results on both the City University and the Ishihara Tests, however the abnormality was apparent on the FM D-15.

To date only one patient with macular dystrophy has been tested with the City University Test. The colour vision abnormality was only apparent on the FM 100 hue Test.

One main advantage of the City University Test is that it can be used on a greater range of age groups than the other colour vision tests. In our department the City University Test has been used on children as young as five years old. In the author's experience young children tend to respond more readily to this test than to the Matsubara Test and similarly a number of children who have not been able to do the Ishihara have managed the City University Test. A survey conducted in 1984 in our department (unpublished) demonstrated that children between the ages of six and ten years are able to perform the FM 100 Hue Test. However the children showed an abnormally high average score of 370 on the test. Rather than implying that all children between the ages of six and ten have colour vision deficiency, this high score suggests that these children lack the ability to perform the FM 100 Hue Test accurately.

CONCLUSIONS

Although this is only a small sample, the preliminary results indicate that the City University Test is very reliable for screening for colour vision anomalies in all cases with the exception of patients with optic nerve abnormalities. In such cases the FM D-15 proved to be the most reliable screening test. (It must be noted that colour vision anomaly is associated with subclinical optic nerve disease. This can be grossly detected at the bedside without formal colour vision testing. This is because the patients suffer from colour desaturation, i.e. colours appear duller in the affected eye. Once an anomaly has been detected the examiner can proceed to more formal colour vision testing. However detection of colour vision anomaly in subclinical macular or cone disease requires formal testing.)

The City University Test is a reliable test to detect congenital and acquired red green or blue yellow colour vision defects. The Ishihara Test is more limited than the City University Test as it does NOT detect acquired and/or blue yellow defects.[3] The results indicate that the FM D-15 is not always reliable in detecting anomalous trichromatic colour vision or acquired colour vision abnormalities associated with cone disease.

Thus when looking for a test to quickly and accurately SCREEN for colour vision defects in the clinic in patients over the age of five years the results of this study suggest that in all cases except those with optic nerve disease the City University Test is the most reliable.

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ABSTRACTS OF STUDENT PAPERS

N.S.W.

The following are abstracts of research papers by third year orthoptic students at Cumberland College of Health Sciences, N.S.W. Copies of particular papers of interest may be obtained by writing to:
The School of Orthoptics,
Cumberland College of Health Sciences,
P.O. Box 170,
Lidcombe, N.S.W. 2141. Tel. (02) 646 6444.

DOES MUSCULAR IMBALANCE INFLUENCE THE CONVERGENCE NEAR POINT—**Susan Moore**

Ocular movements and convergence near point standards were investigated in forty subjects, 20 of whom had convergence near points of 5 cm or better from the eyes (Group 1), with the remaining 20 subjects having near points more remote than 5 cm (Group 2).

It was hypothesised that superior rectus and inferior rectus underactions caused a loss of adduction and resulted in a reduced convergence near point. It was found that there was an increased frequency of inferior rectus underactions in the group with the reduced near points. When statistically correlated, no significant relationship was found to exist between the muscle imbalance and convergence ability. However, a statistically significant difference was found between the deviations in elevation of each group — with greater exo deviations in those subjects with reduced near points.

A NEW TECHNIQUE FOR ASSESSING MICROSACCADIC EYE MOVEMENTS—**Mary Haddad**

This study set out to establish a set of normal responses that can be given to the new testing procedure, the "yellow block" test, that is said to assess microsaccadic eye movements, and whether these responses vary in any way with regard to the age of the subject.

The research involved the participation of 39 children, ranging from 8 to 14 years of age. A preliminary assessment was performed on each child to establish whether there were any eye defects present. The children were then tested with the new apparatus. Once the required viewing time of the block (60 seconds) had elapsed, the child was asked a series of questions regarding as to what they had observed during that time. The responses to these questions were then categorized into four groups, ranging from those who saw no changes taking place during testing time, to those who observed definite changes. It was then determined whether there was a relationship between the age of the child and each of the groups. It was found that a subtle relationship existed between these two factors, that is, the older children tended to see little or no change taking place in the block. It would be of great interest to test dyslexic children with this new procedure, to determine whether these children have defective microsaccede movements.

PUPILLARY ABNORMALITIES IN AMBLYOPIA—**Hetty Cremers**

Two groups of patients, an amblyopic group and a normal group were tested to see if there was any connection between afferent pupillary defects and amblyopia. The pupil defect observed was one of a delay in constriction to a repeated direct light stimulus of the amblyopic eye.

The pupillary defect was found more frequently in the amblyopic group of patients when compared to the normal group of patients. Within the amblyopic group, a correlation between the level of vision of the amblyopic eye and the presence of a pupillary defect was shown to be significant. It was noted that of the amblyopia group (whose visual acuity ranged from 6/6-2 to 1.5/60), of those with acuities of 6/6 to 6/12, 75% showed a pupillary defect.

REDUCED CONTRAST SENSITIVITY IN MULTIPLE SCLEROSIS: AN EVALUATION OF MODIFIED SNELLENS CHARTS USING REDUCED CONTRAST—**Christine Maple**

Reduced contrast sensitivity in multiple sclerosis has been demonstrated previously by previous researchers using special gratings and various forms of electrophysiological tests. The main objective of this research was to evaluate the use of standard Snellens visual acuity charts of varying contrast, as a method for distinguishing between normals and those individuals with multiple sclerosis.

