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THE ORTHOPTIC ASSOCIATION OF AUSTRALIA - 50 YEARS

(OAA Presidential Address - 1993)

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On 1st March 1944 a group of orthoptists held a meeting in Emmie Russell's rooms in Macquarie St, Sydney to discuss the foundation of an association for Australian orthoptists. Later that year the Articles of the emerging Orthoptic Association were signed by 14 orthoptists and we are honoured to have 3 of those orthoptists at this conference. On 26th September 1944 the Orthoptic Association of Australia was declared inaugurated.

Today, as we embark on our 50th Annual Scientific Meeting we will be entering our 50th year. In researching the history of the OAA I came across a commentary about our 21st year. It was written in 1964 by one of the founding members of the OAA, Mrs Diana Craig. I quote:

"The meeting for 1963 having been duly recorded as the 20th, plans went ahead for a (21st) birthday dinner on Saturday 4th April, 1964 and birthday cards were sent to foundation members. But our president, investigating past history in honour of the occasion, learnt that the first AGM was in fact not held until 1945. Celebrations being underway the embarrassing fact she reported was otherwise ignored and a good time was had by all.

But others may ask how an arithmetic anomaly could occur. It appears that a certain honorary secretary, having prepared the report of the 5th scientific meeting, described the coincident AGM similarly as the 5th, in her minutes. Subsequent secretaries followed suit. The 1st scientific meeting having been held within one month of inauguration in 1944, this tidy but inaccurate adjustment added an apparent year to the age of the Association.

The committee had no intention to mislead. Is it right to let the matter rest? Ought we hold a second 21st birthday party in 1966? or may we ignore the formal inauguration date, and regard 1943, when the infant association began to stir, as its true year of birth"[1]

Holding our 50th birthday in 1993, our 49th year, is obviously in keeping with the fine traditions of the OAA. We have apparently always been ahead of our time.

On a more serious note, today we should honour and pay tribute to those distinguished orthoptists who met in Sydney in 1944. As previously stated 3 of those orthoptists, Mrs Retalic, Miss Lance and Miss Balfour are attending this conference. It was with great foresight, wisdom and courage that they founded our Association. Their initiative has proved valuable to all orthoptists in Australia in many ways.

The OAA has enabled us all to maintain close ties with our colleagues both interstate and overseas. This is witnessed by the fact that at this conference we have orthoptists from

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every state in Australia as well as colleagues from the United States, England and New Zealand.

Australia was the third country after the UK and the USA to establish a national association. The OAA was one of the seven founding member nations of the International Orthoptic Association (IOA) which began in 1967. In 1983 the OAA was honoured to have an Australian orthoptist, Shayne Brown, as the IOA chairman. Today the IOA has 15 national associations as members.

From humble beginnings in England in the 1920's when Dr Ernest Maddox trained his daughter Mary to use the amblyoscope in squint training as he found it too time consuming, the orthoptic profession has emerged. Since Miss Maddox the first orthoptist, was trained by her father, orthoptic education has progressed through clinical based training to the current university degree courses that are emerging throughout the world.

Currently the education of Australian orthoptists leads the world. We were the first country to have orthoptic courses in universities and it is perhaps fitting that the first Associate Professor of Orthoptics in the world was an Australian trained orthoptist, Elaine Cornell, at the University of Sydney.

The annual scientific meeting of the OAA enables us to share our questions, problems, research and knowledge and to pass our experience and skills on to others. Research has always been a priority amongst orthoptists in Australia. Every year since 1959 the OAA have published papers. Originally Transactions of the OAA were published following the annual conference then in 1966 the first Australian Orthoptic Journal was published. Today our journal is circulated to libraries throughout the world.

Together with keeping pace with the advances in the area of research there have been many changes in clinical procedures and practice since the days of the first orthoptic clinic at the Alfred Hospital in Melbourne in the 1930's. From their initial role in the management of squint and amblyopia orthoptists in Australia have ventured into fields as diverse as early childhood development, rehabilitation, eye care for the developmentally delayed and multi handicapped, low vision and blind patient management, rural and aboriginal eye health and eye care in underdeveloped countries. Despite this we have maintained our identity with a firm grip on our expertise in the management of strabismus and visual function.

There is no question that orthoptics in Australia has been through enormous development and evolution and we have achieved very high clinical and research standards. Unfortunately our progress has not been recognised outside the eye professions as we have not succeeded in enhancing the public awareness of orthoptics.

In 1993 it is clearly critical that we promote our profession to the public. We have made a start by conducting the nation wide study into the incidence of abnormal vision in 3 year old children, the results of which will be presented at this conference. This project involved a great deal of work for many of our members, however it represents only a very small fraction of the work that will have to be done to promote orthoptics.

To illustrate my point about public awareness I looked up 1944, the year of the OAA's 'birth', in the 1987 Australian Almanac[3]. Some of the prodigy of 1944 included the adventurer, Dick Smith, a sporting great, John Newcombe, the Prime Minister, the radio programme, Blue Hills and the first AFL football teams from Tasmania including one from Sandy Bay, the suburb which we are in today. The major omission from the Almanac was the birth of the OAA.

As we enter our second half century, medicine and patient expectations will continue to evolve. As a result the orthoptic profession will have to continue to adapt clinically and enter new areas of practice just as we have so successfully to date. We will also have to maintain our strong emphasis on research and on the education of both our members and our undergraduate students.

In addition, to attract the limited purse of

public funds needed for future research and development, we also have to increase our public profile. This must involve the active participation of each and every orthoptist.

However as we take on the challenges of the 21st century we must take time to pay tribute to the 14 pioneer orthoptists whose initiative has provided the orthoptists of 1993, the current custodians of the OAA, with a vehicle in which to take on the challenges of the future.

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THE PATRICIA LANCE LECTURE

The next fifty years in Orthoptics and Ocular Motility

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Abstract

To predict what may develop fifty years ahead for ocular motility and orthoptics we must look at current areas of experimental interest: neurophysiological modelling of visual processing, developmental aspects of strabismus/amblyopia, and chemo-therapy among others. As modelling of the visual system proceeds, one may anticipate that it will soon be necessary to test concepts of normal processing in abnormal situations such as strabismus or visual deprivation. We can expect to see emphasis upon psychophysical testing of all types of visual functions in the very young child as well as attention toward visual plasticity in adults. Such testing will involve increased use of electrophysiology as well as the development of additional clinical tests. I predict that the role of the orthoptist will expand to encompass these new areas of diagnosis and documentation.

Key Words: *Strabismus, Amblyopia, Neuro-plasticity, Neuro-chemical, Orthoptist*

THE NEXT FIFTY YEARS IN ORTHOPTICS AND OCULAR MOTILITY

It is indeed an honour to be invited back to Australia, especially on the occasion of this, your 50th anniversary. To be asked to be the 4th Patricia Lance Lecturer is a very humbling experience. Not only do I have the greatest respect for Patricia Lance herself and her work, but the previous Lance Lecturers have set the highest standards.

The research and technological advances of the past decade certainly point toward remarkable potential for future development in motility and in orthoptics. I can see great strides toward opening some of the "black boxes" of ocular motility and strabismus, such as the etiology, heredity and genetics of strabismus; defining and expanding plasticity of the visual system, including further studies on suppression, amblyopia and other sensory disorders of strabismus. I also expect to see great strides in both surgical and non-surgical treatment of strabismus and amplyopia.

FROM SINGLE CELL PHYSIOLOGY TO MACRO SYSTEMS

Inner Image

If we have thought about visual processing at all, the most frequent analogy is that of a camera and film. The retina performs the function of the camera with its inverted image and

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the brain or visual cortex serves as the film or "inner screen". While such a model has heuristic value it does little to advance our knowledge nor does it help us know what object we are looking at or its shape, texture, depth, color size or spatial relationships.

Simple image or photographic representation is often deceptive, as we know from multiple examples of visual illusions such as Frazier's Spiral (Fig. 1). It is almost impossible to

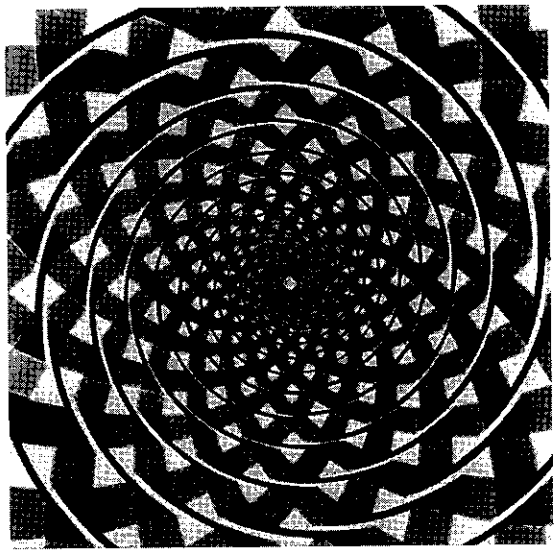

Figure 1. Frazier's Spiral is a popular illusion in which the eye is decieved into believing a true spiral exists.

convince individuals that there is no spiral in this two dimensional figure. The illusion of a spiral effect is so strong that even when subjects are asked to use their finger to follow the lines, they automatically move their finger inward in a spiral direction when no such spiral exists.

The human visual system can be fooled but it is much too sophisticated for it to happen often or easily as would happen on an "inner screen" model of vision. Nor does the inner screen model assist in explaining defects of vision such as strabismus, nystagmus or amblyopia.

SINGLE CELL PHYSIOLOGY

It has been since the epochal studies of Hubel and Wiesel in 1959[1] that the most dramatic advances have been made in our knowledge of how the brain converts an image in visual space to a perceptual concept of that image. The work of Hubel and Wiesel, Kuffler, Mountcastle and others (for a review see Schiller[2]) together with breakthroughs in electro-recording techniques and micro-electrodes have begun to break visual processing down into single-cell activities.

There are more than 160 million receptors in the human eye and the output of the whole receptor mosaic can be thought of as equivalent to a grey level description with each receptor providing one pixel.[3] Each receptor influences not just one brain neuron but hundreds of thousands. The grey level measurement provided by each receptor is used over and over again by many brain cells all working together in parallel. Color information is transmitted by the differential responses of receptors to certain wavelengths and inhibition by others. Specialized cells in the visual cortex respond to orientation, movement, or length, while others are more sensitive to particular spatial frequency patterns. Cortical cells have been found that respond to information from one or the other eye, either eye or both simultaneously. Disparity cells are responsible for stereoscopic coding with some cells responding only to nasal disparity producing the perception of distance stereopsis and other cells responding only to temporal disparity coding an awareness of "nearness" in the three dimensional array. Discoveries of behaviour patterns and cell types continue in animal models and are often supported by psychophysical studies in humans.

This is an exciting time in vision research. The challenge of the future seems to be to organise known single cell activities into systems and sub-systems of vision processing. In strabismus, the task is to relate strabismic entities and their sequelae to specific behavioural deficits and to changes in identified subsystems of the visual machinery.

MAGNO/PARVO CELLULAR

An example of this type of approach can be seen in current models of visual processing which assigns certain functions to proposed

anatomical pathways. Cells from the retina to the cortex can be divided into two functional categories serving visual perception, parvocellular and magnocellular[4,5,6,7] (Fig. 2). Cells

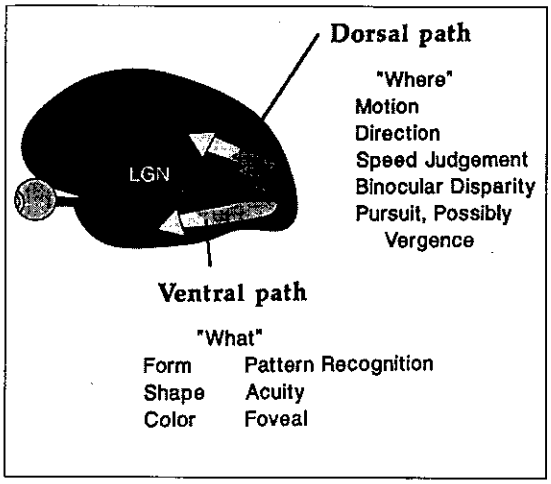

Figure 2. Model of the magnocellular ("where") and parvocellular ("what") paths of visual processing.

within the parvocellular system, the "what system", respond best to high spatial frequency patterns, with slow or no movement. The parvocellular system is largely composed of cells from the foveal area and projects through to the striate cortex then on to visual area 2 and to cortical area MT. The parvocellular system is colour sensitive and is responsible for high resolution vision. This is the system believed to be most involved in amblyopia.

The magnocellular system is composed of large cells with rapidly conducting fibres from the peripheral retina and responds best to moving stimuli and low spatial frequency patterns. This system projects through the two ventral layers of the LGN to the striate cortex and on to visual area 2. This system also has projections to the superior colliculus, that portion of the mid-brain that is so important for ocular movements, especially reflex ones. It is felt that the magnocellular system is responsible for processing motion, vergence control, stereopsis and may play a pivotal role in the etiology of infantile strabismus.[8,9,10] This system is color blind.

OKN

Incrimination of the magnocellular system in strabismus was prompted by asymmetrical optokinetic nystagmus (OKN) which is characteristic of infants to about age three months. The response to objects moving in a temporal to nasal direction is more brisk than for nasal/temporal movement. Binocular OKN's are symmetrical. In infants with infantile strabismus, the asymmetry often persists and is frequently associated with latent nystagmus and dissociated vertical divergence. Some investigators suggest that loss of cortical binocularity and binocular input to a hypothetical "movement system" is the cause of the OKN deficit. Others suggest that maldevelopment of the visual movement system may be the etiology of infantile strabismus.[8,9,10] It is interesting that the magnocellular system is believed to reach mature levels of function at about the same age that binocularity, vergence control and stereopsis are developed in the infant.

Although the Lateral Geniculate Nucleus (LGN) is the main target of ganglion cells, there are at least 9 other nuclei within the brain which receive direct retinal input[11]. All told, the number of neurons allocated to sensory visual function composes approximately 65% of the cortex. Indeed the visual system seems to be the most highly evolved of the brain structures with multiple parallel paths, the function of most of which is largely an enigma.

The role of researchers in the future will be to determine the purpose of these systems and their component parts. This will involve clinical and psychophysical investigation of visual processes not currently considered such as movement and contrast sensitivity, spatial sensitivity, color, and all in combination. Anatomical confirmation will perhaps be possible with some of the new generations of imaging equipment now becoming available such as Positron Emission Tonography, Magnetic Resonance Imaging and others yet to be developed. Patients will be studied in the context of what changes have occurred in specific subsystems to cause the types of behaviour and sensory deficits.

POSITRON EMISSION TONOGRAPHY

Positron Emission Tonography (PET) shows a great deal of promise toward allowing investigators to correlate anatomical areas with function. In a study in which 50 children with significant closed head injury were evaluated, cortical blindness was the most frequently found ocular problem. PET scanning was performed during the rehabilitation of 13 of these patients who suffered traumatic brain injuries with cortical blindness.[12] The presence of metabolic activity in the visual cortex was found to accurately predict which children would recover some form of vision at the time of discharge. PET studies have indicated the ability to detect the areas of the brain that are being used for activities such as reading, judgment of velocity and speaking. The hope is that over time, PET techniques will enable investigators to discriminate normal from abnormal behaviour in many aspects of visual function.

DEVELOPMENTAL FACTORS AND VISUAL PLASTICITY

Normal Visual Development

An area that has made remarkable advances in the past two decades is our knowledge of normal visual development. While much has yet to be learned, we are beginning to acquire some fairly firm estimates of when many visual skills are acquired and to establish a normal range of function for others. After two decades of psychophysical and electro-physiological studies, the evidence is compelling that virtually all aspects of spatial and temporal vision are deficient in normal infants until several months after birth. Various functions appear at different points in development suggesting processing by parallel systems which develop at different rates. For example, visual acuity does not reach mature levels until 2 to 3 years when tested by preferential looking techniques but more rapid gains in acuity are recorded by Visual Evoked Potential (VEP) testing. Crowded visual acuity is not mature until age 10 years. Binocularity as evidenced by vergence responses, binocular summation with VEP and aversion to rivalrous stimuli occur during the third to sixth month. Stereopsis develops between 5 and 7 months but we do not know when these functions reach their mature levels.[13]

It is apparent that the period between 3 and 7 months is critical for visual development. It is known that the infant visual system is susceptible to insult during this time and any insult can manifest itself by strabismus, amblyopia or nystagmus. Nor is the child safe after a process has reached mature levels. Sensory adaptations such as amblyopia can occur at any time during the plastic period of visual development which in some children persists until age 12 years. So the point at which a function is "mature" does not signal the end of changes in response to environmental factors.

An understanding of the factors controlling visual plasticity is essential to the future development of improved programs of treatment of strabismus and the sequelae of strabismus. It is only during the plastic period of visual development that adaptations such as abnormal retinal correspondence (ARC), suppression, and amblyopia are developed. To date our success in eradicating these anomalies has also occurred during this plastic period.

NEUROCHEMICAL ALTERATION

Future research will be devoted to finding ways in which the plastic period can be prolonged or reversed. Various types of anti-inhibitory drugs are likely approaches. In animal models GABA-receptor blockers, catecholamine depletion and norepinephrine have all shown promise in reversing certain aspects of amblyopia and in enhancing cortical plasticity.[14] In a recent study on human strabismics, the administration of Levodopa was successful in increasing contrast sensitivity and reducing the size of the suppression scotomas in amblyopic eyes.[15]

In addition to using L-dopa in amblyopic subjects, the drug has been administered to normal subjects.[16] In all cases, the Dopaminergic drug induced an improvement of contrast sensitivity in the range of medium to high spatial frequency, that is, above 2 cycles per degree.[17] Another study used Levodopa in 5

older amblyopic children who had failed to respond to conventional occlusion therapy. One hour after drug ingestion, Snellen visual acuity temporarily improved as did contrast sensitivity and pattern VER's. This improvement was noted in both dominant and amblyopic eyes. The improvement in visual function started to decrease 5 hours after drug ingestion.[18] In another study of amblyopia, Levodopa was used for one week of daily administration in a cross-over, double-masked design. Fixation scotomas decreased in size and visual acuity improved in 70% of patients treated with Levodopa. This improvement lasted for at least four weeks after the drug was discontinued. Authors in both studies reported side effects including emesis and nausea. These are early pilot studies of the use of only one drug in human subjects and as such are extremely exciting. I would like to suggest that in the future a combination of drug therapy and rehabilitation could be used to both shorten the time needed to improve amblyopia and perhaps prolong or extend the plastic period to the point that improvement in amblyopia and strabismus could be obtained in even older individuals.

ADAPTATION: A FORM OF ADULT CORTICAL PLASTICITY?

Essential to any future manipulation of plasticity, whether it be by drugs or rehabilitation, is a better understanding of the critical period and the plastic period of the human visual system. At one time, cortical plasticity was viewed as changes or adjustments in the anatomical connection patterns that make up the visual pathways. The effect of experience on visual processing was largely ignored in this definition of plasticity. However, it was found that visual deprivation decreased cortical connections from the deprived eye. Surgery to rotate the globe in fish and amphibians caused abnormal ganglion connections to develop. Removal of one eye in an animal caused abnormal projections from the other. It was determined that sensory experience could affect connection patterns. Thus, it became important to study the possible role of adaptive shaping of such patterns. The prevailing view of a rigidly genetically determined connection pattern was abandoned. Plasticity is now considered as the capability to make functionally appropriate adjustments in the anatomical connection patterns (neuro-chemical and neuronal connectivity) that compose the visual pathways. There are many lines of evidence to suggest that cortical plasticity or adaptation to the environment can cause major perceptual changes. Whether this exemplifies cortical plasticity in the classical sense remains to be seen.

AFTEREFFECTS

Visual Aftereffects are phenomena used by psychophysicists to study populations of visual cells acting together. Aftereffects, and in particular contingent aftereffects, strongly suggest that visual plasticity persists into adulthood. A typical aftereffect consists of a technique whereby an attribute is measured on a test pattern. The observer then views an adaptation stimulus for a period of time and is retested on the original test pattern and the attribute remeasured. The McCollough aftereffect is a contingent aftereffect that suggests adaptation of cortical elements can lead to long term alteration in perceptual attributes. Figure 3b is a typical test slide of the McCollough effect, consisting of achromatic gratings of a given spatial frequency. An individual is tested on this slide to make sure that the gratings at all orientations appear achromatic. Then the subject adapts to orthogonal red and green grating of the same spatial frequency as the test grating (Fig. 3a). The subject is instructed to look from one test grating to the other so that

Figure 3a. Red Vertical and Green Horizontal McCollough Adapting Pattern, Adapting Pattern is viewed for 3 minutes.

the stimulus is temporally interrupted. After a period of perhaps 3 minutes when the subject looks back at the test grating, it may now seem tinged with colours complementary to the adaptation grating (Fig. 3b). That is, if the subject

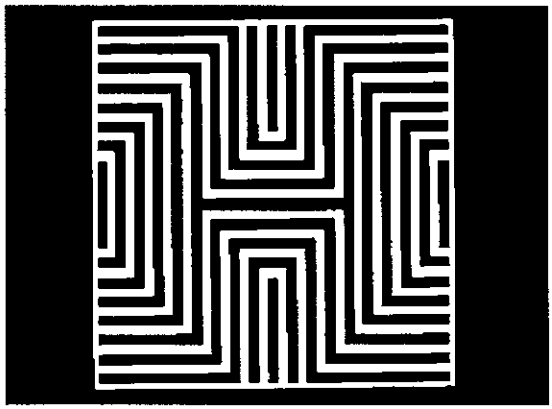

Figure 3b. When the Achromatic Pattern is viewed after adaptation, the vertical lines will appear faintly tinged with green and the horizontal lines will appear pink. The aftereffect can persist for 30 days.

viewed red vertical gratings, the achromatic vertical gratings will appear green. If the adaptation grating was of green horizontal gratings, the horizontal gratings will be faintly tinged with pink. This obviously is reminiscent of an afterimage with which Orthoptists are so familiar. However, there are many attributes of the McCollough Aftereffect which make it totally unlike an afterimage and which implicate a neural model of cortical plasticity or perceptual learning.

First, an afterimage is always associated with a particular area of the retina. In other words, if you adapt to a brightly colored circle, the afterimage will be a circle and will obey Emmert's Law in that the further away the subject looks the larger the circle seems to be. The afterimage is based on fatiguing of photoreceptors in the retina and is retinospecific and lasts for seconds. The adapting gratings for the McCollough effect were simply large rectangles, and the test grating had a totally different shape. The McCollough effect is contingent upon the spatial frequency of the lines in the test grating and is not retinospecific. There are many other differences but the most significant one is that the McCollough Effect is extremely long lasting. After 10 or 15 minutes of adaptation, a McCollough Effect can be elicited from subject 30 to 45 days later.

Obviously this can not be attributed to fatigue. It seems to be more efficient to view the McCollough Effect as a form of perceptual learning in the Hebbian sense, such that along certain synaptic pathways there is a predisposition toward facilitation of synaptic connections produced by repetition or adaptation. These synaptic pathways are believed to be the antagonistic colors and orthogonal gratings. During McCollough adaptation, synaptic paths are established and reinforced, linking color with orientation. There is some neurophysiological and psychophysical evidence that orientation specificity is not a product of excitation but of inhibition. It is unlikely to matter to the nervous system whether a signal is inhibitory or excitatory, thus the Hebbian synaptic modification can occur via inhibitory synaptic connections linking orientation with the opponent inhibitory process of the adapting color.[19] In support of this view, I found that certain neurochemical alterations such as caffeine can decrease the McCollough Effect as can sleep deprivation. In another series of studies which I performed, I found that subjects with suppression or amblyopia had exaggerated McCollough Effects in the amblyopic or suppressed eye.[20] The reason for bringing up the McCollough Effect in this discussion is not simply because it is a fascinating contingent aftereffect, but because it is an excellent example of a phenomenon which can be produced in adult subjects implicating perceptual learning or cortical plasticity.