All three visual acuity charts used showed statistically significant difference between the two groups. Twenty one of the thirty four multiple sclerosis eyes tested, which presented with normal vision for their age on 100% contrast charts, showed significant reductions in contrast sensitivity.

A VISUAL MOTILITY ASSESSMENT OF STUTTERERS—**Terri Leverty**

Twenty six stuttering and twenty six non stuttering subjects were assessed, to determine whether visual and motility standards between the two groups differed.

Eye movements, particularly saccades, were investigated because of the close association of speech and saccadic controlling centres. The assessment included saccades, smooth pursuit, muscle balance, V.A for near and distance, the presence of a latent or manifest deviation, stereo-acuity and responses of pupils to light and accommodation.

Results demonstrated that the only statistically significant difference between the two groups was stereo-activity, where non stutterers demonstrated a higher level. Throughout the study however, the non stutterers demonstrated a consistent and higher standard of visual and motility function, suggesting that further study in this area is warranted.

EFFECT OF OCULAR PROBLEMS AS SEEN THROUGH CHILDREN'S DRAWINGS—**Amparo Herrera**

Fifty patients between the ages of 3 to 14 years, were selected for this study, in order to determine the incidence of children who, by means of drawings of themselves, expressed ocular problems such as strabismus or glasses.

Analysis of these drawings was performed by two psychologists, experienced in the field of children. Out of 50 drawings, 22 children (Group A), actually had ocular problems, 12 of these (or 54.55%), depicted such "ocular problems" in their drawings, while the remaining 10 (or 45.45%), did not. Twenty eight children (Group B), made up the control group, that is, children who had no past or present history of ocular problems. None of these children demonstrated any particular abnormality in their graphic expressions.

It was found that the presence of ocular problems plays a significant role in the child's perception of him/her self. Because the child's drawing is original and individual, one can determine the specific and different problem which affects the child, especially if the researcher carefully observes the child while he is performing the task.

DISSOCIATED VERTICAL DEVIATION IN CONGENITAL STRABISMUS—**Lisa Oakley**

Dissociated vertical deviation (D.V.D.), is a condition often associated with congenital strabismus. A retrospective study of 70 patients with congenital strabismus and D.V.D. was performed, to determine the average age of onset of D.V.D. whether the onset was affected by early surgery, and whether nystagmus, the vertical deviation or abnormal O.K.N. were the first clinical signs noted.

Of these 70 patients, 37 had developed D.V.D. before any surgery was carried out (or who had no surgery at all) and 33 developed D.V.D. post-operatively. The difference between the average age of onset of D.V.D. in the two groups was found to by insignificant in both groups. A D.V.D. movement of the affected eye was the first characteristic noted in the majority of patients.

LONG TERM EFFECTS OF CONVERGENCE INSUFFICIENCY TREATMENT—**Jennifer Gleeson**

A survey was conducted amongst patients who had received and completed a full course of convergence insufficiency treatment three to six years before the time of their study.

The aim was to establish the effect, if any, of the following two variables on the recurrence of symptoms:

1. whether the patient kept a regular self check on convergence after treatment
   and
2. whether the patient can voluntarily converge.

A relationship was established in this study, although it was realized that patients were satisfied with treatment when symptoms returned, which is therefore considered to be an important positive affect of orthoptic treatment.

HEMIANOPIC RESOLUTION IN CEREBRAL VASCULAR ACCIDENTS (CVA)—**Darren Banks**

This study was undertaken to assess whether hemianopic defects caused by C.V.A.s will recover spontaneously, as do hemiplegias.

Eight subjects from the C.V.A. unit of Lidcombe Hospital had their visual fields assessed soon after a C.V.A. These subjects were then monitored for six months, to see if the field defect recovered spontaneously.

Results of this study showed that spontaneous recovery was negligible over this six months' period. A suggestion is made for a rehabilitational treatment plan to possibly aid in the resolution of visual field defects.

THE INCIDENCE OF CONGENITAL ABNORMALITIES IN CONGENITAL EXOTROPIA AND ESOTROPIA—**Nadia Nicotra**

Twenty five subjects with congenital exotropia and twenty five with congenital esotropia were investigated through data from medical histories.

The incidence of major congenital abnormalities amongst the two groups were compared. Results suggest that major abnormalities, such as cerebral palsy, hydrocephalus, brain damage, meningitis and prematurity, are more common in the exotropic group.

EFFECTS OF ECCENTRIC VIEWING TRAINING—**Ingrid Ca'ceda**

A study was conducted on the effect of 'Eccentric Viewing Training' on visual acuity, time taken to achieve each visual acuity and activities of daily living.

The sample consisted of four patients, who were examined before and after the completion of training. Three of the four subjects showed an improvement in visual acuity and recognition time with changes in seven different aspects of tasks of daily living. This was confirmed by the subjects' satisfaction of their improvement. The fourth subject, however, showed adverse results; i.e. both visual acuity and recognition time dropped to lower levels after training, and there were no changes in the performance of activities of daily living. This subject, after training, was confused and became depressed.

It can be seen that factors such as medical health and motivation have great influence on the success of the training.

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On a separate page give a brief abstract of no more than 150 words, giving specific facts, findings, conclusions or opinions. Key words (about 5) or short phrases to assist indexers in cross-indexing the article, should follow the abstract on the same sheet. Key words should not duplicate words in the title but should be mentioned in the abstract.

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* Refer Med J Aust 1982; Dec 11/25, or apply to the Editor for a copy.