THE TILT AFTEREFFECT

The tilt aftereffect is a second example of what may be considered cortical plasticity which can be elicited in normal adults causing a modification of perception after adaptation. Figure 4 is the testing grating which should appear perfectly vertical. Then one adapts to a tilted grating, tilted to the left for a certain period of time (Fig. 5). After looking at this grating for about 45 seconds, when one looks back at the

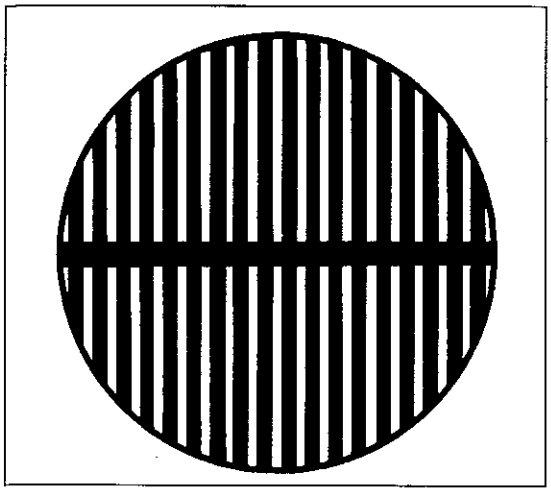

Figure 4. Tilt aftereffect test grating.

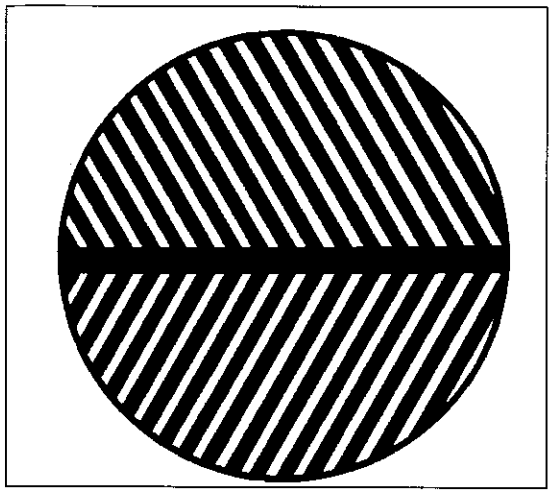

Figure 5. Tilt aftereffect Adaptation grating. After 1 minute of staring at leftward titled lines, when Fog. 4 is again viewed, the vertical lines will seem to tilt to the right before straightening.

vertical test grating, the grating for a fleeting instant will seem to be tilted to the right and then will straighten up. With prolonged viewing of a tilted grating, there is a much longer aftereffect and, in fact, the aftereffect can be appreciated weeks after the original adaptation.[21]

Gisbon, in 1937, studied subjects' ability to judge the verticality of luminescent rods in a darkened room. He adapted subjects to a tilted environment through tilted mirrors or through a peephole at a tilted room. After prolonged adaptation, when subjects were asked to judge verticality, they systematically misjudged the objective vertical for long periods of time. Additional experiments in years past have studied patients wearing inverting prisms. After wearing these prisms, which reversed right and left or up and down, for up to a week or so, subjects were able to readjust their perception to the point that things again seemed "right side up". They were able to ride bicycles, eat, read, and function normally after just a few days of adaptation. When the prisms were removed, they had to readjust. The major question is whether this is an example of cortical plasticity or whether subjects were making cognitive adaptive changes.[22] I would like to present two case histories of patients which present us with exactly that question. Both of these patients also represent an example of what I think will be some of the innovative surgical approaches of the future.[23]

CASE HISTORIES

Mr. C. is a 77 year old male with age-related maculopathy causing him to have visual acuity in his right eye of 2/200 and in the left eye of 20/50. Two days before admission, Mr. C. had a sudden decrease in visual acuity in the left eye to 2/200 caused by a massive subretinal haemorrhage. Fig. 6 shows the left eye before surgery. Mr. C. was admitted for a surgical procedure in which the macula area was moved into a healthy area of retina with normal retinal pigment epithelium. The sub-retinal area was injected with fluid creating a total retinal detachment. A 360 degree retinotomy was

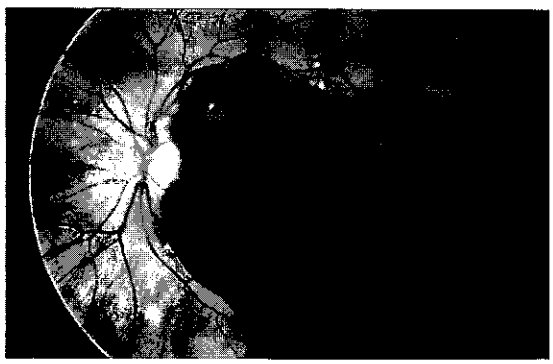

Figure 6. Pre-operative retina photograph of Patient 1.

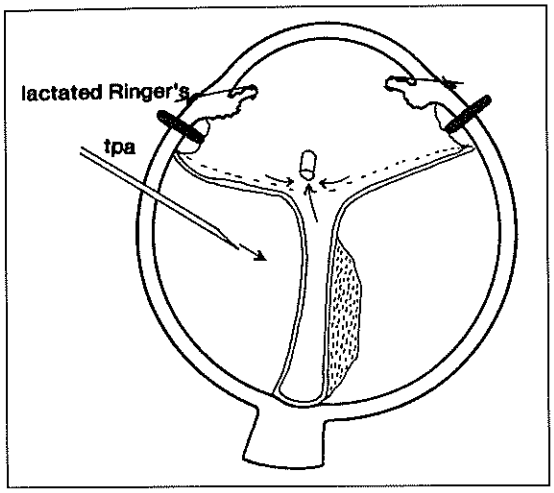

Figure 7. Macula Relocation surgery. The sub-retinal space is injected with air.

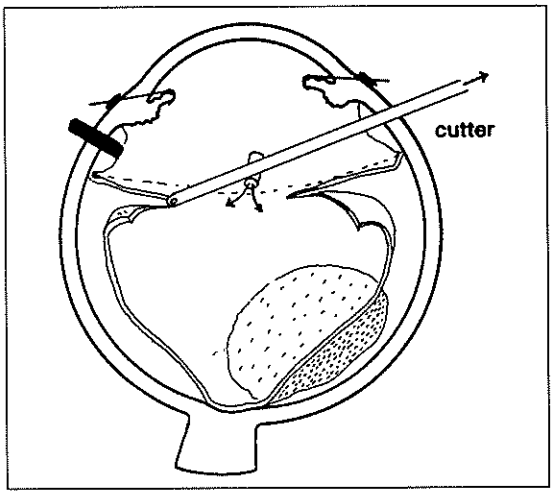

Figure 8. A 180° retinotomy performed.

performed and the entire retina rotated around the optic nerve so that the macula was moved upwards 45 degrees relative to the optic nerve (Fig. 7,8,9). The retina was reattached and silicone oil used to retain the retina immediately post-operatively so that visual acuity in the eye remained poor for several months after the procedure. Once the silicone oil was removed, the visual acuity gradually improved. Fig. 10 is a post-operative photograph of the eye. As visual acuity became acute, Mr. C became very aware of cyclo-torsion as well as past-pointing and displacement of objects of regard.

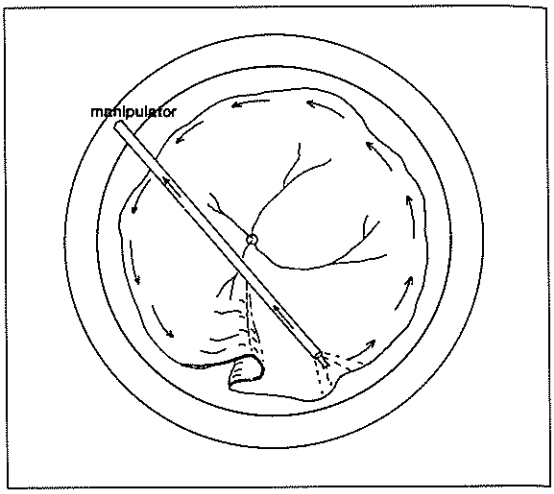

Figure 9. The macula is rotated about the optic nerve to a new area of healthier retina.

After 8 months the vision in the affected eye was 20/125 and this was by far his better eye. At near he could read 20/60 words. However, he was quite aware of subjective excyclo-torsion of the image approximately 45 degrees with a Maddox rod. He had complete inversion of his OKN response so that when the drum was

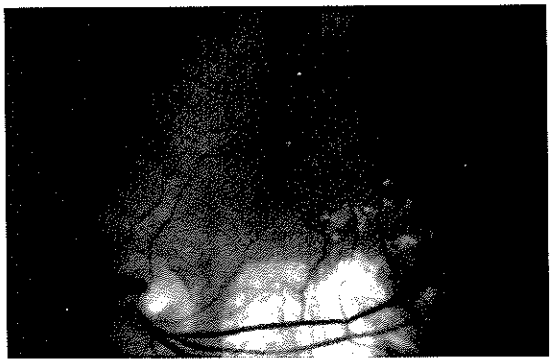

Figure 10. Post-operative photography with macula rotated upward 45°.

rotated vertically, he demonstrated a horizontal movement, and vice versa on horizontal drum rotation. Mr. C was advised to patch his right eye in order to use the eye with the macula relocation as much as possible. Over time, the torsion became variable so that a vertical image was seen to be upright and perfectly vertical. A horizontal image varied 10 to 15 degrees from horizontal. He found that he was

able to watch television and read much better but he still had a great deal of difficulty with eye-hand coordination tasks and especially walking. A planned strabismus procedure to cyclotort his eye was delayed and he was encouraged to use the eye in an attempt to see if he could further adapt and become less aware of the cyclotorsion and proprioceptive problems. At his last exam, 14 months post-operatively, his vision was 20/80 -2 at distance and 20/60 at near. On alternate cover test he measured an Exotropia of 50 and a Right hypertropia of 40. Prism placement did not alleviate his proprioceptive problems. He had no torsion on the vertical or horizontal meridian monocularly but binocularly would still fix with his unoperated right eye and was aware of a tilted "film" over his field of vision. He continued to demonstrate OKN inversion.

A second patient was a 76 year female with hand movement vision in the left eye due to AMD. The right eye had 20/200 vision until ten days before admission, when the vision suddenly decreased and the patient was unable to read secondary to a massive sub-retinal haemorrhage. Figure 11 depicts the pre-operative retinal status, and Figure 12 the post-operative status of the eye after the macula was moved downward 25 degrees into a healthy area of retina. After 4 months, the vision in the right eye was 4/200 and at near was 20/200. The patient was subjectively aware of incyclotorsion of objects of 25 degrees. She also demonstrated OKN inversion. She was encouraged to use the eye with the macula rotation. After 6 months when she returned, her vision was 15/200 in the right eye and she reported that images no longer appeared to be cyclorotated. Despite the fact that she no longer subjectively noted torsion, she still showed OKN inversion. She also had a tendency to past-point on eye-hand coordination tasks, but she felt that this was beginning to improve as well.

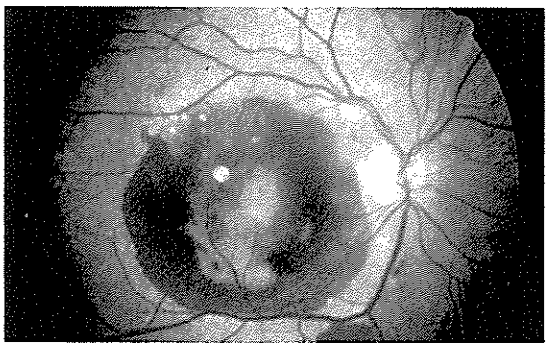

Figure 11. Pre-operative photography of macula haemorrhage in Patient 2.

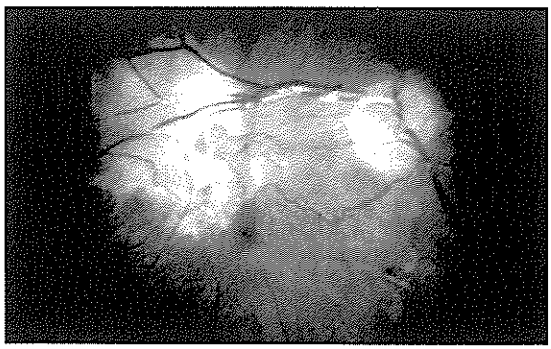

Figure 12. Post-operative photograph of Patient 2 showing macula rotated downward 25°.

In both patients, with use of the torted eye, subjective cyclo-torsion decreased. Proprioceptive anomalies persisted as demonstrated by OKN inversion and past-pointing. The big issue is whether the slow adaptation that these patients made to their altered motor state is a form of cortical plasticity. It is essential that in the future we gain greater understanding of the mechanisms especially the neuro-chemical ones, underlying plasticity including adult plasticity. This might allow for the development of techniques to stimulate these plastic responses in the visual system in both children and adults. Perhaps in conjunction with neuro-chemical therapy the binocular system could be rehabilitated and binocular cells of the cortex reactivated by appropriate visual stimulation.

REVERSAL OF THEORIES

The next fifty years will be a testing ground for many "pet theories". Some will hold up under challenge and others will be replaced. Some of the models that I have suggested in this paper will perhaps not last through the next decade. That is not important. What is important is that

we are now beginning to look past the mechanical "eye turned in, straighten it" thought process into the neuro-mechanisms which presumably could be the etiology of strabismus. Proprioception may indeed be found to play a role in ocular alignment. The accommodative convergence to accommodation ratio may not be fixed and innate but may be proven to be an adaptive and plastic mechanism like many others. If so, approaches to alter it may play a role in treatment. Indeed, neuro-plasticity may be discovered in several systems and ways found to use this adaptability to normalise visual function.

ROLE OF THE ORTHOPTIST

Where will the orthoptist be in the future? I foresee a need for an academically based visual specialist who has a broad understanding of the mechanism of vision, visual development and furthermore who has the clinical skills to apply this knowledge in the evaluation and treatment of patients. I predict that treatment will play a larger role in the future of ocular motility. Such treatment will not only include surgical techniques but advanced non-surgical approaches will be developed and neurochemical approaches may facilitate both. Foremost among these will be treatment to enhance binocularity and effective amblyopia treatment. The orthoptist has demonstrated resourcefulness and adaptability in the past 50 years. That trait will continue to serve the profession. The orthoptist will be the person who administers treatment and evaluates success. Likewise, the orthoptist will be the person who initiates early screening not for amblyopia, but for strabismus within the early, developmentally critical months of a child's life.

To fulfil such a role in a technologically complicated setting could reasonably require a stronger academic base perhaps even in a post-graduate setting. This would only follow trends present in other health care professions and in other countries. Australia has taken the lead in the world in this respect and is to be congratulated. It makes sense to have a clinically based post-graduate program but with the potential for some persons to continue into a more research oriented career involving perhaps a Ph.D. Knowledge in the area of ocular motility is growing at an astounding rate. The orthoptic profession can play a contributing role in the continued expansion of this field as a competent and educated clinical investigator and observer. I challenge you to approach the future with the same enthusiasm and excitement that I have always associated with Patricia Lance. May the next fifty years be the best to come!

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SCIENTIFIC PAPERS

THE INCIDENCE OF REDUCED VISUAL ACUITY AND SQUINT IN PRE SCHOOL CHILDREN AGED THREE IN AUSTRALIA.

Orthoptic Association of Australia Study.

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Abstract

Three thousand and twenty 3 year old children attending pre schools throughout Australia were screened by orthoptists. Testing included visual acuity (VA), using Sheridan Gardiner single letters at 6 meters and cover test. The incidence of reduced VA (6/9 or worse testing with SG singles) when testing in a classroom situation was 14.7%. A difference of more than one line in VA between the two eyes occurred in 14.9% of the children. (6.5% of the children could not do the SG singles VA test at 6 meters). The incidence of strabismus was 2.4%.

Children who were found to have reduced VA together with those with strabismus may be considered to be 'at risk' of having or developing amblyopia. Such risk can only be ruled out by having the child referred for a full ocular examination in a clinical setting. Without regulated pre school assessment many 'at risk' three year old children may not be identified until such time as the child is routinely screened at school. By then valuable time for the treatment has been lost.

Key Words: *Pre school vision screening, visual acuity, amblyopia, strabismus.*

INTRODUCTION

Reduced visual acuity detected during pre school and school screening has been reported to have been secondary to amblyopia and/or strabismus in numerous cases in studies in the literature[1-9].

Amblyopia, the reduction in visual acuity (VA) in one or both eyes in the presence of organically normal eyes, is known to be caused by deprivation during post natal visual system development. The effects of deprivation are most marked in the sensitive period between the ages of three months and three years, then to a decreasing extent, up to the age of eight years[10-13]. The sensitive period is the time when amblyopia is most amenable to treatment and, the earlier the commencement of treatment the better the visual outcome[10-13].

Amblyopia is the commonest cause of visual disability in childhood[14]. In a citation by De Becker et at,[15] it was shown that at pre school age, amblyopia was found to affect between 1.2% to 5.6% of children. Other studies reported higher incidence[4,5]. The incidence of strabismus ranged from 1.4% to 6% in pre school children[1,3,4,6-8] and the combined prevalence of strabismus and amblyopia was reported to be around 5%[9].

Although amblyopia has been recognised for well over two centuries[16,17] it was not until 1925

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that the idea of vision screening appeared in the literature[18]. From 1925 to World War II, vision screening was primarily concerned with the detection of refractive errors, eye safety and incipient blindness[19].

According to Simons and Reinecke[19], amblyopia screening per se did not receive serious attention from the ophthalmic profession in the USA until the mid 1950's. By 1965 an article in the Readers Digest[20] entitled "A Hidden Threat to Children's Eyes" estimated that only 0.2% of three to six year old children in the USA were being screened.

Today vision screening is common in developed countries. Debate on screening in the literature includes; what is the ideal age at which to screen, what methods should be employed to screen VA in children, what is the place of refraction in pre school screening, what constitute the criteria for referral to eye professionals and who should do the screening.

A number of publications advocated screening before school age[4,21,22]. For example, a paper in the Lancet[4] reported on screening of 1506 pre school children. They concluded that there were 50% fewer visual problems overall in children who had undergone pre school screening[4]. On the other hand, some literature stated abnormalities who were missed during pre school screening programmes[5,23]. In these studies most of the children with serious visual disruption had presented for examination without any screening. Shaw et al[23] stated that, following pre school screening, there had been no significant improvement in the age at which amblyopia had been detected over the previous 13 years. They attributed their finding to the fact that there was no existing test that was adequate for pre school screening.

Responses to the question of which methods should have been employed to screen VA in children, ranged from chart based tests, single letters and preferential looking to contrast sensitivity tests and visually evoked responses[21].

The place of refraction in pre school screening had also been discussed extensively in the literature. Some literature suggested that refraction during the first year of life gave the best predictive value of those children likely to develop amblyopia. Those with +3.50D or more in any one meridian often did not attain and retain normal VA even with occlusion[5,23]. On the other hand some papers argued that, as refraction was not a direct test for either amblyopia or strabismus, and as many children have only minimal hypermetropia there may be many under referrals if clinicians relied on refraction results[21].

The question of what constituted the level of VA for referral was another debate that was not resolved. The American Academy of Paediatrics referral criteria was 6/12 or worse in one or both eyes below 5 years old and 6/9 or worse for older children[24]. In many other publications, predominantly those from Britain, the referral criteria was VA less than or equal to 6/9 in one or both eyes[7,15,22,25]. There appeared to be consensus that children with more than one line difference in VA should be referred[7,15,22,24,25].

The other consensus in the literature on pre school screening was the finding that orthoptists were more likely to detect visual anomalies during pre school screening and that the level of unnecessary referrals was much lower when orthoptists perform screening as compared to general practitioners and community nurses[7,22,25].

In the 1989 Australian Census[26] there were 277,000 pre school 3 year old children in this country, however there were no figures available on their visual status. The aim of this study therefore, was to determine the incidence of reduced VA and strabismus in pre school children in Australia. These anomalies may otherwise remain undetected until school age or later.

Referral of children with reduced VA and/or strabismus may result in earlier detection of and treatment for any ocular anomaly including amblyopia. This in turn may reduce the incidence of unnecessary visual loss.

METHODS:

The Orthoptic Association of Australia (OAA) called for volunteer orthoptists throughout Australia to test pre school children. The majority of the children in the study were attending

Long Day Care Centres, administered by the Commonwealth Department of Health, Housing and Community Services. It was considered that these centres should provide a broad socio economic cross section of the Australian three year old population as children from a broad cross section of backgrounds attend these government funded centres.

The orthoptists who participated in the study assessed a total of 3020 children aged 3. For each child the date and location (via postcode) of screening was recorded together with date of birth, gender and whether or not the child wore glasses. All recordings and test results were made on the OAA data form provided.

Prior to screening, written instruction on methods to be used to test VA and cover test (CT) were distributed to the orthoptists. The children were chosen at random by the orthoptists from their class rooms. Best corrected monocular VA was assessed at 6 meters using Sheridan Gardiner (SG) single letter books and matching cards. Acuity was assessed down to 6/3 where applicable and was recorded using the standard 6 meter notation (ie 6/4, 6/5, etc). If a child was unable to perform the test at 6 meters, 'can't do' was recorded.

The criteria selected for analysis of the results of the VA test was based on the criteria used in similar studies conducted by orthoptists in Britain[7,15,22,25]. These criteria were 'pass' (those children with VA 6/9 or better) and 'fail' (those children with VA 6/9 or worse). All VA results from the OAA study were divided into the criterion pass, fail or can't do. VA of 6/9 or worse was used to denote children who would have required further investigation.

Cover test was performed at 6 meters (CT 6m) and 1/3 meter (CT 1/3m) using an accommodative target. Any constant or intermittent strabismus was recorded. Children with these anomalies would also have required further investigation. A space was left on the data form for any comments that orthoptists thought relevant.

Statistical Methods:

For the purpose of analysis it was necessary to convert Snellen's results into simple numbers as there were drawbacks to the Snellen scale which made it difficult to easily convert to a number. The method used was recommended by Berry[27] whereby each line of the chart was allocated a number with 6/4 or better being allocated number 1, $6/4^-$ or $6/5^+$ allocated number 2, 6/5 allocated number 3 and so on. The interval between adjacent numbers on this scale represented roughly equal changes in VA.

The mean VA and standard deviation were estimated for the population sampled. Analysis of variance (ANOVA) and Chi-square tests were performed to determine the effect of independent variables (such as age and gender) on VA and/or the presence of a strabismus and, to test for any variability between the right and left eyes.

Plots of the shape, skewness and kurtosis of the sample measures suggested that most of the data were suitable for parametric analysis. There was some uncertainty whether or not the data from the sub-sample who wore glasses were suitable for parametric analysis because their data showed serious departures from normality in skewness and kurtosis. A conservative decision was made to use non parametric tests on the data. In cases where repeated measures analysis was needed it was decided to use the Friedman repeated measure non parametric ANOVA. The decision was made because bivariate scatterplots of the data showed a tendency towards heteroscedasticity. The analysis programme employed, SPSS, performed repeated measures ANOVA with its multivariate analysis of variance (MANOVA) command. Tabachnick and Fidell[28] recommend against using MANOVA unless the data are homoscedastic. Repair of the data, even if possible, would have required extensive transformations which were not deemed to be warranted in light of the likely returns.

The Bonferroni adjustment was used to produce a family-wise error rate of $p<0.05$. This means that in most cases, where a series of tests were performed, the individual error rate was $p<0.01$.

RESULTS

i. Population Sampled

Three thousand and twenty children aged 3 were assessed, thus there were 6040 eyes in the study. (See table 1 for the demographic breakdown). The age of the children ranged from 3:0 (years : months) to 3:12 with the mean age being 3:6.5. (3:12 included only children who were older than 3:11 but had NOT turned 4 years old at the time of testing). The children were put into 12 separate age groups for the purpose of analysis (see table 2).

Table 1
Demographic Breakdown

State	Total No. of Children Assessed	Percentage Assessed Per Orthoptist	No. of Children Assessed Per Orthoptist
ACT	177	5.9%	44
NSW	1400	46.4%	11
QLD	268	8.9%	16
SA	222	7.4%	27
VIC	926	30.7%	11
WA	27	0.9%	2
TOTAL	3020	100.0%	av = 18.5

Table 2
Age Groups

AGE - Y:M	Number	Percentage
3:0 - 3:1	308	10.2%
3:1 - 3:2	196	6.5%
3:2 - 3:3	206	6.8%
3:3 - 3:4	235	7.8%
3:4 - 3:5	265	8.8%
3:5 - 3:6	259	8.6%
3:6 - 3:7	243	8.0%
3:7 - 3:8	265	8.8%
3:8 - 3:9	294	9.7%
3:9 - 3:10	242	8.0%
3:10 - 3:11	265	8.8%
3:11 - 3:12*	242	8.0%

** Note: 3:12 includes only children who had NOT turned 4 years old at the time of testing.*

In the population tested, 51.4% were males (see table 3). There was no significant difference in the numbers of males or females in any of the 12 age groups (x^2=4.28, df=10, p=0.93). ANOVA indicated that there was no main effect of gender on RVA ($F_{1,2998}$=0.451, p=0.50) nor on LVA ($F_{1,2998}$=0.197, p=0.66).

Table 3
Gender

Gender	No of Children	Percentage
Male	1552	51.4%
Female	1468	48.6%

A total of 29 (or 0.96%) children wore glasses (see table 4).

Table 4
Number of Children with Glasses

Glasses	No. of Children	Percentage
YES	29	0.96%
NO	2991	99.04%

ii. Visual Acuity

Assessment of VA was performed monocularly and the results are summarised in table 5. It is of interest that 21 children, 38 eyes achieved VA of 6/3). There was no significant difference in the VA of the right eye (RVA) when compared to the VA in the left eye(LVA) in any age group (Friedman repeated measures non parametric ANOVA; x^2=0.6707, df=1, p-0.42). For the purpose of analysis, VA results were put into 1 of 4 categories (see table 6) 'Poor vision': 6/9 or worse was selected to denote the level of VA that would be used as a criterion for referral for further investigation. The number of children in each VA category is outlined in table 7.

Table 5
Visual Acuity; RVA and LVA

Vision	Right Eye		Left Eye	
	No.	%	No.	%
6/4 or better	146	4.8	160	5.3
6/4- or 6/5+	114	3.8	130	4.3
6/5	267	8.8	265	8.8
6/5- or 6/6+	329	10.9	329	10.9
6/6	1079	35.8	1044	34.6
6/6- or 6/9+	439	14.5	460	15.2
6/9	248	8.2	222	7.4
6/9- or 6/12+	81	2.7	86	2.8
6/12	79	2.6	68	2.3
6/12- or 6/18+	16	0.5	15	0.5
6/18	20	0.7	25	0.8
6/18- or 6/24+	4	0.1	3	0.1
6/24	1	0.0	4	0.1
6/24- or 6/36+	0	0.0	1	0.0
6/36 or worse	8	0.3	7	0.2
Can't Do	189	6.3	201	6.7

The mean VA was 6/6 to $6/6^-$ (or $6/9^+$; ie

Table 6
Categories of Visual Acuity

Category	Visual Acuity
Pass	6/6- or better
Fail	6/9 or worse
Can't do	not possible to test at 6 metres

Table 7
Visual Acuity in each Category

VA Category	Number of Children RE	LE	Overall &
Pass	2374	2388	78.8%
Fail	457	431	14.7%
Can't do	189	201	6.5%
	3020	3020	100.0%

'pass' category) in either eye. However the standard deviation was three lines of vision in either direction. As a result, the overall distribution was slightly skewed towards poor vision.

A Kruskal-Wallis 1 way non parametric ANOVA suggested that VA tended to improve with age (RE: x^2=106.4623 corrected for ties, $p<0.0001$; LE: x^2=112,5338 corrected for ties, $p<0.0001$). ANOVA indicated that both RVA ($F_{10,3009}$=8:94, $p<0.0001$) and LVA ($F_{10,3009}$=10.01, $p<0.0001$) were affected by the age category. Using age category and VA in non parametric correlations there was a tendency for VA to be better with increased age (RVA r=-0.18 and LVA r=-0.19).

Table 8
Children who 'can't' do Vision

Can't do VA	No. of Children	No. of Eyes	%of Eyes
Both eyes	188	376	6.23%
Can't do RVA	1	1	0.02%
Can't do LVA	13	13	0.22%
Total	202	390	6.47%

Of the 3020 children 390 were in the category of 'can't do' VA at 6 meters ('can't do' either eye = 188; RVA = 1; LVA = 13. The small numbers in some cells precluded statistical analysis; see table 8). These numbers represented 6.5% of the population tested. Cross tabulation of age group with ability to do VA revealed that if VA could not be assessed the child was most likely to be a young 3 year old (x^2=41.12055, df-10, $p<0.001$).

Criteria for referral:

a)Fail VA: In the whole population of 6040 eyes there were 14.7%, (771 eyes in 444 children) with VA 6/9 or worse hence were categorised as fail VA. There were a total of 10.8%, (327 children in 654 eyes) with 6/9 or worse in both eyes. Another 3.9%, (117 children in 117 eyes) had 6/9 or worse in only on eye (see table 9).

Table 9
Children with 'poor' vision

Poor VA	No. of Children	No. of Eyes	%of Children
Both eyes	327	654	10.8%
One eye	117	117	3.9%
Total	444	771	14.7%

Of the total of 444 children with 6/9 or worse in one or both eyes only 12 children (2.7%) wore glasses at the time of testing. (Seven had 6/9 or worse in both eyes and five had 6/9 or worse in one eye).

(The American Academy of Paediatrics standard for referral in visual screening is 6/12 or worse in either eye in children under the age of 5 years. Only 4.1% of the children in the OAA study would have met the criteria for referral VA screening in the USA; see table 10).

Table 10
USA screening standards

Visual Acuity	Number of Children RE	LE	% of Children
6/9+ or better	2703	2696	89.4%
6/12 or worse	128	123	4.1%
Can't do	189	201	6.5%
Total	3020	3020	100.0%

Table 11a
More than 1 line difference in VA;
All Children

Visual Acuity	No. of Children	% of Study
Poorer RVA	209	6.9%
Poorer LVA	240	8.0%

(Poorer RVA = RVA more than 1 line worse than LVA.
Poorer LVA = RVA more than 1 line worse than LVA).

b)Difference of more than one line: In this study there were 449 children (14.9%) who had a difference in VA of more than one line between the two eyes (see table 11a). If there

Table 11b
Difference between RVA and LVA;
Children without glasses versus children with glasses

Visual Acuity	Number of Children Without Glasses	With Glasses	Total
Equal	1722	9	1735
1 line diff	630	8	638
> 1 line diff	439	10	449

was no greater chance of poorer VA occurring in the right eye (poorer RVA) than the left eye (poorer LVA) the expected frequency for poorer RVA and poorer LVA would be 224.5. Analysis revealed poorer RVA in 209 cases and poorer LVA in 240 cases. Statistical analysis revealed that VA was no more likely to be poorer RVA than the poorer LVA (x^2=2.4, df-1, p<0.05).

Only 10 of the 449 children with more than 1 line difference in VA between the two eyes wore glasses at the time of testing (see table 11b). Statistical analysis revealed that children wearing glasses were more likely to have more than one line difference between the two eyes than children who did not wear glasses (x^2=11.71091, df=2, p=0.003). A difference of more than one line in VA between the two eyes was also statistically more likely to occur in children who had squints (x^2=44.15842, df=4, p<0.0001).

Statistical analysis revealed that age (x^2=15.60211, df=20, p=0.74) and gender (x^2=0.78033, df=2, p=0.68) had no effect on this outcome.

As mentioned previously 29 (or 0.96%) of the population sampled wore glasses (13 males and 16 females). These children were statistically analysed separately. A Kruskal-Wallis 1 way non parametric ANOVA suggested there was no effect of gender on VA in this sub group; (RE; x^2=1.97 corrected for ties, p=0.16, LE x^2=1.89 corrected for ties, p=0.17). There was no significant difference between RVA and LVA in the children who wore glasses (Friedman repeated measures non parametric ANOVA; x^2=1.2414, df=1, p=0.27).

When looking at the breakdown of VA into the 3 categories it is apparent that there were

Table 12
Children who wear glasses

Visual Acuity Category	Number of Children RE	LE	Overall %
Pass	17	18	60.4%
Fail	10	9	32.7%
Can't do	2	2	6.9%
	29	29	100.0%

fewer children wearing glasses in the 'pass' VA category (see percentages in table 12 compared to table 7). Despite this observation statistical analysis revealed that only RVA was significantly worse in children who wear glasses (Kruskal-Wallis 1-way ANOVA, x^2=5.638 corrected for ties, p=0.02). The same statistical analysis of LVA in children who wore glasses compared to those who do not revealed no significant difference (Kruskal-Wallis; x^2=3.25 corrected for ties, p=0.07).

iii. Strabismus

A total of 76 children (2.52%) had strabismus.

Table 13
Children with squint
Type of Squint* (n=76)

ESO 1/3m	ESO 6m	No.	EXO 1/3m	EXO 6m	No.
Et	ET	17	XT	XT	3
IET	IET	5	IXT	IXT	6
IET	NAD	10	IXT	NAD	26
			NAD	IXT	9

**ET = constant esotropia, XT = constant exotropia, IET = intermittent esotropia, IXT = intermittent exotropia, NAD = no tropia*

Of the 76 children there were 20 with constant strabismus and 56 with intermittent strabismus (see table 13).

The VA of the children with strabismus was fairly evenly divided between the categories

Table 14
VA of children with squint

VA	No. of Children	No. with Glasses
Pass	37	4
1 poor eye	15	3
2 poor eyes	21	2
Can't do	3	0
Total	76	9

'pass' (n=37) and 'fail' (n=36) with 3 children with strabismus unable to do VA at 6 meters,

(see table 14). This finding is in keeping with the larger proportion of intermittent strabismus. Statistical analysis revealed that VA tended to be worse in children with strabismus compared to children who did not have strabismus (Kruskal-Wallis 1-way ANOVA; CT 1/3m, RVA; x^2=33.0146 corrected for ties, p<0.0001: LVA x^2=19.3007 corrected for ties, p<0.0001; CT 6m, RVA; $x^2$28,3836 corrected for ties, p<0.0001: LVA; x^2=22.6118 corrected for ties, p<0.0001).

Nine of the children with strabismus wore glasses at the time of screening. Numbers of children wearing glasses were too small to do a meaningful statistical analysis.

ANOVA revealed that neither age nor gender had an effect on the chance of finding strabismus (constant or intermittent) when testing at near. A Bonferroni adjustment was used to ensure a family-wise error rate of p<0.05. With the significance level for individual comparisons set at 0.0125 there were no significant main effects of gender ($F_{1,2998}$=0.046, p=0.83) or age ($F_{10,2998}$, p=0.02) and there were no intersections ($F_{10,2998}$=0.687, p=0.74). (Although the main effect for age was not significant it was close to significance. With a very large sample size small sized effects can approach or achieve significance but will not be of clinical use. The fact that this result did not achieve significance means that it is probably a very small effect in the population and therefore not of clinical significance to orthoptists). Similarly for distance cover test there were no main effects of gender ($F_{1,2998}$=0.014, p=0.90) or age ($F_{10,2998}$,p=0.61) and there was no interaction of gender with age on strabismus.

DISCUSSION

Children who were found to have reduced VA as indicated by their failure to meet the'pass' criteria for VA when tested in a classroom situation, together with those who were found to have strabismus, may be considered to be 'at risk' of having or developing amblyopia. Such risk can only be ruled out by having the child referred for a full ocular examination in a clinical setting, hence the 'at risk' children's parents must be made aware that such an examination is necessary. Without some routine method of pre school screening in Australia, these 'at risk' three year olds may not be identified.

The major findings in this study were the incidence of reduced VA detected during screening; (14.7%), the number of children with more than one line difference in VA between the two eyes; (14.9%) and the incidence of strabismus detected during screening; (2.5%). The other finding of interest was that 6.5% of the children could not do the SG singles VA test at 6 meters.

The incidence of reduced VA in the OAA study was very high when compared to a study by Beardsell[22] in which 2475 three and a half year old children were assessed by orthoptists using SG singles at 6 meters and cover test and the same referral criteria. Beardsell's incidence of reduced VA (and thus referral) was 4.12%. (Their assessment included stereopsis, ocular movements and 20 dioptre prism test). On the other hand, Feldman et al[4] conducted a study using the illiterate E test and the same referral criteria as the OAA, and their incidence of reduced VA was 12.5%.

In another study, where orthoptists tested SG linear VA at 6 meters and VA 6/12 or worse and/or squint was used as referral criteria, Ingram et al[5] reported a referral rate of 8%. This represented a higher incidence of reduced VA and/or strabismus than the OAA study. Using 6/12 or worse as the referral criterion only 4.1% of the OAA study subjects would have been referred for reduced VA and 2.5% for strabismus. This finding may be partially explained by the use of single letter optotypes to test VA in the OAA study. Ingram used linear optotypes. Single letter optotypes are generally considered to over estimate visual acuity[23]. Ingram et al found that 5.3% of their study could not do the SG linear chart at 6 meters in a screening situation. This finding was slightly lower than the finding of 6.5% in the OAA study.

Results demonstrated that, of the children who could not do VA in one eye only, there

was one child who could not do RVA while 13 children could not do LVA. From this finding it is tempting to assume that RVA may have been tested first in many cases and thus to speculate that the larger number of 'can't do' LVA suggests that the children became bored or lost concentration following RVA testing.

The finding in the OAA study of 2.52% of children with strabismus was lower than a study by Bruce et al[8]. In Bruce's study orthoptists found 4.2% of the 2:6 to 3 year old children tested had strabismus. In another study by Edwards[7] et al, orthoptists found an incidence of 1.4% of 3:6 to 4 year old children with strabismus.

There were numerous studies in the literature which reported on the incidence of reduced vision and/or squint in children following screening[1-3,6,9,15,21,25,29] however, it is difficult to compare the OAA study to these as the referral criteria and/or the ages of the children differed too greatly from those in the OAA study.

CONCLUSIONS:

We know that there are over 277,000[26] pre school 3 year old children in this country. The results of the OAA study have demonstrated that there is a high level of reduced VA when testing vision with single letters in a classroom situation. The children found to have reduced VA and/or strabismus in such situations should be referred for clinical examination. Without regulated pre school assessment many 'at risk' three year old children may remain undetected until school age or later.

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

In a commentary on the incidence of amblyopia and its detection rate Sir Stewart Duke Elder[30] quoted Franceschetti; and stated

That this enormous number of people suffer from a serious visual defect is a grave economic and social matter, and that it should be tolerated with complacency is a depressing reflection of the neglect shown by modern civilisation towards its human material.

It is up to the OAA to ensure that our findings are brought to the attention of the public so that 'at risk' children can be fully examined and amblyopia in this country is no longer tolerated with complacency.

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VIZASSESS A COMPUTER GENERATED TEST OF VISUAL FUNCTION

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Abstract

Assessment of the visual function of children with severe intellectual disability or gross developmental delay is a challenging and difficult task. Conventional tests of visual function are inappropriate and generally unsuccessful. Clinicians are often forced to rely on assessments which are of limited interest to the child and do not provide a quantitative result. Vizassess is a software package developed to determine the presence of vision and where possible a measure based on the standard Snellen fraction. The test presents a range of familiar images constructed in Snellen equivalent sizes. Movement and image manipulation are used to attract attention and facilitate non verbal responses. Clinical trials indicate that the test is reliable and able to be used with some of the children incapable of response to conventional tests of vision.
Key Words: *Computer Software, visual function, visual assessment, non verbal communication.*

INTRODUCTION

What do I see and how well do I see it? For the majority of people this question can easily be answered by administering appropriate tests of visual function. Clinical experience suggests that children with developmental delay, severe intellectual handicap or a severe communication problem are not so easily assessed. However a number of studies have reported a high incidence of ocular dysfunction in this population[1,2]. These studies report the incidence of sight threatening ocular pathology excluding refractive error and strabismus as 30 to 40%.

The importance of gaining an insight into the level of visual function is apparent given the significance of vision as a sense through which information about our environment is gained. Commonly used resources for learning such as; books, videos and computers are based on visual input. When vision is impaired a range of devices are used to ameliorate the effects of vision loss on learning and daily living[3,4]. Such devices are only provided following careful assessment of the extent of vision loss.

Clinically, the assessment of visual function in children with developmental delay, severe intellectual handicap or severe communication problems is difficult[5,6]. Some of these difficulties relate to features of the clinical tests in use. Conventional tests of visual acuity rely on the patient providing a definitive response to the test applied by the clinician. This response can

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take one of two forms:

1. A verbal response identifying a letter or shape.
2. A response where the patient matches the test optotype to a choice card which contains a number of possible selections.

Both responses require a degree of cognitive function which is beyond the capabilities of a severely intellectually handicapped subject[6]. Other researchers note that lack of verbal, manual and locomotor responses contribute to the difficulty of assessment[7]. Research involving children with cerebral palsy and mental retardation has shown that the children with severe motor disorders varied more from test to test and produced lower mean acuity levels. Children who were severely retarded showed greater day to day variability[8].

These difficulties have been acknowledged and have resulted in the development of tests of vision which do not require a formal response from the subject. A preferential looking paradigm incorporating the presentation of picture cards and the use of visual pointing as an indicator of vision has been reported[7,9]. Limitations are reported in using preferential looking technique which include the short attention span of the children and the boring nature of the grating optotypes[9]. One solution to this problem has been the incorporation of vanishing optotypes[10].

One researcher reports an intensive training program aimed at teaching an intellectually handicapped child to match shapes as being successful[11]. It should be noted that this solution is time consuming and only appropriate for children who have the cognisance to learn and the motor skills to match.

An alternative form of testing is to employ tests of reflex responses which do not require any level of cognition such as, the pupil response to light. Such tests do not provide any graded measure of visual function and do not contribute to our knowledge of the child's ability to use vision as a means of obtaining information.

Accurate knowledge of the visual function of children with developmental delay, severe intellectual handicap or a severe communication problem is intrinsic to understanding the child's development and ability to obtain information. This data is also valuable when determining the presentation of new information and developing training programs for that child. To facilitate the collection of data on the visual function of the target group a preliminary test program has been developed. This test is computer-based using movement to attract the child's attention and allowing the child to respond using visual pointing. A computer-based test was chosen as computers are used routinely in special development programs and to date have provided a stimulus of interest to the target group (personal communication, KF, teachers and therapists Glenallen school). The computer can store and provide access to a large variety of optotypes and allow the size, shape and presentation to be easily varied. A description of the pilot program and the results of clinical testing are presented.

METHOD

Subjects:

Thirty seven multihandicapped children attending the Glenallen school in Victoria were tested. The subjects ranged in age from 6 to 13 years with 16 being female and 21 male. The general pathology is shown in table 1, cerebral

Table 1
General Pathology

Subject Pathology	
Diagnosis	%
Cerebral Palsy	61
Rett's Syndrome	8
Developmental Delay	5
Spina bifida	5
Alexander's Syndrome	3
Peripheral neuropathy	3
Down's Syndrome	3
Myotonic Dystrophy	3
Kidney failure	3
Microcephally	3
Joubert's Syndrome	3

palsy being the most common. Permission was obtained from the Regional Education Authority, the school and informed consent was obtained from the parents. All children who returned an informed consent agreement were included in the study.

Procedure:
This trial compared the subject's performance on the computer-based assessment to that using the Kay's picture test. The tests were presented in alternate order. A positive response to the Kay picture test was considered to be an accurate definition of the picture displayed or an accurate match using a choice of picture cards. This test was administered at 3 meters. A positive response to the computer test was considered to be a positive identification of a picture, an accurate matching of a picture or the ability to change fixation to accurately locate a picture or accurately follow a moving picture. The computer test was viewed at 0.5 meters to give a minimum 6/12 acuity or 1 metre to provide a 6/6 equivalent measure.

Vizassess:
The computer test consists of 3 modules:
Module 1. A test which presents a moving target. The target can be varied in form and size. The speed of movement can also be varied.
Module 2. A test which presents a target which will increase in size. The target can be varied in form.
Module 3. A test which presents one or two targets at specified locations on the screen. The targets can be varied in form, size and location.

The shapes generated are of specified size both in terms of the overall shape and the width of the lines used to compose the shapes. These sizes are consistent with those used to generate conventional tests of visual acuity and relate to the area of retina to be stimulated. The smallest shape is equivalent to 6/12 at the viewing distance of 0.5 meters and 6/6 at a viewing distance of 1 metre. The range of size being 6/60, 6/48, 6/36, 6/24 and 6/12 at 0.5 meters and 6/30, 6/24, 6/18, 6/12 and 6/6 at 1 metre. The shapes chosen for this pilot test were: a house, cat bottle, cup, man and hand. These shapes were chosen to reflect commonly seen objects and they are consistent with shapes used in the Kay's picture test and other shape based tests for reliability of comparison. The subjects were presented with all three modules and a positive response on one or more modules considered an indication of vision. Each module is intended to stand alone as a test of acuity, the variety in modules allowing for a change in stimulus and to allow for the range of response capabilities between subjects. Vizassess was presented to all subjects at the 0.5 metre distance, those subjects who demonstrated a 6/12 equivalent at this distance were retested at 1 metre to provide assessment at 6/6 equivalent size.

RESULTS
Of the 37 subjects tested 38.7% were able to respond to both the Kay's picture test and Vizassess, a further 30.61% were assessable using the computer generated method. 30.61% were unable to be assessed using either test, figure 1. The percentage of subjects responding to each module is summarized in table 2.

Figure 1
Subject responses to VA Tests

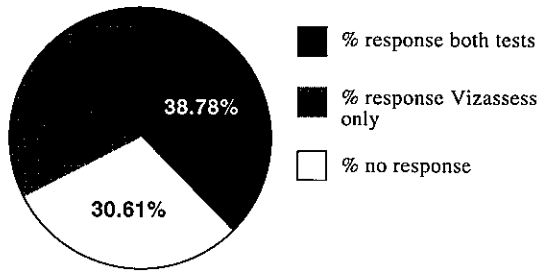

Table 2
Percentage of subject responses to Vizassess modules

Module 1	Module 2	Module 3	Modules 1,2 &3
82%	67%	82%	67%

N = 27

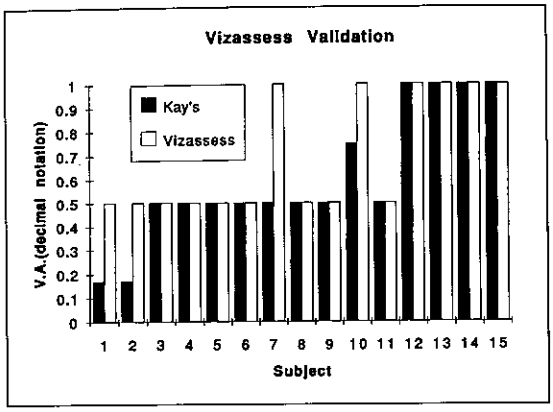

Figure 2.

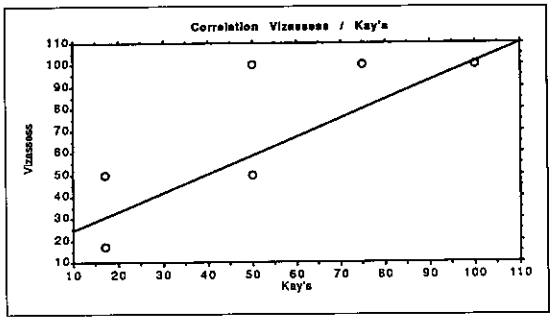

Figure 3.

Figure 2 indicates the results obtained by those subjects able to respond to both Kay's pictures and Vizassess. The level of visual acuity in the majority of cases is the same using either test. The correlation of these results is shown in Figure 3, which indicates a strong positive correlation (r=.847) and this relationship was significant at the 0.0002 level of confidence.

DISCUSSION

The clinical trial sought to answer two questions:

1. Are the vizassess optotypes a reliable measure of visual acuity?
2. Will the Vizassess program provide information about the visual function of severely impaired subjects who can not be assessed by conventional tests of vision?

The strong positive correlation between visual acuity results obtained on each test used during clinical trialing suggests that the optotypes presented in the Vizassess computer program are a reliable assessment of visual acuity when compared with the standard Kay's picture test. Four of the subjects showed a variation in visual acuity between the two tests. The variation for two of these subjects was two or less lines of Snellen acuity (6/12 and 6/9 Kay's; 6/6 Vizassess) the remaining two subjects showed a larger variation of 6/36 Kay's to 6/12 Vizassess. There is no clear reason for these differences and fatigue seems unlikely as the order of test presentation varied between these subjects. The difference may have been due to the level of interest created by the use of a computer or the dynamic presentation of optotypes however there is insufficient data to provide more than speculation in relation to these four subjects.

Sixty one percent of all subjects could not provide a reliable response to the Kay's picture test. Half of the non-responding subjects were able to provide a reliable response to the Vizassess test. Features of the Vizassess method which may have facilitated subject responses include the short working distance of the Vizassess method. A number of subjects did not maintain concentration over the 3 metre testing distance of the Kay's test but did maintain concentration at the 0.5 metre working distance of Vizassess. This finding is consistent with that of other researchers[9]. The close working distance also enabled the clinician to maintain contact with the subject, this facilitated observation of responses such as visual pointing and promoted rapport increasing subject confidence.

Subjects who were unable to communicate using the required responses to the Kay's picture test were able to visually follow the optotypes of module 1 or refixate to locate a single optotype of module 3. These ocular indicators were repeated with optotypes of the same size before being accepted as a reliable response. The use of visual pointing as a reliable indicator of response is supported by the finding of previous researchers[7,9]. These subjects were unable to communicate by matching and the Vizassess program at this stage does not provide a series of images for subject choice so that visual acuity in these cases reflects the

subject's ability to see an image, but not necessarily recognize it.

Further advantages of the Vizassess method include the ease of changing the size and presentation of the optotypes. This enabled the presentation of a range of stimuli incorporating continuous movement (module 1), change of location (module 3) and change of size. This dynamic form of presentation was not possible using the Kay's test. The Vizassess method was easily operated by one person where as some subjects required a second clinician to facilitate the use of the Kay's picture test.

The computer is used in training activities with the children attending Glenallen school. Teachers at the school report that the children are motivated by the use of computers and are familiar with them. This provided a motivational advantage for the Vizassess test. The children generally were keen to look at the computer screen and susceptible to the suggestion that the test was a game, this concept was more difficult to convey in relation to the Kay's test.

CONCLUSIONS

This pilot project supported Vizassess as a reliable test of visual acuity. The use of computer generated optotypes has provided a test which can be used successfully with some subjects who are unable to respond to conventional tests of visual acuity. The trials and subsequent discussions with teachers and therapists have raised several suggestions for improvements to the pilot program. Such improvements may facilitate assessment of the remaining 30% of subjects who did not respond to either test. A revised computer program based on the information obtained from this pilot trial is currently under development.

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IMPROVING EYE GAZE COMMUNICATION THROUGH OCULAR MOVEMENT EXERCISES

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Abstract

A thirteen year old girl with severe athetoid cerebral palsy, visual and intellectual disabilities was given a series of exercises in maintenance of fixation and saccadic and smooth pursuit eye movements. The aim of the exercises was to give her improved control of eye movements to enable her to access an eye gaze communication board. The exercises were performed daily as part of her school programme with measurements of fixation and refixation time being taken twice weekly. Results showed an improvement in both measurements, however fluctuations were obvious with fatigue, illness and varying levels of co-operation.

Key Words: *saccades, smooth pursuit, cerebral palsy.*

INTRODUCTION

The saccadic and smooth pursuit eye movement systems have been clearly defined as to their pathways, characteristics and function[1]. It has also been determined that developmental changes can be plotted in the saccadic and smooth pursuit systems and in fixational control[2]. The literature clearly defines the different types of cerebral palsy, methods of treatment and the type and extent of improvement that can be expected from that treatment[3,4]. Whether the child with athetoid cerebral palsy can be taught to control eye movements is yet to be determined.

The eye movement systems are represented in the frontal lobes for the saccadic system and the occipitoparietal lobes for smooth pursuit. The saccadic system is responsible for refixation movements, fast phases of vestibular nystagmus, optokinetic nystagmus and microsaccades. The smooth pursuit system is responsible for following or tracking a slowly and smoothly moving target once the saccadic system has placed it on the fovea. The position maintenance system is a non optic reflex system. Its function is to maintain an object of interest on the fovea or to maintain a specific gaze position. Cortical representation is in the frontal and occipitoparietal areas[1].

Development changes occur in the eye movement and position maintenance systems. The velocity of smooth pursuit movements improves

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with age as does the ability to change direction when visually pursuing an object. The saccadic system is thought to be the first well co-ordinated motor system in humans with attention and arousal being the determining factors in how well a saccadic oscillation is performed. Position maintenance or fixational control has been found to improve with instruction and practice[2].

Cerebral palsy has been defined as "a disorder of movement and posture due to a defect or lesion in the immature brain"[3]. Athetoid cerebral palsy results from damage to the extrapyramidal pathways or basal ganglia due primarily to perinatal insults[5]. Children with athetoid cerebral palsy show an unsteady and fluctuating type of postural tone, all movements are jerky, uncontrolled and extreme in range with the uncontrolled movements being increased when the child tries to perform a purposeful movement. The involuntary movements that are seen in the athetoid child consist of intermittent tonic spasms and mobile spasms where the limbs are involved in rhythmic alternating movements of pronation and supination, flexion and extension. Head control is often poor and is frequently associated with disturbances of eye control, speech and hearing[3].

The disturbance of postural tone and difficulty with purposeful movement in these children can result in unintelligible speech. This necessitates the use of an augmentative communication system, where an alternative communication system is used to either complement existing speech or provide the child with a sole means of communication. When developing a communication system for the child with athetosis, direct selection may be used where the alphabet or a series of pictorial representations are pointed to using any part of the body for example a finger, foot or the eyes. Because of the eyes' physical and neural relationship to the brain they are often the last system to be impaired. Eye movements are also low energy movements and are largely independent of postural and other body movement systems[6]. These advantages apply particularly to the child with athetoid cerebral palsy due to the difficulties with controlling movement whilst maintaining a normal background posture.

The relationship between visual and ocular motor disorders and cerebral palsy has been well documented, with there being a high incidence of strabismus and nystagmus[8,9]. Research has also been conducted into eye movements in children with cerebral palsy with results showing that smooth pursuit and saccadic movements tend to be irregular with jerky pursuit movements and slow saccadic movements being described[10,11,12]. It should be noted however that similar irregularities were also observed in children with learning disabilities[11] indicating that the irregularities are not con-fined to cerebral palsy. Whilst the type of cerebral palsy was mentioned in each case, the subjects were thereafter considered as a group with no distinction being made between eye movements in the different types of cerebral palsy, the application of these observations to a child with athetoid cerebral palsy is therefore limited. Whilst the irregularities were identified, there was no attempt to improve these eye movements through any form of exercise in any of the aforementioned studies. The slower saccadic movements have been linked to poor reading and writing skills, motor achievement and visuo-spatial abilities[12].

The relationship between ocular motor disorders and cerebral palsy has been established, but as previously mentioned, it has not been determined whether exercises in these areas will lead to any improvement in the saccadic or smooth pursuit systems. Erhardt (1987)[13] has used smooth pursuit exercises to increase visual awareness and Waters (1987)[14] advocates the "training of visual pursuits and saccades" in children with cerebral palsy as part of their Occupational Therapy programme. both of these authors aim for an improvement in the subject's visual awareness, with the eye movement training being part of an overall stimulation programme. Whilst awareness is stated to improve the authors fail to indicate whether there is any observable change in the subject's ability to perform smooth pursuit or saccadic movements.

Treatment techniques that are specific to

athetosis include developing cognitive control to overcome deficits in specific areas and applying the principle of repetition to "strengthen and consolidate" normal movement patterns[15]. The child with athetosis does not have a problem with initiating movement but rather with controlling that movement and maintaining a normal background posture[5]. Due to these problems the principles of repetition and cognition would need to be applied consistently in all aspects of the child's disabilities. Whilst these techniques have been applied to the general treatment athetosis it has not been determined whether the same principles will apply to control of the ocular motor system.

The purpose of this study was to determine whether visual fixation time and refixation time would be improved through exercises in maintenance of fixation, saccadic and smooth pursuit eye movements. A thirteen year old girl with athetoid cerebral palsy was the subject for the study and the exercises were included as part of her I.E.P. The purpose of the exercises was to improve control of visual fixation and refixation movements and ultimately provide the subject with a means of accessing an eye gaze communication system (figure 1).

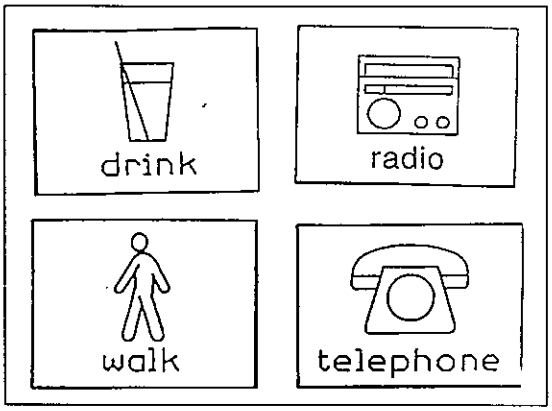


Figure 1. Example of an eye gaze communication system using a perspex card with pictorial representations of activities.

The type and extent of improvement that was recorded is discussed including the effect the exercises had on other aspects of the subject's I.E.P. Possible limitations to the method of measurement and how this project was influenced by other factors apart from eye movement exercises is included.

METHOD

This project was based on a single subject. The subject being a thirteen year old girl with severe athetoid cerebral palsy, a moderate intellectual impairment, optic atrophy, myopia and a left ptosis. Prior to commencing any exercises a baseline measurement was taken of the subject's ability to take up fixation, and then maintain fixation for as long as possible. Both tasks were timed using separate stop watches with the measurements being taken in four positions of gaze to correspond with an eye gaze communication system that was to be introduced (figure 1). The baseline was taken over a two week period with the measurements being taken in the morning prior to the subject commencing school and at the end of the school day.

Having established a baseline the subject was given a series of exercises to be done twice daily five times a week. The exercises are outlined in Table 1.

Table 1:
Orthoptic programme for the classroom

1. Saccades – horizontal-four horizontal eye movements including initial take up of fixation. The two targets to be held at eye level approximately 15 cms apart.

   – vertical-four vertical eye movements including initial take up of fixation. The two targets to be held on the midline, the upper target at eye level with the other target approximately 10 cms below.

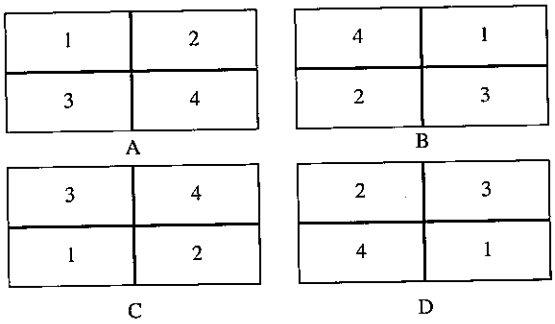
2. Smooth Pursuit - visually follow a slowly moving target firstly on the horizontal plane twice then twice on the vertical plane.

3. Maintenance of Fixation - maintain fixation on a centrally held target for as long as possible.

The exercises were included in the subject's classroom programme with the teacher supervising. This involved an initial demonstration of the exercises by the Orthoptist and then the teacher carried out the programme holding all fixation targets as the subject was unable to do so due to the nature of her disability. Twice

weekly the subject was seen for an individual orthoptic treatment session, a measurement of fixation and refixation times was taken at the commencement of each session, this was then followed by a number of exercises based on the programme in Table 1. Measurements were taken in four different sequences, one sequence per session, to take into consideration fatigue and difficulty maintaining fixation in different positions of gaze. (see figure 2)

Figure 2
Sequences used for measurement



Coloured discs were used to mark each position with the subject being requested to look at a nominated colour and to maintain fixation for as long as possible. Once fixation was lost, the subject was told how many seconds she had maintained fixation then after approximately one minute she was requested to look at the next colour in the sequence. The position of the colours was kept constant, only the order in which the subject looked at them was changed.

RESULTS

Results are presented in the form of line graphs. Figures 3 and 4 show an average of the measurements taken over a morning and afternoon session on the same day for the baseline period and then an average of the four positions of gaze from each measurement session thereafter.

Figure 3 shows an average of the time to take up fixation for each measurement session. As can be seen, measurements over the two week baseline period are erratic with the majority of measurements being over 15 seconds. Once the ocular movement exercises commenced there was an initial peak measurement of 28.5 seconds. Results then show a rapid reduction so that the measurements of time to take up fixation all fall below 10 seconds. The 2 week holiday period resulted in an initial increase in time to take up fixation but this was then followed by a rapid reduction in times which was sustained until the end of the project.

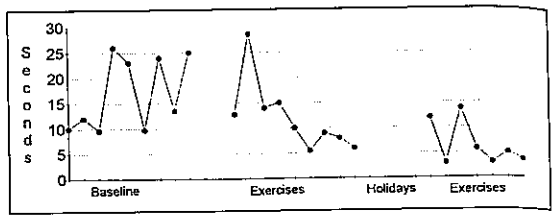


Figure 3. Time to take up fixation - Averages

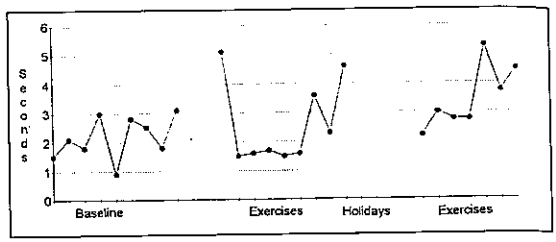


Figure 4. Fixation maintained - Average measures.

Figure 4 demonstrates an average of the time that fixation was maintained for each measurement session. Predictably the baseline period was again somewhat erratic with all measurements falling below 3 seconds. Once the eye movement exercises were commenced there was an initial reduction in fixation time with the average measurement falling below 2 seconds. After 3 weeks of exercises an improvement is evident. The 2 week holiday break was followed by a reduction in fixation time for a further 2 weeks with improvement then becoming evident for the remainder of the project.

It is worth noting that the lower left position of gaze gave the most variable results of the four positions, with both time to take up fixation, and fixation maintenance being longer in this position.

DISCUSSION

The aim of this project was to determine if a subject with athetoid cerebral palsy could improve in her control of eye movements in order to use eye gaze to access a pictorial

communication system. No mention in the literature was found as to whether exercises in ocular movements will result in any improvement however, it has been found that the principles of repetition and cognition when applied to the physical management of athetosis will "strengthen and consolidate" normal movement patterns[15]. These principles plus the statement by Johnston and Pirozzolo (1988)[2] that position maintenance and fixational control have been found to improve with instruction and practice were applied as a basis for this project.

When the results were analysed some improvement was evident in that there was both a reduction in time to take up fixation and a slight increase in the time that fixation was maintained. This improvement was shown in figures 3 and 4 where an average of the measurements from the 4 positions of gaze was taken. It would appear from these two graphs that a greater improvement was achieved in the time to take up fixation. This may reflect an improvement in saccadic eye movements or a combination of the saccades and improved head control.

When the 4 positions of gaze were compared graphically it was found that the lower left position of gaze was the most difficult position for the subject to firstly gain fixation on and then maintain it. This difficulty was particularly evident over the baseline period, with an improvement being evident once the exercises were being done consistently.

Some of the variation in results can be explained by the subject's mood, state of health and level of fatigue on individual days. It was found that fatigue particularly influenced results with time to take up fixation being the most affected. If the subject was very tired, this particularly being evident after she had been involved in a physical activity such as swimming, time to take up fixation was noted to increase, on 3 occasions taking over 1 minute. These longer periods only occurred on the third and fourth measurements taken regardless of position or gaze. It was also found that if the subject was tired or unwell she could frequently maintain fixation for longer perhaps owing to the fact of her being able to "lock" her head into position. All of the subject's random body movements decrease significantly when she is very tired or unwell, this is reflected in her ability to maintain fixation for longer probably due to the fact that random head movements also decrease. Results were also noted to be affected if the subject was in a good mood with laughter tending to influence results by increasing the subject's random body movements. This produced the effect of time to take up fixation increasing markedly and fixation being maintained for shorter periods. This occasionally necessitated taking a second series of measurements after the subject's full co-operation was sought.

Once an improvement in the measurements became obvious it was found that this improvement was reflected in other aspects of the subject's I.E.P. As her understanding of the purpose of the exercises became evident this was reflected in her being more willing to co-operate. The subject's classroom teacher was then able to use eye gaze for choice making in a number of situations throughout the school day. For example: providing the option of the choice of a drink for morning tea by holding up a carton of milk and one of fruit juice and asking the subject to look at her preference. Once the subject had successfully utilised eye gaze for selection on several occasions she became more interested in the project and more eager to participate in the exercises as she could see the positive benefits of gaining control over her eye movements.

The fact that the subject had a ptosis made it easier for people involved in her I.E.P. to interpret her requests. Once it had been explained that she was only using her right eye and that the person who was interpreting her request should disregard the eye with the ptosis, it was found that people were more willing to use the eye gaze communication system.

The introduction of 4 positions of gaze for this project was made to give the subject a wider variety of choices. It was found however that the 2 lower positions were more difficult for the subject to fix upon thus leading to

frequent misinterpretation of her choice if the person interpreting her choice was not familiar with the subject. The choice could be confirmed by asking the subject if she was looking at the interpreted selection, which she could confirm by using her YES/NO response (lips pursed for no, smile and "ah" for yes). Due to this difficulty, people who were not familiar with the subject were advised to provide her with 2 choices which allowed for a more rapid selection by the subject and a more accurate interpretation by the person she was communicating with.

CONCLUSIONS

The use of exercises that involve repetitive saccadic and smooth pursuit movements and the training of maintenance of fixation was found to be beneficial in teaching a subject with athetoid cerebral palsy to control her eye movements. This was reflected in a reduction in time to take up fixation and a slight increase in the time that fixation was maintained. It was found that a corresponding use of eye gaze for communication in all aspects of the subject's I.E.P. increased the subject's motivation to co-operate as she could see the positive benefits of the programme. It is felt that the eye movement exercises assisted in the measured improvement but it cannot be ascertained whether this improvement was also brought about by the increased use of eye gaze for communication. The subject's motivation and understanding of the project were key factors in gaining a reliable response and by providing her with feedback on how results were showing an improvement, both within a treatment session and over a period of time, her enthusiasm for the project was maintained.

The principle of repetition and the use of cognitive skills in the general management of athetoid cerebral palsy have been found to be effective[15]. By using exercises that are based on repetitive eye movements and by seeking the full co-operation of the subject these principles were applied for the purposes of this project. It would appear from the positive results that were achieved, that a person with athetoid cerebral palsy can be taught to control eye movements as well as learning to control postural tone and general body movements. The results gained in this project are based on observation, and a number of factors need to be taken into consideration when accounting for the improvement. These factors include the exercises in saccadic and smooth pursuit eye movements, the increasing use of eye gaze throughout the subject's school programme, the subject's motivation and her degree of co-operation.

In order to determine whether the eye exercises resulted in an improvement in saccadic or smooth pursuit eye movements a more accurate measurement system would be necessary to record any change to the eye movements than the method used in this study. The electro-oculogram has been used to record saccadic and pursuit movements in children with cerebral palsy[10]. It is suggested that this would provide the means to determine whether saccadic and smooth pursuit movements can improve with practice. Physical restrictions on this project meant that this form of measurement was not possible. The fact that this project was conducted within a school meant that the use of the eye exercises and their application could be constantly monitored, however the disadvantage was that the abovementioned clinical measurements were not possible. One other means of determining if the eye movements could be improved would be to limit the use of eye gaze to the exercises and measurement sessions avoiding the use of eye gaze for choice making. If these restrictions were applied it would be difficult to maintain the motivation and co-operation of the subject, however it would provide a more "pure" set of measurements.

ACKNOWLEDGMENTS

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THE EFFECTS OF CENTRAL AND PERIPHERAL BINOCULAR VISUAL FIELD MASKING ON FUSIONAL-DISPARITY VERGENCE

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Emmie Russell Prize 1993

Abstract

Clinicians have long recognised the effectiveness of the peripheral fusion mechanism in eliciting fusional movements and have employed this in the investigation and treatment of squint. In 1939, Burian was the first to demonstrate that fusible stimuli placed in the peripheral binocular field only, were capable of producing fusional movements. The aim of the present study was to investigate the effects of masking of the central and peripheral binocular fields to determine the influence of each on the horizontal fusional amplitudes, and on the duration of jump fusional-disparity convergence movements in normal binocular vision. Twenty one (n=21) young adult subjects took part in the study. The fusional amplitudes were measured with a Risley rotating prism. The jump vergence movements were assessed using an infra-red eye movement recording system. Measurements were performed at 3 meters using full, central and peripheral field stimulus conditions. It was found that under central stimulation, the fusional amplitudes for both base-in ($p=0.0008$) and base-out ($p<0.0001$) directions were significantly lower than those under peripheral stimulation. Also, peripheral field masking significantly prolonged the duration of a disparity vergence response induced by a 10Δ base-out prism ($p=0.0009$), but not by one of 5Δ ($p=0.0507$). It was concluded that peripheral fusion plays an important role in the production of fusional-disparity vergence eye movements.
Key Words: *Peripheral fusion, peripheral field masking, fusional amplitudes, disparity vergence, jump convergence.*

INTRODUCTION

Clinicians have long recognised the effectiveness of the peripheral fusion mechanism in eliciting fusional movements and have employed this in the investigation and treatment of binocular dysfunction and squint. Even when orthoptics was still in its infancy, Swan and Laughlin[1], in 1944, emphasised the importance of utilising peripheral fusion to train fusional reserves in amblyopic patients with anomalous binocularity. Similarly, Burian[2] stressed the place of peripheral fusion in orthoptic training. Indeed, orthoptists have moved away from training patients with anomalous binocular vision[3], but the use of peripheral stimulation in strabismic patients with 'normal binocular functions' is still advocated[4]. This is certainly the case in the assessment and treatment of the vergence system's dysfunctions[5-8]. However, despite the acceptance of peripheral fusion as a

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powerful factor in the production of fusional movements and the current knowledge of the intricacies of the vergence system, little research has been done into the relative contributions of the peripheral and central fusion mechanisms to fusional vergence. This paper will attempt to address the effects of peripheral and central fusion, firstly on the horizontal fusional vergence amplitudes, and secondly on the duration of a jump convergence movement.

In 1939, Burian[9] was the first to demonstrate that fusible stimuli place in the peripheral binocular field only, were capable of producing fusional movements. In that experiment, targets subtending angles of 0.5° and 1° were haploscopically projected 12° from the fovea and separated vertically to produce fusional sursumvergent movements. Hampton and Kertesz[10] and Winkelman[11] carried out similar investigations and confirmed Burian's findings. In another experiment in which a solid black circular target was surrounded by an annulus of print, Burian[9] found that peripheral fusion could dominate central or foveal fusion and disrupt it if the peripheral stimuli covered a sufficient area. In spite of the subjects' attempts, the central diplopia could not be fused as long as the disparate peripheral stimuli were present. Burian concluded that the peripheral retina plays an important part in the fusion process.

Since Burian[9] introduced the now widely used term *peripheral fusion,* similar findings have been reported[11-15]. Sullivan and Kertesz[14] demonstrated the influence of the peripheral and central binocular fields on the cyclofusional response. They concluded that peripheral fusion exercises a stronger influence than central fusion on cyclovergence or motor cyclofusion. Using objective recording, it was found that the largest target (50° in diameter) produced the greatest cyclovergence component of the response, this being about 60% of the total cyclodisparity (5.75°). In fact, targets of less than 30° apparently did not induce any motor component. Also, when conflicting torsional disparities in the centre and periphery were simultaneously presented, cyclovergence only occurred in the direction of the peripheral disparity. Furthermore, when the torsional disparity in the central 30° region was removed, an even greater cyclovergence component was found, this being about 76% of the total cyclodisparity. It would appear then that the presence of central field stimulation actually hindered the motor response. However, it must be pointed out that only one subject underwent all the experiments and that this subject demonstrated a training effect on immediate retesting that diminished on a subsequent test session about one month later.

Kertesz[7] also investigated the effect of increasing peripheral stimulation on horizontal and vertical fusional responses. It was found that increasing the stimulus size from 5° to about 58° resulted in a three-fold increase of the horizontal fusional range in both of the subjects tested. Indeed, the average increase in the two subjects tested was from 9.04° and 10.98° to 27.75° and 26.62°, respectively. Kertesz and co-workers[16,17] also demonstrated that step disparities of 1.5 and 5° could induce jump vergence movements (in both convergent and divergent directions) when the large-field stimulus of one eye contained a 10° central scotoma. It is evident therefore that increasing levels of vergence stress and jump vergence to step disparities can be sustained by the peripheral fusion mechanism alone.

That peripheral fusion plays an important role in producing fusional vergence movements has therefore been supported clinically and experimentally by many researchers. However, the belief that peripheral fusion is a more powerful force than central fusion has been disputed by other researchers[18-20]. Lyle and Foley[20] employed a central fusion stimulus of 0.5° and varying peripheral stimuli of 2.5°, 5° and 10° presented either anaglyphically or by polarisation. They found that most of their subjects (64% with the red-green method and 80% with the polaroid test) reported peripheral target doubling before central, indicating that central fusion was the stronger mechanism.

Ludvigh and co-workers[18,19] objectively recorded fusional convergence movements made in response to small (6 x 25') targets positioned

at 0°, 0.25°, 0.5°, 1°, 2° and 4° from the fovea. It was demonstrated that even when the stimuli were positioned as little as 0.25° off centre, the vergence response decreased in all three of their subjects tested (even though 0.25° or 15' is certainly within the normal limits of fixation disparity[21] and is smaller than the precision of fixation itself[22]). These findings therefore suggest that fusional responses made to identical stimuli are maximal when they are foveally positioned. However, it may be of significance that the target was moved at a rate of 1.1° per second, and that it has previously been reported that peripheral fusion is more robust with prolonged exposure time of the peripheral stimulus[11]. In another experiment that was similar to Burian's[9], Ludvigh et al[18,19] used competing peripheral stimuli (consisting of six concentric split rings) in an attempt to interrupt central fusion. They found that these "powerful peripheral stimuli" were incapable of disrupting central fusion "except, perhaps, momentarily" (Ludvigh et al[19], 1965, p. 120).

It is clear from the above studies that there is some confusion as to the relative contributions of the peripheral and central components of the binocular visual field to the fusional response. Indeed, while there is general agreement that the peripheral fusion mechanism is effective in producing fusional movements and can, in fact, sustain a position of orthotropia or microtropia in the absence of central binocularity[23,24], some quite contradictory findings are apparent in the literature. This problem was recently addressed by Cooper, Feldman and Eichler[25]. In their initial experiments, it was demonstrated that central fusion was dominant, firstly when equal-sized stimuli were placed at various retinal eccentricities, and secondly when larger peripheral stimuli were used. Hence, their findings confirmed those of Lyle and Foley[20] and Ludvigh et al[18,19]. However, a subsequent investigation using a central circular stimulus surrounded by a peripheral annulus produced converse findings: that peripheral fusion dominated. This explains and is consistent with the results of Burian[9] and Sullivan and Kertesz[14], but not of Ludvigh et al[18,19], whose experiments were all very similar. Finally, Cooper et al[25] increased the extent to which the peripheral stimulus surrounded the central one and found that this was the most important variable controlling fusional movements. Although this was a most important finding and explained the discrepancy in the literature over the previous five decades, it must be acknowledged that Burian[9] (1939, p. 489) had previously stated:

> "One of the most important results ... is that peripheral retinal stimuli are strong enough to break the fusion of images situated on corresponding areas of the macular region, provided they cover a sufficient area...".

Previously, most studies that have addressed the issue of peripheral versus central fusion used stimuli that were projected haploscopically and usually in the dark. To the author's knowledge, no study has attempted to determine the relative contributions of the two mechanisms to normal fusional vergence by masking of parts of the binocular visual field. It is the aim of this study to address the effects of central and peripheral field masking, firstly on the positive and negative fusional vergence amplitudes, and secondly on the duration of a jump convergence movement induced by prismatic step disparity.

METHOD

Subjects

Twenty one (n=21, mean and median age=19 years) orthoptic students, semi-naive to vergence testing, participated in this study. All subjects had normal bifoveal binocular single vision, meeting the following inclusion criteria:

i. random-dot stereoacuity to 550" on the Lang stereotest;

ii. no heterotropia on cover test;

iii. no significant esophoria (>2Δ), hyperphoria (<Δ2) or exophoria (>6Δ) on alternative cover testing at 3 meters;

iv. bifoveal motor fusion response on the 4Δ prism test. Two of the subjects had a small amount (<-2.00 DS) of myopia that was corrected for testing.

Apparatus

Objective recording of the fusional vergence movements was done using the Ober-2 Eye Movement Registration System (*Permobil Meditech AB*, Timrå). The principle of this infrared unit is the differential reflective properties of the iris and the sclera. It involves the subject wearing goggles that house an emitting light source and two pairs of infrared sensitive photoelectric cells that intercept the reflected light. As the eye moves, the cell in the direction of movement receives less illumination since the infrared light is absorbed by the iris, while the other cell receives more illumination from light reflected off the sclera. The amplitude of the potential change from the cells is proportional to that of the eye movement. Recordings were made with the sampling rate set at 100 Hz. The unit's goggles have been fitted with a pair of Halberg clips on which trial frame type glass prisms were mounted during the experiments. The fusional vergence amplitudes were measured using a Risley rotary prism and glass trial frame prisms. The fixation target consisted of a full sized Bailey-Lovie LogMAR visual acuity chart, modified with a 6/12 sized letter on a white background in the centre. Peripheral field masking was achieved using a thin tube (18mm in diameter, 95mm in length) mounted onto a circular trial frame lens holder. This allowed an 8° central binocular visual field. Central field masking was achieved by an after-image produced by a visuscope. This precluded binocular vision in the central 8° of the visual field[26].

Procedure

All measurements were performed at a fixation distance of 3m. The subjects were instructed to fixate the 6/12 letter on the LogMAR chart fixation unit which was positioned at eye level. The testing was carried out in an artificially lit room of 360 lux.

Testing under full (central-plus-peripheral) binocular field stimulation was done first. This was followed by peripheral binocular field masking with the tube before the left eye. It was ensured prior to each measurement that the subject saw the fixation target inside the central field of the left eye. Testing with central binocular field masking, was accomplished using a visuscopic afterimage applied to the left eye for 30 seconds. Holding the visuscope close to the left eye while closing the right, the subject was instructed to fixate only the star in the centre of the grid, so that no more than the central 8° of the retina was 'bleached'. Foveal fixation was confirmed objectively during the 30 seconds. This process was repeated before each measurement or recording in order to maximise the central masking effect. Testing under peripheral stimulation was performed last, that is, after the fusional amplitudes were measured and jump convergence responses recorded under both full and central stimulation, since complete visual re-adaptation did not occur for at least several minutes afterwards.

The fusional vergence amplitudes were assessed first, the base in (BI) direction being measured before base out (BO). A larger sized letter was fixated when the BO amplitude was being measured, since the increasing convergence accommodation would have blurred the 6/12 letter. The total values were adjusted appropriately when the prisms were divided between the two eyes or stacked upon each other[27]. The limits of the fusional vergence amplitudes were recorded as being the strongest prism that would allow single vision to be maintained. When measuring under peripheral stimulation only, the subject was instructed to take note of peripheral diplopia, that is, doubling of the edges of the LogMAR chart, as central diplopia would not be appreciated.

The jump convergence responses to prisms were recorded after measurement of the fusional amplitudes. The 5Δ BO prism response was tested before the 10Δ response, firstly under full field† and then central field stimulation. The responses under peripheral stimulation were assessed afterwards. The subject was positioned at a head and chin support unit with a BO prism before the right eye which was

† The unit's goggles restrict the binocular visual field to at least 60° horizontally and 40° vertically.

neutralised by a BI prism. At the onset of the recording, the BI prism was quickly removed from the goggles' trial frame, effectively creating a step convergent disparity of 5Δ. The subject was instructed to join the resultant double images as quickly as possible and remain fixating on the target.

The beginning of a vergence movement in response to a prism disparity was in most instances (83.9%) preceded by a saccade, with or without a vergence component, in the direction of the prism apex. As this *vergence saccade* has been thought to play an important role in the execution of a vergence movement[28,29], it was included in the duration measurement of the vergence response. Therefore, the time taken to fuse the BO prism included the vergence phases as well as this initial unequal saccadic movement (and any other vergence saccade that may have been apparent during the fusional vergence response). A typical recording is shown in Figure 1.

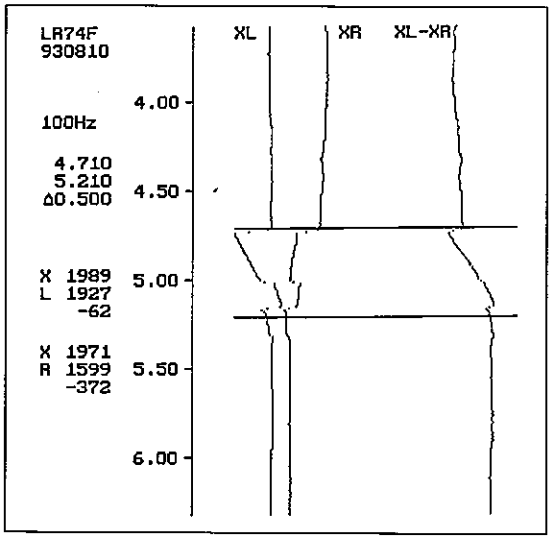

Figure 1. A typical recording of a 10Δ BO response (prism before RE) under full stimulation. XL is the trace of the left eye, and XR the right. Lefward movement of the trace indicates that the eye is moving to the left. XL-XR represents the difference between the two eyes and therefore indicates changes in vergence. For this trace, rightward movement indicates overall convergence and leftward movement indicates overall divergence. The upper calibration line indicates the beginning of the response and the lower line indicates the end, the duration of this response being 0.50 seconds.

Data Analysis

The statistical tests used were: the 1-way (single factor repeated measures design RMD) analysis of variance (ANOVA) test and the 2-way (2 factor RMD with 2 repeated measures) ANOVA. The level of significance (∞) was set at 0.05.

RESULTS

Fusional vergence amplitudes

The mean BI amplitude for full field stimulation was 5.91Δ (with a standard deviation, sd, of 1.45). For central stimulation, the mean was 5.57Δ (sd=1.91), while for peripheral stimulation it was 6.86Δ (sd=1.96) (Table 1.). The BI amplitudes for peripheral stimulation were significantly higher than those for full and central stimulation ($F_{2,40}$=8.505, p=0.0008).

Table 1
Summary table of result means

		Stimulus Field		
		Full	Central	Peripheral
Fusional vergence amplitudes	BI (Δ)	5.91	5.57	6.86
	BO (Δ)	36.29	18.38	36.24
% subjects able to converge to	5Δ	100	100	96.2
	10Δ	100	76.2	100
Duration (s) of prism responses to	5Δ	0.45	0.67	0.52
	10Δ	0.72	1.79	0.71

The mean BO amplitude for full field stimulation was 36.29Δ (sd=10.48). For central stimulation, the mean was 18.38Δ (sd=5.87), and for peripheral stimulation it was 36.24Δ (sd=8.49) (Table 1.). As would be expected from these values, the BO amplitudes for both full and peripheral stimulation were significantly higher than those for central stimulation ($F_{2,40}$=54.863, p<0.0001).

Execution of jump convergence movement to a step disparity.

All subjects were able to execute a jump convergence movement to a 5Δ BO prism under all three stimulus conditions (Table 1.) However, two recordings were excluded from the statistical analyses: one subject's 5Δ response to peripheral stimulation was accidentally deleted from the computer's hard disc; in another subject, the 5Δ response to peripheral

stimulation was not clear, even on a repeated recording. In fact, the response appeared more like a divergence movement when the recording was analysed.

All of the subjects were able to execute a jump convergence movement to a 10Δ BO prism under full and peripheral field stimulation, however, under central field stimulation, only 16 of the 21 (76.2%) subjects could do so (Table 1.). The 5 subjects who could not fuse the prism, in fact, noticed diplopia for the whole 20 second recording period.

Effect of peripheral and central field masking on duration of the jump convergence movements to a step disparity.

The mean duration of the jump convergence movements made to a 5Δ step disparity for full field stimulation was 0.45 seconds (s) (sd=0.13) (Table 1.). The mean duration for peripheral stimulation was 0.52s (sd=0.35), and slightly longer for central stimulation, 0.67s (sd=0.23), although these differences just failed to reach statistical significance ($F_{2,36}$=3.244, p=0.0507). Therefore, masking of the peripheral and central binocular visual field did not have a statistically significant effect on the duration of a vergence response to a 5Δ prism.

Nevertheless, this was not the case for the 10Δ responses under the three stimulus conditions. The mean duration for central stimulation was 1.79s (sd=1.46), which was significantly longer than for full and peripheral stimulation, 0.72s (sd=0.22) and 0.71s (sd=0.32), respectively (Table 1.) ($F_{2,30}$=8.940, p=0.0009). This difference between the 5Δ and 10Δ responses also showed up a statistically significant interaction effect ($f_{2,28}$=4.712, p=0.0172). That is, the effect of peripheral field masking differs according to whether a 5Δ or a 10Δ prism was used.

DISCUSSION

Unlike most previous experiments, normal viewing conditions were used in this study in that the subject viewed in free-space while light adapted. However, one limitation was that the order of the prism strengths presented and the binocular visual field conditions used was not random. It is the author's contention that this was perhaps the reason for the marginally non-significant difference in the mean duration of the jump convergence movements to a 5Δ prism under the three viewing conditions. It is possible that the responses under central field stimulation, for example, were made easier since the subject had just fused a 5Δ and then a 10Δ BO prism under full stimulation.

There was an extraordinary difference in the BO fusional vergence amplitude under the three stimulus conditions. In fact, the mean amplitude for central stimulation was half that for both full and peripheral stimulation. Peripheral fusion obviously plays an important role in producing and maintaining a gradually increasing position of fusional convergence. This finding is in agreement with the observation of Parks.[26] The sheer extent of the retinal periphery must play a role here. Furthermore, it is probable that central vision is degraded by the associated convergence accommodation to the point where fusion in that region is eventually disrupted. Central fusion is the fine-tuning vergence mechanism[30], partly because of the visual integrity there, so blur must be a hindrance to its function, despite the fact that disparity has been considered to be quite robust to blurring[31,32]. Peripheral vision and indeed fusion, on the other hand, is not as sensitive to this blurring while the BO disparity is being increased[33]. Moreover, that this was not the case for the BI amplitudes lends support to how blurring due to the associated convergence accommodation plays a role in decreasing the BO amplitudes under central stimulation. Clearly, this does not occur when fusional divergence is stimulated. Alternatively, because the BI amplitude is inherently lower than the BO, the central 8° binocular field may have been sufficient to sustain fusion. The latter explanation is probably unlikely, however, since vertical and torsional fusional amplitudes are also relatively small, but they are demonstrated to be larger using peripheral stimulation[6,14].

The difference in the BI amplitudes under the

three stimulus conditions, however, was less striking than that for the BO amplitudes. It was found that the mean BI amplitude under peripheral stimulation was significantly higher than for both full and central stimulation. That this was due to a training effect is unlikely, although the fact that the diplopia threshold is greater in the periphery, given the increase in Panum's areas and decrease in visual resolution may explain this.

Peripheral field masking also had adverse effects on the execution of jump convergence movements, at least to a 10Δ step disparity. Whereas all subjects could jump converge to a 5Δ BO prism, about one quarter of them could not overcome a 10Δ BO prism under central stimulation and experienced diplopia for the whole recording period. Similarly, the duration times of the 10Δ responses were significantly prolonged under central stimulation, although the slightly longer duration times under central stimulation for the 5Δ responses were found to be statistically insignificant. Obviously, peripheral fusion is important to the processing and dynamics of step disparity vergence. The fact that the 10Δ responses were more affected than the 5Δ responses is probably in part a function of the amount of disparity that was induced within the central 8° binocular field. Also, the lesser disparity of 5Δ, allowed a greater overlap of the two central fields seen in diplopia than the 10Δ disparity. Consequently, it would be a relatively easier task to fuse the 5Δ prism, whereas greater voluntary effort would be required to fuse the 10Δ prism. Besides, it is not unreasonable to expect that the higher the disparity induced, the greater the voluntary or conscious effort required to achieve fusion.

Owing to the anatomic and physiologic differences between the retinal periphery and centre, and their respective topographic representation in the 'binocular centres' of the cortex, it is not unreasonable to expect that peripheral and central fusion have quite differing features and functions. Therefore, it is difficult to impartially compare the performance of the two systems for a set binocular task, especially when artificial viewing or stimulus conditions are employed. Besides, it is very probable that peripheral and central fusion complement one another, given the many factors involved in the fusional process and the production of fusional-disparity vergence eye movements. For example, as was evident in the present study, during gradually increasing fusional convergence stress, peripheral fusion may assume a greater role as it is more resistant to the blur that results from the convergence accommodation than central fusion. Finally, it is recognised that the control of vergence and the extent to which it is influenced by the central and peripheral fusional mechanisms is susceptible to voluntary control and selective attention[34,35], thus adding to the difficulty of evaluating the integrity of the two systems.

CONCLUSION

It seems that the peripheral fusion mechanism in normal binocular vision is very capable of producing full fusional vergence amplitudes in both BI and BO directions, whereas the central fusion mechanism is not. One of the most significant findings was that the BO amplitudes tested under central stimulation, were about half those for the full and peripheral stimulus conditions. It was found that peripheral fusion was more effective than central fusion in eliciting jump convergence movements made to a 10Δ BO prism, but that there was no significant difference in the responses to a 5Δ BO prism. Furthermore, it was demonstrated that the duration of a jump convergence movement to a 10Δ BO prism was significantly longest under central stimulation, taking on average about 1 second longer than for full and peripheral stimulation. In conclusion, there can be no doubt that peripheral fusion plays an important role in the production of fusional-disparity vergence eye movements and that masking of this component of the binocular visual field can be debilitating to vergence dynamics.

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DO TABLE TENNIS PLAYERS HAVE BETTER EYE MOVEMENTS?

A study of the latencies of horizontal saccades in table tennis players and non-table tennis players.

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Abstract

Previous research using qualitative measures of saccadic eye movements suggests athletes make faster and more accurate saccadic movements than do non-athletes. The latencies of horizontal saccadic eye movements were measured in a group of fifteen elite table tennis players and a group of non-table tennis players who had only a moderate involvement in sport. Subjects in each group had normal binocular single vision and little or no refractive error. To stimulate horizontal saccades a computer generated stimulus was presented at intervals of 0.5 and 0.3 seconds and at random horizontal amplitudes between 5 -10°. The resulting eye movements were recorded and analysed using an Ober2 infra-red eye movements system.

Results showed that, whilst the mean latencies of the table tennis players did not differ significantly from those of the non-table tennis participants, there was a group of table tennis players whose saccadic latencies were distinctly faster than the norm. These players demonstrated anticipatory saccades, ie, they predicted, and initiated a saccade before the presentation of the stimulus. It was concluded that future research should use larger number of subjects and investigate the role of training in the development of anticipatory saccades.

Key Words: *Saccadic eye movements, Table tennis players, Saccadic latencies, Anticipatory saccades.*

INTRODUCTION

Many studies have demonstrated differences between athletes and their non-athletic counterparts on a number of visual tasks, such studies having their origins in the work of Winograd in 1942[1]. Many of the earlier studies listed below have used imprecise observation techniques to gather their data on eye movements of athletes. Trachtman[2] in a study of 36 little leaguers (baseball players) aged 10 to 12, compared ocular motility with batting averages. The results showed a correlation co-efficient of +0.44, significant beyond the 0.01 level between ocular motilities (saccades and pursuits) and batting averages. Falkowitz and Mendel[3], in a study similar to Trachtman's, showed that the best little leaguers generally have the best saccadic and pursuit movements. Christenson and Winkelstein[4] also found that the athletic population had significantly better visual performances, including saccadic movement. However, it appears that much of this research on saccadic movements and sport (including the above three studies) has been

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based on purely qualitative analysis of saccades. For example, Christenson and Winkelstein[4] had their subjects read numbers from left to right as quickly as possible and recorded the time it took to complete the task, whilst noting factors such as head movement, postural deviation and finger pointing.

Versino et al[6] emphasise that a quantitative rather than a qualitative approach to measuring saccades is important to improve the clinical applicability of saccadic eye movement analysis. Modern eye movement systems such as the EOG and infra-red systems are capable of documenting such eye movements to a high degree of precision and most current analyses rely on quantitative data from such systems.

Table tennis is a sport in which two opposing players hit a small ball back and forth across a table often at high speeds. To be played successfully and at a competitive level it requires precise reflexes, and accurate visual judgement and eye movements. Saccadic eye movements in these subjects must not only be accurate but, because of the speed of the game, must also be initiated as quickly as possible. Experienced and skillful players could then be expected to have eye movements superior in speed and accuracy to those of their non-athletic counterparts as has been suggested by the above authors[1,2,3]. This study aims to test one of the hypotheses that can be derived from the above arguments: that the latencies of horizontal saccades of a group of elite table tennis players will be shorter than the latencies of horizontal saccades of non-table tennis players.

METHOD

Subjects

The subjects consisted of 15 elite table tennis players (age range 15 to 25 years) who were recruited from New South Wales Table Tennis Association, and 15 university students (age range 18 to 25 years) who had no previous experience in competitive table tennis or any other competitive sport requiring precise vision. Subjects in each group had normal binocular vision and little or no refractive error.

Materials

Eye movements were recorded and analysed using an Ober2 infra-red eye movements system. Infra-red reflectometry is based on the different reflective properties of the various parts of the eye: the sclera will reflect more infra-red light than the coloured iris. By using this difference, the position of the eye is defined, based on measurements made by optical detectors and associated electronics. With four such circuits arranged around the eye in a pair of goggles, both horizontal and vertical eye movements can be recorded and quantified. (Further information about this infra-red measuring technique can be obtained from the manufacturer [Permobil Meditech AB, S-861 00 Timra, Sweden]).

Procedure

The procedure of the study was fully explained to subjects who then gave their informed consent.

The subjects were seated 50cms from a computer screen. A head stabilising frame was fitted and a bite bar was also used to minimise head movement. Both groups were tested in a room lit by a standard 15 watts fluorescent light, with no external distractions, to allow the subjects to concentrate on the computer screen and stimulus presentation. The computer generated stimulus of a vertical white cross 3mm high by 3mm wide was presented in random horizontal positions between 5 degrees and 10 degrees over a period of approximately 16.5 secs. Subjects were exposed to two series of presentations. For the first run the stimulus was presented at intervals of 0.5 secs then, for the second run, at intervals of 0.3 secs. It was decided to select a sample of saccades for analysis as analysing every saccade would have been very time consuming. In order to equalize the number of right and left saccades in a sample it was decided to select every third saccade. It was intended that, for the first run(where the stimulus moved every 0.5 secs) the saccadic movements analysed would be those made in response to the stimuli presented at 1.5, 3.0, 4.5, 6.0, 7.5, 9.0, 10.5, 12.0, 13.5,

Table 1
Results at each measured stimulus, presentation at 0.50 seconds

Time	2.00	3.00	4.50	5.50	7.00	8.50	10.00	11.50	12.50	14.00	15.50
Degree	9.70	5.20	9.40	6.60	7.00	7.40	7.00	6.00	9.00	8.40	5.60
Table Tennis Players											
Mean	101.07	53.00	82.47	43.80	7.93	3.67	-17.40	37.60	-28.27	-23.20	54.60
Standard Deviation	50.86	156.70	112.68	149.59	125.14	146.76	179.07	142.08	107.01	122.18	141.33
Control Group											
Mean	180.80	142.93	83.47	75.20	65.07	74.13	62.40	102.27	27.73	29.33	109.87
Standard Deviation	53.29	28.82	79.14	71.18	92.12	70.86	78.09	65.15	91.11	110.91	71.54

Table 1
Results at each measured stimulus, presentation at 0.30 seconds

Time	1.30	2.20	3.40	4.90	6.30	7.70	9.10	10.60	12.10	13.60	15.60
Degree	5.00	7.30	9.40	6.90	9.20	8.20	8.90	6.30	10.00	7.10	5.60
Table Tennis Players											
Mean	152.62	68.92	103.69	103.38	123.69	128.00	127.08	100.62	126.85	103.54	115.69
Standard Devistion	14.79	89.24	86.95	92.82	63.10	78.31	75.56	120.18	84.91	123.06	115.50
Control Group											
Mean	167.20	113.07	134.07	125.13	140.53	153.13	177.00	147.80	147.53	151.13	154.33
Standard Deviation	29.18	45.41	62.78	80.43	43.20	43.44	54.00	75.10	115.67	79.20	75.14

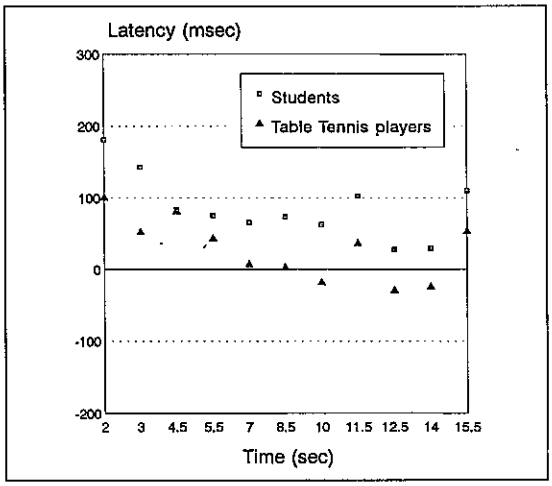

Figure 1. Mean latencies of saccadic eye movements at each measured stimulus in the experimental and control groups. Stimulus presented at 0.5 secs

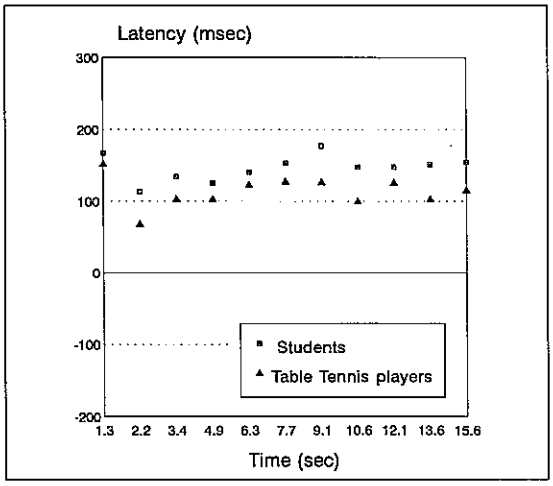

Figure 2. Mean latencies of saccadic eye movements at each measured stimulus in the experimental and control groups. Stimulus presented at 0.3 secs.

15.0, and 16.5 secs. Unfortunately at some of these times there were more than two corrupt measurements (usually due to a blink) across the group. To avoid including these corrupt measures the sampling times were slightly adjusted, in that if there were more than two corrupt recordings within the group at a particular time the next non-corrupted recording time was taken. Similar adjustments were also necessary at the 0.3 secs level. (See figs. 1 & 2)

Design

There were two groups of subjects; table tennis players, and non-table tennis players. There were two conditions of measurement; targets moving at 0.5 secs, and targets moving at 0.3

secs. Results from these two measurement conditions were analysed separately, In each measurement condition 11 repeated responses were sampled from each subject. consequently the research design for each one of the two latency conditions was a 2 (subject type) x 11 (saccadic movements) analysis of variance with repeated measures on the second factor. These data were analysed using the MANOVA program from SPSS.

Results

The research hypothesis tested in this study was that the latencies of horizontal saccades of the group of elite table tennis players will be shorter than the latencies of horizontal saccades of non table tennis players.

The mean latencies and standard deviations for each group are summarised in Tables 1 & 2. A comparison of the mean values at each measured presentation between groups can be seen in Figures 1 & 2, and show that the table tennis players appear to have shorter mean latencies than the non-table tennis players for all occasions at both 0.3 and 0.5 sec stimulus presentation. However, the variation in each group for each measured presentation was considerable, giving large standard deviations, particularly amongst the table tennis players. These larger standard deviations meant that the differences between each group were not significant at the p=<0.05 level. A two (group) x 11 (presentations) analysis of variance, with repeated measures on the 2nd factor, showed there was no difference between the table tennis players and the non-table tennis players at 0.5 secs ($F_{1,28} = 3.86$, p=0.06) nor was there a group by time interaction ($F_{10,280} = 0.68$, p=.739). Similarly, there was no group difference at 0.3 secs ($F_{1,26} = 1.76$, p=.196) and no group by time interaction ($F_{10,260} = 0.39$, p=.948). Consequently, the research hypothesis has not been supported by the analysis.

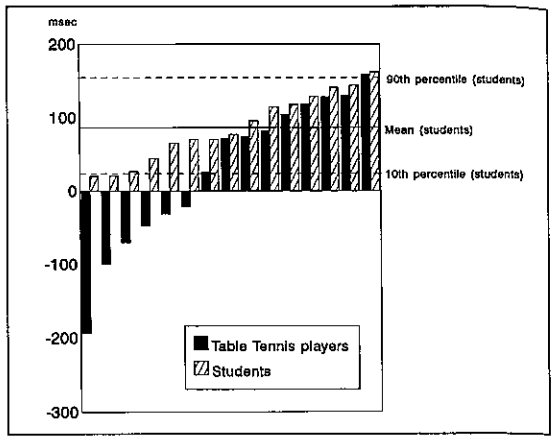

Figure 3. Individual mean latencies of all subjects. Stimulus presented at 0.5 secs.

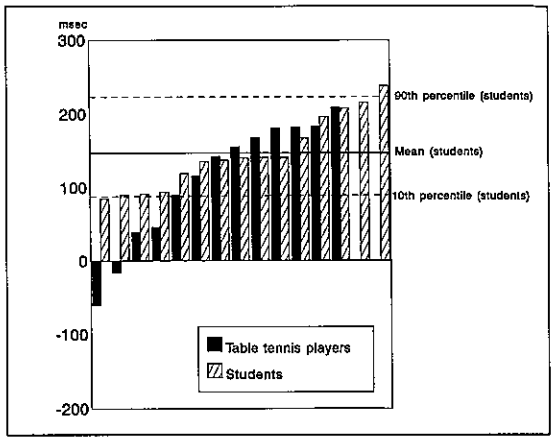

Figure 4. Individual mean latencies of all subjects. Stimulus presented at 0.3 secs.

DISCUSSION

Failure to support the research hypothesis suggests either that it is wrong or that the method of testing has been inappropriate. The present results are inconsistent with those of previous researchers[1,2,3] whose work led to the hypothesis. This study used more trustworthy measures of saccades so its results would be expected to be more reliable. This argument would lead to rejection of the hypothesis as being false. However, inspection of the data indicates another possible explanation. As noted in the results section, there was considerable variation in subjects' latencies. Figures 3 and 4 illustrate the mean individual latencies in each group When the stimulus was presented at 0.5 sec intervals, it can be seen that, whilst the mean latencies of nine table tennis players did not differ from those of the students, six (40%) players had saccadic latencies which were

distinctly faster than the normal, being below the tenth percentile of the student group. These players usually had negative latencies i.e. they predicted and initiated a saccade before the presentation of the stimulus. (In fact any latency shorter than approximately 80 milliseconds can be regarded as a prediction, as this represents the minimum processing time for a saccade to be initiated.)[6]

This pattern persisted when the stimulus was presented at 0.3 sec intervals. Although the latencies of the saccade were relatively longer, there was still a group of four table tennis players (30%) whose latencies were below the tenth percentile of the student group (Unfortunately the data from two table tennis players was corrupted and could not be used). Although the stimulus in this study was presented in a random position with regard to position from the midline, there was a consistent pattern in that the stimulus was always presented in a horizontal position, always crossed the midline, and was between 5° and 10° from the previous stimulus. This pattern was deliberately chosen, not only to limit variables, but to simulate, to a certain extent, the pattern that exists during table tennis. Therefore the subjects were able to make certain predictions before the onset of the saccade, that is, the better subjects were able to make predictive saccades which were affected by cognitive as well as neuromuscular factors. Kowler discusses the evidence that saccadic programs can be prepared, at least in part, before the location of targets is fully discerned.[7] Fischer[6] mentions that anticipatory saccades can have a latency of less than 80 msecs. This form of saccade is when the motor reaction (the refixation saccade) is initiated before the sensory event (the displacement or appearance of the visual target). It is likely that the movements of the table tennis players are examples of such anticipatory saccades.

If a saccade to a random position is predicted, it is likely that it will not be precise, and that additional correcting saccades would need to be made, and this was seen to be the case. One might therefore question the value of such anticipated saccades, however, the corrections were usually very small, and allowed time for another anticipated saccade to be made before the next stimulus appeared. It could be that a final small corrective movement was more effective than a larger, precise saccade made after the stimulus appeared. It is also possible that the saccade was not made to foveate the target (the ball is moving so quickly in table tennis that this would be impossible), but rather to move its image close the the fovea in order to make correct judgements regarding its position.

Corrective movements were not usually made when the saccade was not anticipated. In most cases they were remarkably accurate showing little evidence of over or undershoots.

The ability of some players to successfully anticipate stimulus movements has two implications for testing the research hypothesis. One is that the large variations in latencies between players means large numbers of subjects would have to be tested before statistical analysis would detect a significant difference between players and non-players, even if one exists.

The second implication is that one of the assumptions made in designing the experiment may well have been wrong. It was assumed that all competition-level players would be of approximately equivalent ability. This may not have been the case. Future research should use larger numbers of subjects, and should compare non-players to player of superior ability, as defined by criteria other than their saccadic speed.

CONCLUSION

It appears that 40% of the table tennis players when presented with a stimulus moving at 0.5 secs, and 30% when the stimulus is presented at 0.3 secs are better at predicting the position of a saccadic stimulus than their team mates and the non-table tennis players. Further research will be aimed at determining whether these skills, and other aspects of saccadic eye movements, can be improved with training and ultimately, whether such training can effect sporting performance.

ACKNOWLEDGMENTS

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CASE REPORTS

SUPERIOR OBLIQUE MYOKYMIA - A CASE STUDY

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Abstract

Superior Oblique Myokymia is a rare spasmodic contraction of the superior oblique muscle giving rise to monocular vertical and rotary microtremor and accompanied by torsional shimmering oscillopsia. A fifty-five year old woman presented with a long history of right ocular discomfort and a lack of synchronisation between the eye movements of the two eyes without diplopia. This was diagnosed as being due to right superior oblique myokymia. The unusual clinical presentation of this condition is discussed and treatment options outlined.

Key words: *vertical and rotary microtremor, oscillopsia.*

INTRODUCTION

Superior oblique myokymia is an unusual disorder of ocular motility[1]. It was first described by Duane in 1906 (cited in Walker, 1985) as a *unilateral rotary nystagmus*[2], and has also been described by Hoyt and Keane[3] as *intermittent uniocular microtremor*. It is characterised by recurrent episodes of monocular vertical and rotary microtremor, and accompanied by torsional shimmering oscillopsia. The affected eye intorts for seconds during each episode[4]. In some cases the patient is able to define the precise ocular movement which precipitates the attack[2], however for many patients episodes may occur without a change in ocular position[4].

Symptoms may vary between patients, and visual symptoms are often accompanied by physical ocular sensations. The visual symptoms can consist of intermittent oscillopsia or intermittent torsional or vertical diplopia[5]. Patients may also describe ocular sensations of twitching, jumping or moving, and a pulling or drawing sensation may accompany or precede an attack[5].

This condition has no known associations with other neurologic disease. Disordered activation and/or inhibition at the level of the 4th cranial nerve nucleus are proposed as the mechanisms of production of this unique ocular condition[4]. Hoyt and Keane[3] have suggested that this distinctive neuro-ophthalmic condition represents a self limiting motor neuron disorder.

CASE STUDY

A 55 year old woman, J.F., presented with a history of right ocular discomfort which she had experienced for many years. She stated that on an intermittent basis the movements of the right eye did not synchronise with those of the left eye. On further questioning regarding other symptoms such as diplopia, she stated that she experienced blurred vision like a heat haze during the episodes but not diplopia.

J.F. felt that the condition was aggravated by

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stress, driving and bright lights, and alleviated by changing head posture, particularly by adopting a chin down head posture. During history taking she commented that she sometimes thought she was going mad or imagining the symptoms, and felt very frustrated by the fact that previous eye tests had failed to disclose the nature of her problem.

On examination, visual acuity was 6/5, N5 right and left with correction. The Lang's test demonstrated stereopsis to 200 seconds of arc (with and without use of a chin down head posture). On cover testing at 6 meters J.F. was orthophoric initially, but then an intermittent microtorsional tremor of the right eye was noted. At 1/3 metre she had an exophoria initially, with an intermittent microtorsional tremor demonstrable after a short time of cover testing. Each episode of the tremor lasted only a few seconds and J.F. was able to report when it was about to happen because the right eye 'felt different'.

On testing ocular movements J.F. was found to have a downshoot of the right eye on laevoversion followed a few seconds later by a recovering updrifting movement. These unusual eye movements did not occur on every occasion of laevoversion but when they did occur they were quite obvious. Overaction of the right superior oblique and underaction of the left inferior rectus could only be demonstrated occasionally. On dextroversion and dextroelevation there was a definite torsional movement of both eyes (right more than left). Convergence was full with no microtremor noticeable during testing of this function.

J.F. was diagnosed as having right superior oblique myokymia and when this was discussed with her she was very pleased that her unusual symptoms were actually due to a clinical condition rather than to her imagination. She was commenced on Carbamazepine (Tegretol) 100mg three times a day, to be reduced to twice daily over a three week period. This provided immediate relief from her symptoms and she has continued with this regime for the past 12 months without the need for any increase in strength or frequency of the prescribed drug.

DISCUSSION

Superior oblique myokymia has such distinctive signs and symptoms that the diagnosis can be made without subjecting the patient to a whole battery of unnecessary neurodiagnostic tests[4]. Slit lamp examination is sometimes needed to see the fine tremorous ocular movements of this condition[1], otherwise the patient may be diagnosed incorrectly as having a functional problem.

Management of this condition is by drug therapy or surgery. Carbamazepine is usually the drug of choice[1], and it provides dramatic relief of symptoms for most patients. Some patients remain symptom free following its discontinuation but others require a continuing low dosage to remain symptom free[2]. However it is not suitable for all patients because for some it has no effect and for others may cause side-effects. In these cases surgery may be considered[1]. Intrasheath tenotomy of the superior oblique muscle and recession of the insertion of the inferior oblique of the involved eye[3] has proved useful in those cases which are unresponsive to drug therapy.

CONCLUSION

Orthoptists need to be aware that while rare, superior oblique myokymia does exist, and may pass unrecognised unless credence is given to the patient's symptoms and sufficient time is spent during the ocular examination to allow the condition to manifest itself. It is important for the orthoptist to reassure these patients that their unusual symptoms are due to a condition which is benign and self limiting.

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A CASE STUDY - UNUSUAL FEATURE OF CHRONIC PROGRESSIVE EXTERNAL OPHTHALMOPLEGIA (CPEO)

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Abstract

A sixty-seven year old man presented with bilateral marked ptosis, gross restriction in elevation, reduced lateral gaze and mild restriction in depression. These ocular movements were associated with an unusual feature of wriggling of the ears on attempted laevo and dextro depression.
Extensive investigation of this patient found mitrochondrial DNA deletion which is associated with ophthalmoplegia, thus the diagnosis of CPEO.
The type of innervational problem seen here may be associated with similar innervational characteristics that occur in cases such as Duane's Syndrome, Marcus Gunn Jaw Winking Syndrome, third nerve palsies and in a case of congenital fibrosis syndrome.
Key Words: *chronic progressive external ophthalmoplegia (CPEO), mitrochondrial DNA, ocular myopathy, wriggling ears.*

CASE STUDY

A 67 year old Indian man presented to clinic complaining of reduced visual acuity in his right eye over the previous twelve months and 'droopy' eyelids. His general health was good and he was not on any medication. He stated there was no family history of eye problems, however both sons had bilateral ptosis one more severely affected than the other (this was noted when the sons presented with their father at an appointment).

The diagnosis of right cataract was made and he underwent a right extracapsular cataract extraction with intraocular lens (IOL) insertion later that year. No comments were made concerning his droopy eyelids.

A year later, he presented to clinic concerned about his 'droopy' eyelids. History taking revealed the following:-

- the ptosis had been present for more than 30 years.
- he had seen a specialist in Bombay who had advised him that his condition was probably congenital.
- he had become aware of the bilateral ptosis during the last 4-5 years.
- he stated that the eyelids often felt heavy and had to be held up.
- the ptosis was worse in the evenings and when tired.
- he was not aware of diplopia, but was aware of impairment of gaze in both the vertical

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and horizontal direction, moreso in elevation.

The treatment options for his ptosis problems were discussed with him. These included: - ptosis sling that attach to glasses frame or surgery involving leavator excision with a bilateral frontalis suspension[4]. Given these options he was happy to continue as he was.

Orthoptic assessment revealed the following:-

Visual Acuity - right & left 6/9

Abnormal head posture - chin up, tilt to right, slight turn to left (see figure 1)

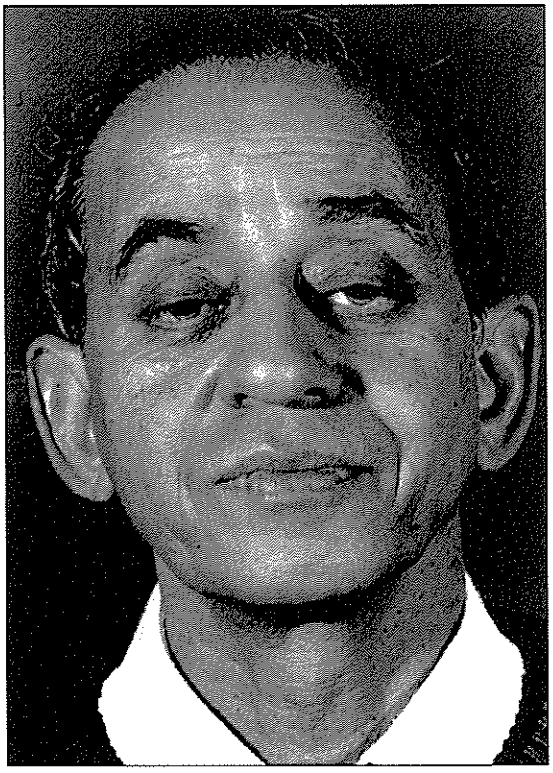

Figure 1.

Bilateral ptosis - poor to fair levator function
R10mm L11mm

Cover test - small left divergent squint with right hypertropia far greater than near

Prism cover test - FR 6-12Δ exo, R/L 4Δ

Visual fields - full to confrontation

Fundi - discs & maculae NAD

Pupillary responses - normal

Ocular movements

- marked restriction in elevation both eyes (no vertical upgaze)

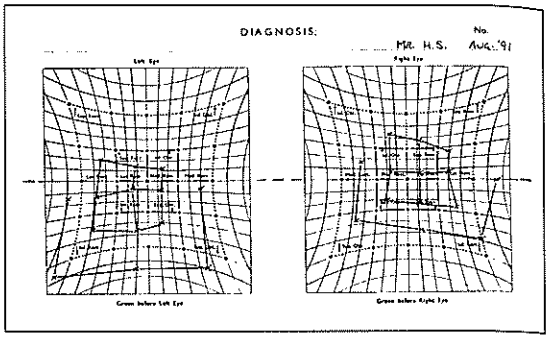

Figure 2.

- reduced lateral gaze R & L
- mild restriction in depression

A Hess chart was plotted (Figure 2.) This showed a restriction in elevation, an exo-deviation and a small right over left deviation.

The above ocular movements were associated with an unusual feature that involved wriggling of the right and left ears on movement of the eyes in dextro-depression and laevo-depression. The ears moved forward and backwards.

It was following these findings that he was referred to the medical clinic for further investigation.

Neurological testing was unremarkable. He was found to be alert and well oriented. All modalities of sensation, motor system and coordination testing were normal. Reflexes and ECG were also normal. An impression of ocular myopathy was the diagnosis made.

Several months later he returned to clinic complaining of horizontal and vertical diplopia. Orthoptic assessment was as the previous examination. The diplopia was overcome by incorporating a 2Δ base in prism in the left lens and a 3Δ base down prism in the right lens of his current glasses.

Following this visit, Mr. H.S. had been symptom-free for up to twelve months. Shortly after this time he presented to the Neurology clinic complaining of 'no energy', tiring very quickly and continual eye muscle weakness. Further extensive investigations were undertaken. Using Polymerase Chain Reaction (PCR) techniques, a molecular defect in mitrochondrial DNA was found and the diagnosis of Chronic Progressive External Ophthalmoplegia (CPEO) was made.

He was commenced on co-enzyme tablets to help his energy problem, but on last follow up was having side effects.

DISCUSSION

CPEO is a slowly progressive muscle disease with onset early in life and characterised by ocular myopathy. It is characterised initially by ptosis and is followed by progressive paralysis of all extraocular muscles. There is no involvement of the intraocular muscles[1].

In the case of Mr. H. S., not only were all these features present, but the additional unusual feature of wriggling ears on eye movements added interest. This unusual feature may be consistent with the innervational type characteristics seen in other ocular syndromes such as Duane's Syndrome[2,3,6,8]. Marcus Gunn Jaw Winking Syndrome[2,4,5,6,9]. Third nerve palsies[2,6,9], and Congenital Fibrosis Syndrome[4,6].

The most common aetiological factors responsible for many of these conditions is anomalous innervation as a result of any of or a combination of the following:

- miswiring of nerve fibres, at the level of the brainstem, during embryonic development[5,7].
- anatomical abnormalities which are either congenital, that is absent nuclei or reduced innervation[2,3,6] or acquired following lesions, injuries or other pathology that cause regeneration of nerve fibres responsible for peripheral anomalous innervation[2,5,6,9].
- normal nerves have dual innervation[2,4,6].

Many of these findings have been confirmed by autopsy examination[3], or by electromyographical studies[2,4,6].

The most likely explanation for the wriggling ears is neural misdirection as a result of abnormal neural sprouting secondary to the neuropathology of the CPEO. This may affect the pathways controlling the eye movements and ears. With the progressive nature of this disease the paralysis of the muscles means a possible loss of innervation allowing for peripheral aberrant innervation to take place. Another possible explanation is that the muscle controlling movement of the ears is the auricularis posterior muscle controlled by the facial (VIIth) nerve. During embryonic development neurones making up the VIIth nucleus and VIth necleus develop together and are in close proximity to each other (Figure 3). It is therefore possible that connections at this level persist and would at least explain the activity of the auricularis posterior muscle and activity of the lateral rectus muscle occurring simultaneously[7]. Lateral gaze and depression were least affected. Absence of innervation in elevation may have lead to nerves regenerating and activating other nerves, for example the ear.

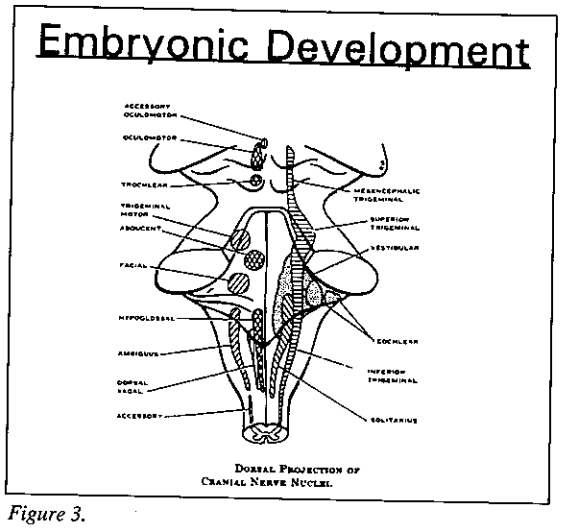

Figure 3.

CONCLUSION

The recent testing procedure of PCR testing to screen blood samples has been very advantageous in the diagnosis of many diseases. Early diagnosis of CPEO is most important prior to commencement of any form of treatment because of the progressive nature of the disease.

In this patient the wriggling ears was probably an acquired feature secondary to CPEO.

We, as clinicians, when confronted with neuromuscular diseases like this case should in future, not only observe ocular movements but possibly ear movements as well.

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A CASE STUDY IN CORTICAL PLASTICITY

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Abstract

Much research has gone into establishing the critical or sensitive period for the development of binocular vision in humans. An understanding of this and the plasticity of the visual system has had implications for the treatment of amblyopia in children. Occlusion therapy is thought to be effective until approximately eight years of age and most effective below the age of four years.
This paper presents a case study of an eleven year old boy who is diagnosed with cortical visual impairment, cerebral palsy and epilepsy. At six months of age no visual responses were evident on both observation and VER testing. At eleven months, responses to light were noted and vision continued to slowly develop. Part-time occlusion to decrease right amblyopia began at the age of four years and eight months. Greatest improvement was noted between the ages of nine and ten years when vision improved to approximately 6/45, equalling the vision in the left eye.
Literature in the 80's, describing experiments on cats, found a lengthening of the "critical period" following an early period of no visual attention. The hypothesis is made that in cases of delayed visual development in children there is the possibility of a lengthening of the sensitive period and thus response time to occlusion therapy.
Key Words: *critical/sensitive period, vision, amblyopia, occlusion, development, cerebral palsy.*

INTRODUCTION

The critical or sensitive period, as described by Von Noorden[1] is a time span known to exist in humans and experimental animals during which certain visual functions are modifiable by decreased or abnormal stimulation.

Foundational research into the critical period was carried out by Hubel and Weisel in the 1960's. By studying the receptive fields in the visual cortex of the cat they discovered that by altering the binocular visual experience of kittens through monocular deprivation and artificially induced strabismus, that marked changes in the way in which the visual cortical cells processed information occurred[2,3]. In studies comparing monocular lid suturing at different ages, in the newborn and older cat, they established the critical period which extends from 3 weeks to approximately 3 months of age[4] (see Figure 1).

In similar experiments, a critical period for Macaque monkeys was also discovered. The Macaque monkey is most sensitive to monocular deprivation during the first 6 weeks of life,

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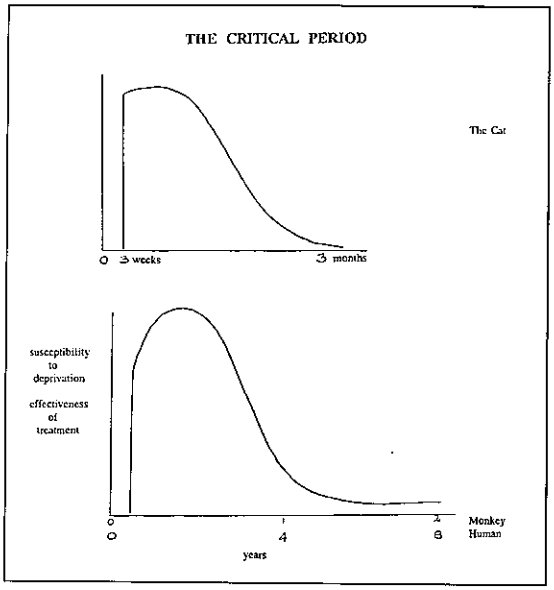

Figure 1.

sensitivity then declines progressively to 1½ to 2 years when the monkey loses this type of cortical plasticity[5].

The development of the visual system in humans is thought to be approximately four times longer than in monkeys, therefore in humans the critical period is thought to extend from birth until approximately 6-8 years, however a precise age has not been demonstrated[1].

An understanding of the 'critical or sensitive' period and the plasticity of the visual system has had marked implications for the treatment of amblyopia in children. It has long been recognised, in fact since 1742, that by patching the 'good eye' one can improve the vision in an amblyopic eye[6]. Amblyopia therapy today takes on many forms, from occlusion of the better eye, to penalisation using lenses and the use of cycloplegic drugs, however the general principle remains the same. It is experimental research that has given us an understanding of the mechanisms behind amblyopia and the cortical changes that take place, but more than that, research into the critical period has also given us a time frame within which to work.

Recent clinical studies based on the age of onset of the disruption to binocular vision have estimated the sensitive period for different

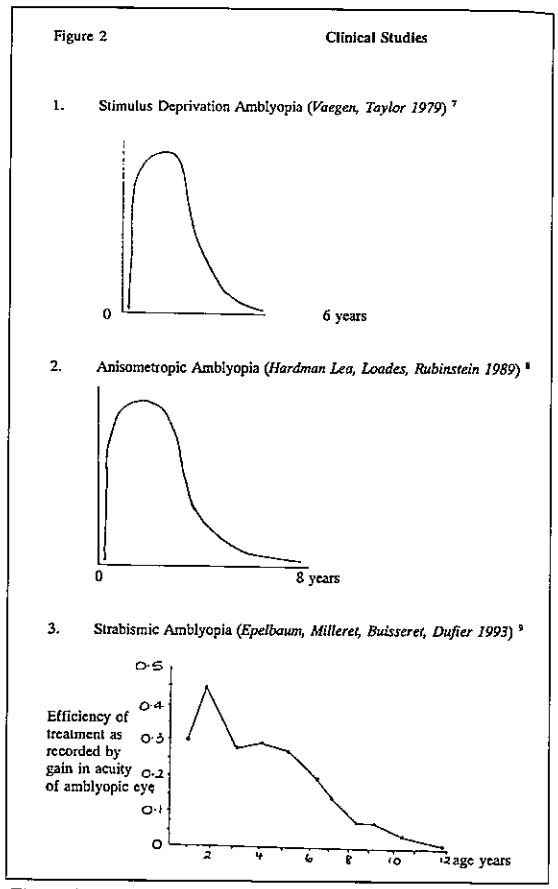

Figure 2.

types of amblyopia[7-9]. For example the sensitive period for stimulus deprivation amblyopia is thought to last to approximately 6 years[7] and anisometropic amblyopia to 8 years[8]. Another study published earlier this year, by Epelbaum, Milleret, Buisseret and Duffier, looked at the sensitive period for strabismic amblyopia[9] (see figure 2). In this paper however, the authors chose to refer to the age of initiation of occlusion therapy rather than try and estimate the age of onset of strabismus. Epelbaum et al discovered, using a population of 407 children with strabismic amblyopia, ranging in age from 21 months to 12 years, that occlusion therapy was most effective between 2 and 3 years of age followed by a progressive decrease in efficiency to 11-12 years of age where therapy was no longer effective. The following case is presented in light of these findings.

CASE STUDY

J. was born at 33 weeks gestation via caesarean section as his mother had suffered chronic renal failure. His birth weight was 1660 grams. J.'s motor development was queried at one month of age and subsequent ongoing investigations led to the diagnosis of cerebral palsy and epilepsy at 9 months.

J.'s visual development was first queried at the age of 6 months, and an ophthalmological assessment at that age found no visual responses. Eyes and fundi were found to be normal. A VER performed at that time failed to elicit a response. A diagnosis of cortical blindness was subsequently made.

Follow up over the next 4 years revealed a gradual development of vision with J. responding to light at 11 months followed by gross movements at 16 months. At 2 years and 8 months J. would reach towards toys held 20cms from his eyes and occasionally roll towards large, brightly coloured toys up to 1m away. By the age of 3 years 4 months it was possible to measure his vision using stycar balls. An acuity of 1.5/60 was achieved, which improved to 6/90 by the age of 4 years 8 months. Part-time left occlusion was commenced at this stage due to J.'s objection to having the left eye covered and his apparent inability to take up right fixation. Occlusion then continued for the next 6 years.

Vision in the left eye continued to improve reaching 6/45 at the age of 5 years 8 months. A gradual improvement of the right vision was observed with the most significant improvement being apparent from the age of 8 years. (See Table 1).

Occlusion was commenced using a face patch. Part-time occlusion was favoured over full time due to J.'s multiple disabilities, extremely poor right vision and resultant limited attention. No improvement was apparent in the first 12 months. The type of occlusion was consequently changed to plano lenses with a translucent occluder to eliminate the effect of intermittent latent nystagmus. Over the next 12 months a slight improvement was documented with J. being able to detect hand movements. This improvement continued with a Teller Acuity Card reading of 1/60 being obtained at the age of 8 years. From the age of 8, visual improvement was increasingly evident and J.'s ability to respond to the Teller Acuity Cards improved thus making test results more reliable. The resumption of a face patch at 8½ years due to J. persistently removing the plano lenses further enhanced the visual improvement with a result of 6/45 being obtained at the age of 10 years. Occlusion continued with equal vision in each eye being maintained over the next 12 months. The fluctuation of acuities from 6/45 to 6/60 in either eye are more a reflection of J.'s co-operation during testing than actual visual fluctuations.

Table 1
Pattern of Visual Improvement

Age	Right Eye	Both Eyes Open	Left Eye
11 months		response to light	
16 months		gross movement	
2.08		large objects	
3.04		1.5/60	
4.08		6/90	
5		left occlusion commenced	
6	?light perception		6/45
7	hand movements		3/60
8	1/60		6/24
8.07	5/60		-
9.03	5/60		6/60
9.05	6/60		6/45
10	6/45		6/45
11.02	6/60		6/60

DISCUSSION

Having reviewed J.'s early history and subsequent development it is thought that Delayed Visual Maturation (DVM) is a more appropriate term than the previous diagnosis of cortical

Table 2
Delayed Visual Maturation - classification

Group 1
- I DVM only
  - (i) poor vision on presentation
  - (ii) history of poor vision, improved prior to examination
- II DVM with systemic illness/perinatal problems

Group 2
- DVM with intellectual impairment

Group 3
- DVM with nystagmus/ocular abnormality

Fielder, Russell-Eggitt, Dodd & Mellor 1985

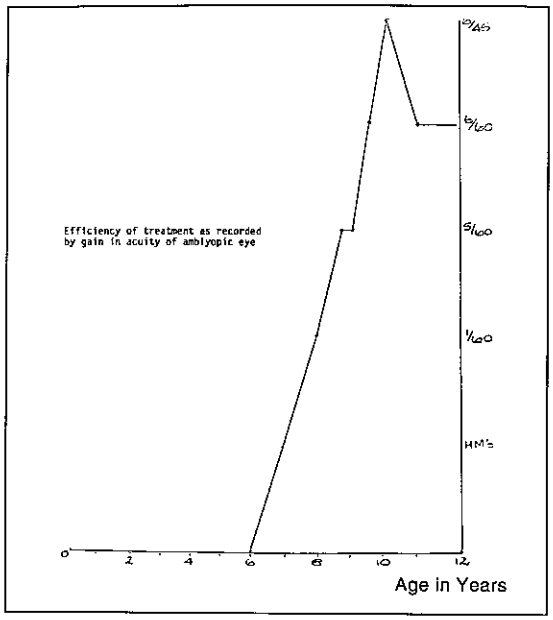

Figure 3. Recorded visual improvement by J.

blindness. Fielder, Russell-Eggitt, Dodd and Mellor reported on three groups of infants with DVM[10] (see Table 2). J.'s visual development is similar to those in group 2 of this classification which consists of children with DVM and a significant degree of mental retardation. These children characteristically have severely reduced visual interest, however, pupil responses and fundal examination are normal. Divergent strabismus is common. The manner in which visual improvement occurs in group 2 infants is characteristically very slow and it can often take many months to decide whether any visual change has taken place. The recovery period has been shown to extend from approximately 6 months to 2 years of age. Mellor and Fielder had also stated in a previous paper that where visual improvement has occurred the possibility of later perceptual problems can not be eliminated[11].

In cases of normal visual development, visual sensitivity is almost complete at 6 months[12] with the critical period for the treatment of strabismic amblyopia extending to approximately 11 years of age[9]. As previously mentioned, occlusion therapy for this type of amblyopia is most effective between 2 and 3 years of age. But what of children with DVM?

J.'s vision did not appear to begin to develop until he was 11 months of age. Development of vision was slow with the maximum binocular visual acuity being recorded at approximately 6 years of age. Occlusion therapy for J. was most effective between 8 and 10 years of age (see figure 3).

During the course of this study the authors were unable to find any clinical studies that looked at extended critical periods in children. However, there is some experimental research which may shed some light on the subject, particularly one study by Cynader and Mitchell[13] that looked at the effect of dark rearing on the visual plasticity of the cat. They found that by rearing cats in the dark they were able to prolong the critical period. They compared the results of monocular lid suturing in dark reared cats with normally reared cats of different ages and found that the time course of deprivation effects in 4 month old dark reared cats was equivalent to that observed in normal kittens who were sutured at 5-6 weeks of age. Cynader and Mitchell concluded that the cortical age of the dark reared cats may be only 5-6 weeks.

Previous studies of social imprinting in birds[14] came to a similar conclusion that restricted exposure leads to responses in older animals which would normally occur only during clearly defined critical periods early in life.

It is possible that there may be a similar lengthening of the sensitive period and the response time to occlusion therapy in cases of DVM in children. There are of course other factors which may contribute to observed visual improvement. Increasing levels of maturity and cognition, for example, may demonstrate an improved ability to co-operate with formal testing procedures and thus show an apparent improvement in visual acuity results. It must be noted however, that in J.'s case although these factors may have contributed to his overall visual improvement there was also notable monocular improvement coinciding with persistent occlusion therapy.

CONCLUSION

The implications of this paper are that children with a diagnosis of delayed visual maturation or perhaps a general developmental delay may have an extended critical period. It is therfore the responsibility of the Orthoptist to consider continuing to implement occlusion therapy beyond defined critical periods.

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DYSTHYROID EYE DISEASE FROM THE INSIDE LOOKING OUT.

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Abstract

The actual incidence of dysthyroid eye disease or thyroid ophthalmopathy in thyroid dysfunction is probably not known, as not all patients with thyroid disease are referred to endocrinologists or other hospital physicians, but are managed by their General Practitioner. Also endocrinologists tend to apply variable criteria for referral to an ophthalmologist. Some only consider ophthalmic opinion when there is obvious thyroid ophthalmopathy with serious visual disturbance, diplopia in the primary position or marked exophthalmos, and others will request ophthalmic advice when there is only minimal evidence of dysthyroid eye disease. It is possible therefore that some patients with thyroid eye disease may remain undiagnosed and untreated.

This paper discusses the author's own experience of thyrotoxicosis and thyroid ophthalmopathy particularly with regard to the understanding of the general disease process, the cause of some of the signs such as excessive blinking, lacrimation and adoption of abnormal head posture; and the importance of the right psychological approach to the management of some of the more unsightly cosmetic problems.

INTRODUCTION

Thyroid ophthalmopathy is a well known condition, characterised by exophthalmos, lid lag and lid retraction, peri-orbital and conjunctival oedema and mechanical restriction of eye movements. A characteristic sign is the abnormal head posture, usually a raised chin. The patient frequently complains of excessive lacrimation, red eyes and painful, uncomfortable eye movements. There is frequently diplopia, and visual disturbance which in some cases may lead to irretrievable visual loss due to optic nerve damage from compression by the muscles, or from raised intra-ocular pressure. The condition may be unilateral or bilateral, symmetric or asymmetric. Dysthyroid eye disease can occur with hyper or hypo thyroidism, and may present in patients with no sign of thyroid abnormality. It may occur at any time after the onset of the disease, and often presents when the patient is in an euthyroid state. Recurrence is said to be rare, but it is easy to find evidence of the disease several years after the active phase has passed, and the symptoms have resolved, due to the fibrosis of the formerly affected muscles and the tethering of one or both inferior recti.

SIGNS AND SYMPTOMS OF THYROID DYSFUNCTION

I was given a diagnosis of thyrotoxicosis, some months after I had first started to experience

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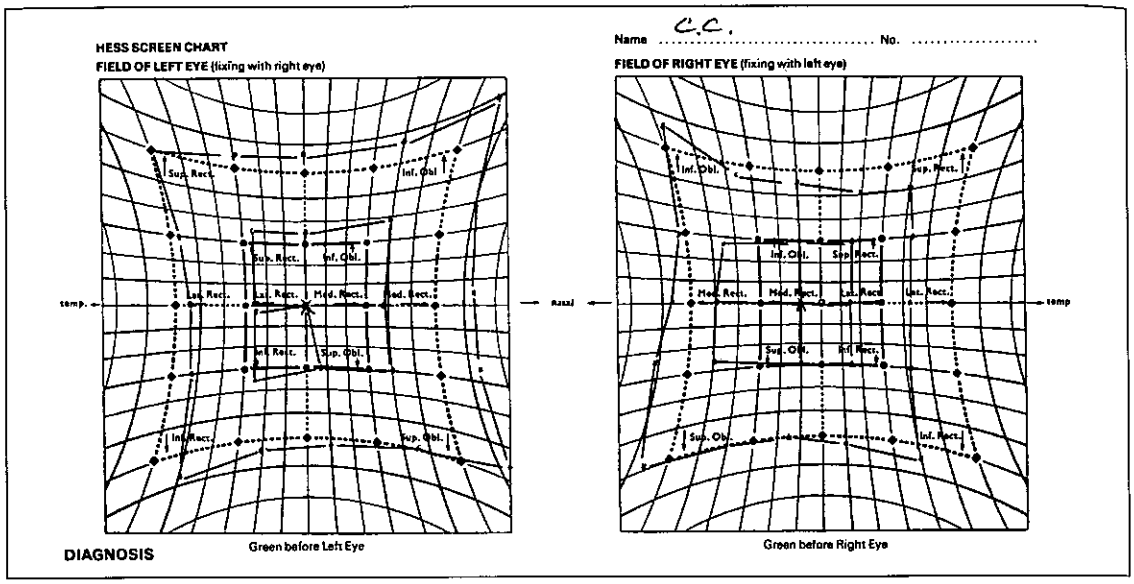

Figure 1.

anxiety attacks, hand tremors, fits of sweating, weight loss and metabolic disturbances. Whilst I do have a family history of thyroid dysfunction, I had attributed my initial symptoms to my age, stress, a recent orthopaedic operation and a possible reaction to my drug regime for rheumatoid arthritis. However, the onset of lid retraction and lid lag, and episodes of tachycardia at night convinced me that I had thyrotoxicosis. In referring me to the endocrinologist, my physician commented on the known relationship between thyroid dysfunction and rheumatoid arthritis in patients attending hospital for treatment of rheumatoid disease.

MANAGEMENT

The management of thyroid dysfunction is to normalise the levels of thyroxine in the blood, with hyperthyroidism by inhibiting thyroid function with drugs, radio-active iodine or in some cases by partial or total ablation of the gland itself, and with hypofunction by introducing thyroxine artificially. It may take time to stabilise the condition and the patient may feel unwell for some long period. I was treated with carbimazole which rapidly rendered me euthyroid, although my symptoms of hyperactivity, metabolic disturbance, overemotionalism, and sweating attacks persisted until, unfortunately, I became hypothyroid. I became depressed, lethargic, oedematous, could barely summon the effort to walk, and found concentration difficult. Happily for me this state was shortlived, but it is important to note that in both thyroid states, not only did I feel most unwell, but I was aware that I was not functioning normally either physically or psychologically. My mental state in particular did not approach normality for some time after a stable euthyroid state had been achieved. This is, I am sure, a major contributory factor to the difficulties experienced with dysthyroid patients, who are time-consuming to manage and are often labelled neurotic and unco-operative.

SIGNS AND SYMPTOMS OF DYSTHYROID EYE DISEASE

My eye disease presented initially during my hypothyroid state, some 4 months after the commencement of treatment for my thyroid disorder. I had become oedematous in appearance, and suddenly began to experience pain on eye movements and diplopia in downgaze. Recent studies[1] have suggested that there may well be an increase in cases of thyroid ophthalmopathy due to the rapidity with which patients

are rendered euthyroid with current treatment modalities, and although this might have been the cause of the onset of symptoms in my case, I have no personal clinical evidence of an increase in dysthyroid eye disease.

Because of the ocular symptoms associated with thyrotoxicosis, I found if very difficult, until I began to experience diplopia, to differentiate where the symptoms associated with the general disease stopped and where those due to thyroid ophthalmopathy started, and this dividing line is one which I suspect is not well appreciated by physicians, and is the reason that many cases with mild to moderate dysthyroid eye disease are not referred to ophthalmologists, particularly when diplopia is not present in the primary position.

I rapidly developed bilateral exophthalmos, left more than right, and retraction of the left upper lid, which was accompanied by acute discomfort on pursuit movements which worsened on saccades. My peri-orbital oedema increased, combined with conjunctival oedema and my eyes became red due to the dilated ciliary vessels over the insertion of the rectus muscles. My intra-ocular pressure on upgaze was raised to 27mmHg. Subjectively, there appeared to be fairly symmetric restriction of upgaze, with right hypotropia on dextro-elevation and left hypotropia on laevo-elevation, but the Hess chart (Fig. 1) showed that there was greater restriction of the right eye on up gaze. I had a right esotropia with diplopia on right gaze. There was an elevation of the chin as a compensatory head posture. The diplopia was only troublesome when I was driving a car, as the restriction of upgaze with diplopia made using the overhead mirror difficult, and the presence of right esotropia on right gaze made joining a motorway somewhat hazardous! However, provided I continued with my compensatory head posture, or turned my head to look to the right I remained relatively asymptomatic from the motility point of view.

MANAGEMENT

The management of dysthyroid eye disease is clearly defined. There is no surgical intervention unless the sight is at risk, until the patient is euthyroid and any muscle imbalance has been stable over a three month period. In the short term Fresnel prisms and botulinum toxin therapy may help overcome diplopia in the primary position and increase the field of binocular single vision, and may even be all that is necessary in less severe cases. I was managing without prisms, but I did consider botulinum injections for my inferior recti in view of the possibility that it might inhibit the amount of fibrosis occurring at a later stage in the disease process.[2] However the risk of rendering my fairly symmetric condition asymmetric with botulinum injection was considered to be too high. Later, the response to the toxin injections was questioned.[3]

Whilst I was surprised at the speed at which I developed a compensatory head posture, and the manner in which my vertical fusional range increased, I was having some difficulty in coping with the other ocular symptoms, in particular the change in my appearance, mainly caused by the exophthalmos, peri-orbital oedema and lid retraction. Rapid eye movements were uncomfortable and produced excessive lacrimation, which in turn increased the blink rate. Blinking, unfortunately, did not stimulate my binocularity, but rather acted as a dissociating mechanism! I had red eyes and conjunctival oedema and a recurrent corneal erosion caused by my loss of Bell's phenomenon. Artificial tears helped the discomfort, but sleeping sitting up, using cold compresses or taking diuretics all failed to have any effect on the peri-orbital oedema. Guanethidine drops and lid exercises made absolutely no difference to the lid retraction. I felt miserable and realised why so many dysthryroid patients are unhappy and depressed and why some will consult plastic surgeons in an effort to improve the cosmesis. Once the more acute symptoms such as diplopia and loss of sight have been addressed, the patient has more time to consider their appearance. They want to feel well and look normal again. The clinician should be aware of this and once the patient is euthyroid, every effort should be made to explore the possibili-

ties of minor plastic surgery to the lids and periorbital tissue to improve the cosmetic appearance. To say, as some ophthalmologists do, that cosmetic surgery should not be undertaken for any reason shows a lack of understanding of, and sympathy for the patient. In my case, a Henderson procedure to the left upper lid normalised by appearance and reduced almost all my other ocular symptoms to within tolerance levels.

With time, the active disease phase passes and the oedema regresses, so that the appearance normalises although the mechanical restriction of eye movements persists. The patient however must be convinced that this will occur and it has been very helpful to be able to assure my patients that relative normality can be restored. They only have to look at their orthoptist!

COMMENT

One of my concerns has been that there are many patients with dysthyroid eye disease who do not have access to ophthalmic treatment, because their physicians are unaware of the necessity for the early differential diagnosis between dysthyroid eye disease and the ocular signs of thyroid dysfunction. Whilst patients with severe thyroid ophthalmopathy will always be referred, there is evidence that many less serious cases may never have reached the ophthalmic clinic, and may have forced to endure a considerable period of discomfort, and may even have suffered diplopia and irretrievable visual damage. It is imperative that patients with thyroid dysfunction with any signs or symptoms of dysthyroid eye disease are referred for management to the ophthalmology team, as this can only improve the management of the condition and early referral may result in earlier alleviation of symptoms, increased support of the patient and improved understanding of the ocular condition.

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LETTERS TO THE EDITOR

CONTINUING INTRIGUE WITH BROWN'S SYNDROME AND IT'S ASSOCIATION WITH ESOTROPIA

In 1992, we reported on a patient who had congenital Brown's syndrome in association with accommodative esotropia.[1] Although Brown's syndrome is recognised to be mostly associated with normal binocularity and absence of heterotropia in the primary position,[2] accompanying horizontal strabismus, particularly esotropia, has been described by numerous authors over the past 40 years. We apologise for omitting Clarke and Noel[3] and Bourne[4] from our 1992 literature review. Clarke and Noel reported horizontal strabismus and amblyopia in 8 out of 28 Brown's syndrome patients. Bourne, in a series of 20 patients, found 2 had constant esotropia with amblyopia, and 4 had intermittent esotropia in forced primary gaze. Further, Gregerson and Rindziunski[5] have since reported on 10 Brown's syndrome patients who were followed for between 7 and 19 years, 3 of whom had co-existing constant or intermittent esotropia and 1 who had exotropia. In this communication, we wish to update the 'case-law' and report on a further three patients who have congenital Brown's syndrome associated with either accommodative or non-accommodative estropia. Are these coincidental or does the ocular motility deficit seen in Brown's syndrome potentiate an *esotropic force*, either sensory and/or motor?

Case 1. Master R.M., aged 8 years, was diagnosed with bilateral Brown's syndrome of moderate severity (grading of severity based on features described by Eustis, O'Reilly and Crawford, 1987[6]) and an accommodative esotropia that was reduced to microtropia with +4.00/+1.00 correction OU. There was good fusion and stereopsis to 100" (Titmus®), despite the presence of a moderate degree of amblyopia, 6/6 and 6/15.

Case 2. Miss K.K., aged 5 years, presented with severe bilateral Brown's syndrome and large-angle esotropia. She required anisometropic correction of +0.50DS OD and +3.00DS OS. There was no binocular vision and a high degree of amblyopia, 6/10 and 6/30. The ocular rotations were restricted such that on cover testing there was esotropia and hypotropia of each eye under cover. Following some cycles of right occlusion, surgery was undertaken to correct the Brown's sydrome (superior oblique tenotomies) and the esotropia (bimedial rectus recessions). A severe Brown's syndrome on the right side persisted post-operatively, however.

Case 3. Master C.C., aged 8 years, presented with moderate bilateral Brown's syndrome leading to a chin-up compensatory head posture. There was a small-angle intermittent esotropia, tenuously controlled in the primary position and breaking to esotropia with diplopia on slightest chin depression. Correction of the minimal refractive error, +0.25/+0.25 OD and +0.25/+0.50 OS, made no difference to the control. Random-dot stereopsis could be demonstrated during periods of fusion. In view of the problematic head posture, bilateral superior oblique tendon spacer surgery was performed which successfully cured the Brown's syndrome, but not surprisingly caused a troublesome V-pattern and increased esodeviation that will require further operation.

The three patients described all had bilateral Brown's syndrome associated with acquired esotropia. R.M. had a microtropia with fully accommodative esotropia characteristics, K.K. had a non-accommodative constant esotropia, and C.C. had a non-accommodative intermittent esotropia. Forceps duction testing confirmed mechanical restriction in K.K. and C.C. at the time of operation.

We continue to be intrigued by the mechanism that caused esotropia to develop in these patients. It is recognised that mechanical and structural anomalies of the extraocular muscles like Brown's syndrome may be ætiological factors in strabismus.[7] But is this motility deficit alone enough to compel a convergent ocular posture? Since Parks' 1977 report that the superior oblique tendon does not have an anterior sheath, it is generally accepted that the cause of congenital Brown's syndrome in at least the majority of cases lies in the superior oblique tendon itself and/or the trochlea.[8] The mechanical restriction of elevation in adduction could result from a tight or inelastic superior oblique muscle or tendon, or an intrinsic anomaly of the trochlea complex whereby the telescopic movement of the tendon through the pulley during relaxation of the muscle is interfered with. Whatever the exact mechanism, the following occurs in Brown's syndrome. From the primary position, adduction of the globe along the midline plane of movement is not allowed because of the increased linear distance from the trochlea to the insertion of the superior oblique.[9] Rather, adduction is accompanied by a gradual infraduction, thus the characteristic downshoot that is usually seen in Brown's syndrome. Abduction, on the other hand, is not disturbed. It is also unlikely that the inferior oblique's secondary action of abduction could be inhibited when the eye is in the primary position. Therefore, there is no obvious direct mechanical or anatomical esotropic force in Brown's syndrome. Moreover, co-existing exotropia has also been reported in the literature[3,5,6].

The interruption of adduction may have significant sensory consequences however, especially if the condition is bilateral. [3] Vertical incomitant disparity would have adverse effects on the retinal correspondence. Nasal hemiretinal suppression is the most likely antidiplopic mechanism to prevail, certainly in congenital Brown's syndrome. This alone could potentiate an esotropia given the abundance of convergence tonus in early childhood. Its combined presence with hypermetropia of moderate degree, as in Case 3, would have similar effects in producing esotropia.

Having presented these three further cases of congenital Brown's syndrome with acquired esotropia, we wish to reiterate that it is not Brown's syndrome *per se* that induces esotropia, but the consequences of the motor anomaly on the binocular visual system that lead to its suppression and the arrest of fusion, and ultimately, strabismus. Stressing the significance of sensory anomalies in the ætiology of strabismus.

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TESTING STEREOPSIS - WHICH TEST SHOULD BE USED?

Stereopsis has been defined as the binocular appreciation of depth made possible by the fact that the two eyes view objects in the external world from slightly different vantage points[1]. More specifically it is the perception of three dimensions brought about by fusion of images received by slightly disparate points on the retinae. Stereopsis is the quality of binocular vision which enables us to perceive depth independently of monocular clues and contours. The demonstration of stereopsis is believed to be of great importance[2], for it can reveal the presence of visual dysfunctions such as refractive errors, strabismus, or amblyopia.

But what exactly are we doing when we assess stereopsis? Hinchliffe[3] has suggested that before conducting clinical assessment of stereopsis, it is necessary to consider why a stereotest is being used. She has identified the three following factors: to determine the presence of binocular single vision; to determine the stereoscopic threshold; and to monitor the consolidation of binocular single vision during treatment. Large numbers of studies have been conducted to compare how effectively existing stereotests assess stereoscopic ability and screen for visual dysfunction[3-13]. In many of these studies, however, contour stereograms such as the Titmus and Randot stereotests were compared and used interchangeably with random-dot stereograms such as the Lang, TNO and Frisby stereo tests, when clearly random-dot and non-random-dot stereotests assess different aspects of stereoscopic function.

It is the authors' opinion that too frequently random-dot and non-random-dot stereotests are used interchangeably without consideration of the essential differences which exist between the two types of tests. We believe that a stereotest should be chosen discriminately depending upon the nature of assessment and ocular status of the patient. A thorough orthoptic examination should include both types of stereotests, each being used to assess different aspects of binocular function. For example, in a screening situation the most appropriate test to use is a random-dot stereotest such as the TNO, Lang 1 or Lang 2 stereotest. It should be remembered that the TNO and Lang stereotests were specifically designed as screening tests because, in most cases, stereopsis cannot be demonstrated on these tests in the presence of manifest deviations and unequal visual acuity. The effectiveness of these tests has been investigated in a number of studies,[4,6,14-19] which confirm, in particular, that the Lang stereotests are easily administered and successfully detect the presence of manifest strabismus, microtropia in 70% of cases[19] and a significant number of individuals with anisometropic amblyopia.

In comparison, in patients with confirmed microtropia or small angle deviations with anomalous retinal correspondence, a contour stereotest is more appropriate[20], as it allows for the measurement of stereoacuity in the presence of a manifest deviation with abnormal retinal correspondence. The Titmus and Randot

stereotests appear to be better indicators of the presence of peripheral stereopsis in patients with small angle esotropia,[10] thus providing results which may confirm the presence of binocularity in patients who lack bifoveal fixation and are unable to demonstrate stereopsis when presented with random-dot stereograms. It is conceded, however, that the results of a study by Clarke and Noel[21] suggest that the Randot test may be superior to the Titmus test as monocular cues in the Titmus test may influence the results. This aspect of stereotesting requires further investigation.

This letter serves to remind orthoptists to choose the appropriate stereotest for each clinical setting. Is the stereotest being conducted in order to screen for visual defects, or is it attempting to establish whether any form of binocularity exists in patients with known binocular anomalies? In each situation, a test for stereopsis is important, but one must remember to choose the type of test appropriate for the desired investigation.

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BOOK REVIEWS

ULTRASOUND OF THE EYE AND ORBIT

Sandra Frazier Byrne and Ronald L. Green.
St Louis: Mosby, 1992. 505 pp. $195.00
ISBN 0-8016-1968-8.

In the preface to this text the authors' stated goal is to present a comprehensive overview of ultrasound as it applies to the eye and orbit. This is the first text of this sort to appear in the last decade and that alone ensures it a place as an important reference for ocular ultrasound.

The authors are the heads of the ophthalmic ultrasound units at the Bascom Palmer and Doheny Eye Institutes and have been active in the development and teaching of ophthalmic ultrasound techniques for the last fifteen years.

As both authors have been advocates of the standardised echography techniques this gives the text a more comprehensive coverage of ophthalmic diagnostic A and B scan ultrasonography than previous texts.

Chapters 2 and 7 are excellent descriptions of examination techniques and the coverage of the orbit in the second half of the book is the most extensive coverage of this subject to date. Chapter 5 gives an exhaustive evaluation of all forms of ocular melanoma that is again unparalleled in previous texts.

As a comprehensive overview of ophthalmic ultrasound a more thorough coverage of the history and development of ophthalmic ultrasound may have been expected. This could include more detailed explanation of the physics and instrumentation, especially recent research in differentiation of tissue type.

Chapter 3 provides an adequate presentation of vitreoretinal disease. Compared with the emphasis on the other areas in the text, vitreoretinal disease, as one of the most difficult diagnostic areas of ophthalmic ultrasound, could be considered to warrant a more extensive discussion.

The quality of the ultrasound illustrations is generally good, however the quality of fundus photographs prevent a good illustration of the various lesions' clinical appearance. Further illustration of the ultrasound findings using explanatory diagram may have improved the text for the ultrasound novice trying to understand the components of the A and B scan photographs.

Overall this text is an excellent overview of ophthalmic ultrasonography that provides an interesting reference for the advanced ultrasonographer as well as a good basic introduction for the novice.

Chris Prior.

ATLAS OF CLINICAL OPHTHALMOLOGY - 2nd Edition

David J Spalton, Roger A Hitchings, Paul A Hunter
Wolfe Publishing London 1994 $244.00
ISBN 0-397-44632-2

The purpose of this atlas is to provide an introduction to clinical ophthalmology with detailed color illustrations a key part of the format. It has contributions from many ophthalmologists and a neurophysiologist, all from hospitals around the UK. As the atlas is intended primarily for students of ophthalmology the text, although concise, assumes a thorough knowledge of medical terminology and unfortunately does not provide a glossary for the less knowledgeable.

The book begins with a section on methods of

ocular examination. This chapter includes the theory and the use of both common clinical tests and the latest technology including electrodiagnostic testing, medical imaging, contrast sensitivity, computerized perimetry and keratoscopy. Anatomical groupings form the basis of most of the following chapters. Each section of the eye is included as well as the orbit, eyelids and lacrimal system. The retina has 4 chapters alone. In addition to this there are chapters on glaucoma, intraocular inflammation, strabismus, neuro-ophthalmology, infections of the outer eye and allergic eye disease.

Each subject area is covered by information on normal physiology as well as a large selection of disorders. There is a moderate amount of text but the illustrations are of primary importance. These include photographs of fundi, histology, electron and light micrographs, fluoroscein angiography, composite paintings and graphs. Each photograph is accompanied by an extremely useful simplified diagram showing the relevant pathology more clearly.

The comprehensive atlas is an interesting textbook which covers most areas of ophthalmology, many in surprising detail. The illustrations are a reminder of the huge number of unpleasant eye diseases it is possible to suffer from and to see in clinical practice.

Anita Egan

OCULAR ANATOMY AND PHYSIOLOGY.

Saude, T. 1993.
Blackwell Scientific Publications Oxford. $275
ISBN 0-632-03599-4

This is a basic reference book with the emphasis on ocular anatomy. The text has been translated from Norwegian and is presented in an easy to follow readable style. The book presents a regional approach to the ocular anatomy, that is each chapter provides information relating to a region of the globe including structure, function(s), blood and nerve supply. The exceptions to this approach are the two chapters which describe blood and nerve supply to the globe in greater detail.

The following topics are included as chapters:

The gross anatomy of the orbit including osteology and the nasal sinuses. The outer coats of the eye in which the cellular structure of the cornea and tissue structure of the sclera are presented. The middle coat of the eye in which the tissue structure of the choroid, ciliary body and iris are presented and pupil responses to light are discussed. The internal ocular media in which the lens, aqueous and vitreous are discussed at a cellular and biochemical level. the internal media also includes discussion of intra ocular pressure and the process of accommodation. The chapter on the retina includes cellular anatomy of the retina and a discussion of the visual process. The visual paths provides an overview of the visual pathway from retina to visual cortex. Structures external to the eye include surface anatomy of the sorrounding features and the anatomy of the lids at the tissue level. The lacrimal apparatus provides an overview of the structures involved in tear production and drainage with detailed discussion of the tear film and the mechanism of blinking. The extrinsic ocular muscles includes discussion of the extraocular muscles at the cellular level with an overview of ocular movements and clinical testing. The chapter relating to orbital blood vessels provides a brief overview of the branches of the ophthalmic artery and venous drainage of the globe. The chapter on nerve supply to the orbit presents each of the cranial nerves related to the orbit or structures within the orbit and traces the nerve pathway from nucleus to orbit and/or globe. Ocular embryology presents a general introduction to appropriate terminology and a brief introduction to development within the globe.

Generally the chapters follow a similar format beginning with an overview of the region to be discussed. This overview is written in an easy to read style and provides the context for the more detailed material to follow.

Anatomical descriptions vary in depth with greater detail supplied on the cornea, retina, extraocular muscles and the extended chapters on the orbital blood and nerve supply. The text includes numerous diagrams which are clear and easy to follow. The diagrams are predominantly black and white sketches with colour used to highlight specific structures, four microphotographs are also included. There are eight colour photographs of external features of the globe, fundus and posterior surface of the anterior uveal tract.

Unlike other contemporary texts this book does not contain applied anatomy in the form of case studies or clinical examples. The chapters relating to the internal ocular media and extraocular muscles include descriptions of appropriate clinical tests for the measurement of intraocular pressure and ocular movements respectively. this is a text which describes structure and function leaving the reader to interpret this information clinically.

The title indicates that this book is primarily an anatomy text with secondary reference to physiology which is a reasonable summary of the content. Only physiology which is of significance in the clinical setting is described including corneal metabolism, retinal metabolism, aqueous production, tear production and a brief description of extraocular muscle metabolism.

In summary Saude's book is written in a readable style using technical language. Where a structure is named after an individual Saude tends to use the anatomical term and include the alternative name, this is useful in a clinical reference as the alternative name is often the one in common clinical use. Ocular anatomy and physiology is a text book and not intended for the lay reader. It would be a useful reference for the practicing clinician wishing to review an area of anatomy to provide background for clinical decision making. Students will find this a good basic text to be used in conjunction with more detailed specialist references.

Kerry Fitzmaurice.

MANAGEMENT OF DIFFICULT GLAUCOMA

Higginbotham, E.J. & Lee, D.A. (Editors)
Blackwell Scientific Publications Boston 1994
$275.00 ISBN 0-86542-257-5

Management of Difficult Glaucoma outlines the current advances in the research of glaucoma epidemiology and risk factors. It provides information on the basic principles of intra-ocular pressure measurement techniques, optic nerve head evaluation and visual field evaluation techniques. Recent advances in psychophysiological testing, including electrodiagnosis are outlined, as are the basic principles in medical therapy.

Treatment approaches to ocular hypertension, low-tension glaucoma and angle closure are discussed, as well as that of the more complicated forms of glaucoma. Management of factors which may complicate glaucoma, or conditions in which glaucoma may cause complications are also discussed. Each management approach is accompanied by easy-to-read flow diagrams or 'decision trees'.

The latter half of the book is devoted to surgical intervention, laser and filtration techniques. the discussions in these sections are accompanied by clear, step-by-step diagrams.

Overall this book provides a good, easy to read, general reference text on all forms of glaucoma, their diagnosis and management.

Alice Rota-Bartelink.

OCULAR DIFFERENTIAL DIAGNOSIS.

Frederick Hampton Roy.
Lea and Febiger, Philadephia, 1993. Fifth Edition.
ISBN 0-8121-1594-5

This text is an extensive book of lists, and is designed to facilitate ocular differential diagnosis where each ocular symptom and sign is indexed under the ocular structure affected. All

possible associations of the sign or symptom are subsequently listed, allowing the clinician to then consider the further tests required in order to differentially diagnose the condition. Cross referencing of signs and symptoms is easily facilitated.

In the fifth edition of this text the author has also developed a new method of diagnostic decision tables. These tables which include history, physical signs and laboratory tests allow progression from differential to specific diagnosis for several of the ocular conditions.

This text will serve as an exhaustive reference to those clinicians requiring comprehensive lists detailing the relationship of ocular signs and symptoms to specific ophthalmic and systemic conditions and syndromes.

C. Devereux

CLINICAL MANAGEMENT OF STRABISMUS.

E Caloroso and W Rouse.
Butterworth Heinemann.

As its own preface states, the book is written by an optometrist for optometrists and therefore excludes much of the vital role of the orthoptist in the diagnosis and treatment of these conditions. Chapter one, the model of the visual pathway, introduces the concept of good visual acuity in hand with binocular single vision, which incorporates a very simple overview of motor and sensory perception. The chapter on diagnostic evaluation covers history taking, the practice of refraction through to the cover test and 4Δ prism test all of which are accompanied by clear diagrams. The next three chapters on conservative and surgical therapy are somewhat confusing as there is no classification of strabismus and concomitant and incomitant strabismus are combined, whereas the chapter on strabismus management strategy is far clearer. The topics of suppression, amblyopia and ARC are not related to specific conditions and the lack of any modern clinical input is apparent. The last chapter on vision therapy again includes a broad overview but is not specific. The book is recommended with reservation to those already possessing a basic knowledge of orthoptics. The volume is well written and shows the authors attention to detail, particularly in the standard of line drawings. The particular emphasis would be of interest to 3rd and 4th year orthoptic students.

J. D. Green

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The above must be stated in a covering letter.

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