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TINTED LENSES AND DYSLEXIA: A REVIEW OF THE LITERATURE

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Abstract

Recent neuroscientific evidence has suggested that dyslexia may be the result of micro architectural changes in the language areas of the brain. However, treatments for dyslexia aimed at helping or curing one or more signs of dyslexia are still widespread despite a lack of any empirical or statistical evidence to support the validity of the treatment.

Amongst the therapies which have emerged for dyslexia is the use of tinted lenses.

Since 1983 when the use of tinted lenses to treat dyslexia was suggested much interest has been generated into the effects of this technique. Since then the treatment has been used to treat dyslexic patients in Australia despite the lack of supporting clinical evidence.

The review of the literature on tinted lenses revealed that there is no consensus of opinion on the efficacy of this treatment. The main questions that remain unanswered in relation to the treatment are firstly; does it work and secondly; if so does it affect vision or is it a placebo effect?

Key words: *Tinted lenses, dyslexia, contrast sensitivity, children, reading.*

INTRODUCTION

Dyslexia, a severe reading problem that cannot be accounted for in terms of low intelligence, has been reported to occur in between 4 and 10% of school children. Many of these children have other problems such as poor spelling, clumsiness, poor motor control, difficulty distinguishing between shapes and difficulties differentiating between right and left. They also have lower verbal than practical intellectual skills.[1]

Many studies on dyslexia have pointed to abnormalities in the visual system, including visual acuity problems,[2,3] ocular motor control anomalies and defective binocular vision. Despite this no definite causal relationship between the visual system in dyslexia has emerged.

Recent evidence of cortical abnormalities coupled with the mounting evidence of linguistic anomalies suggest that dyslexia is more likely to be a problem of language secondary to micro architectural changes in the brain.[4-7]

As dyslexia manifests itself in tasks other than reading it appears that activities supported by the affected parts of the brain are also affected.

Following a "60 Minutes" television programme broadcast in Australia in which Irlen, a psychologist from USA, advocated the use of tinted lenses in patients with learning difficulties much interest was generated into the effects of this technique. The report on television suggested that tinted lenses improved reading abilities instantaneously. During the lectures given in Australia and one given in the USA claims advocating improvement after one month were made.[8,9] These conclusions were based on observation and self report from 37

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learning disabled adult students. Unfortunately no control group was tested and motivation and/or placebo effects were not taken into account. Also no data or statistics to back the claims of reading improvement have been forthcoming.

These subjects complained of symptoms such as words going out of focus while reading, words moving about on the page, words appearing washed out on the white paper, disturbances and distortions of perceptual lines, photophobia, unstable appearance of the print, reduced span of recognition, reduced field of vision and complaints that the page was too bright and the page competed with the print for the subject's attention. These symptoms have been described in many publications discussing the symptoms associated with dyslexia.[10-14]

Irlen suggested that the symptoms were due to a retinal defect causing sensitivity loss to a particular part of the retina. She called the condition "scotopic sensitivity syndrome". The name "scotopic sensitivity syndrome" implies that it is sensitivity to very dim light conditions (when the rod system is activated) which causes the problems experienced by dyslexics, however, the problems are generally reported to occur under normal reading conditions (i.e. when using cone receptors in photopic conditions). No other reports that the sensitivity of the scotopic system in any way affects reading ability in dyslexics or normals were found in a medline search.

Irlen claimed that tinted lenses improved reading in patients with scotopic sensitivity because the tinted lenses excluded a wavelength of light that had interfered with reading ability. The optimal tint was selected by a procedure to determine the appropriate strength and density of tint needed. Details of this procedure have not been published to date.

Over the years there have been a number of studies on the effect of using coloured letters and/or paper on reading. Tinker and Paterson[15] compared reading speeds for 10 combinations of coloured ink on coloured paper as well as black ink on white paper. They found that reading was faster for black print on white paper than for any other combinations. As a result they suggested that luminance contrast was more important in determining reading speed than was colour.

Another study by Luckiesh and Moss[16] also showed that reading was fastest with black print on a white background. They measured reading rates for black letters on 10 different coloured papers ranging from white to red. They suggested that the different coloured backgrounds affected the contrast and that although subjects read faster at higher contrasts (black letters on a white background) there was only a 7% difference in mean reading rates across the whole range of colours.

In 1980 Meares[11] attributed reading difficulties experienced by some dyslexics to the difficulty in seeing the words due to the high contrast between the words and the background. She suggested the use of dirty perspex sheets over the reading material of dyslexic subjects improved their reading and that the improvement may be secondary to a reduction in the brightness of the printed material.

Rozin et al[17] taught dyslexics and normals to read using black ink Chinese figures as well as English words on white paper. They demonstrated that the dyslexics did not have any 'visual' problems when learning to read the Chinese figures. They performed as well as the normal subjects. The dyslexics 'visual' problems emerged when the tasks involved alphabetic sequencing. They concluded that it would be impossible for the dyslexics to learn to read Chinese figures if their problem was one of vision or visual integration. Furthermore the finding that many dyslexics show prowess at technical drawing and become architects and draftsmen casts more doubt on the theory that high contrast affects the ability to resolve fine detailed print.[18]

A number of researchers in Australia have assessed the effect of tints of the patient's choice on patient's reading performance. All groups have tested reading disabled subjects with symptoms considered to be characteristic of the so called "scotopic sensitivity syndrome". Stanley[19] (1987) reported on a study of dyslexic children who were divided into two groups and given coloured overlays to see if there was any improvement in their reading. Some of the chil-

dren showed faster reading after three weeks and others did not (detailed results of this paper are still in press). Stanley concluded that his experience with coloured filters suggests that ". . . there can be quite real motivational effects produced when coloured filters are used, but the basic problems of reading disability are not removed".

A similar study by Robson and Miles[20] demonstrated immediate positive effects on some aspects of reading performance (word matching, letter recognition and number recognition) using coloured overlays. However the subjects word recognition did not improve.

O'Connor and Sofo[21] investigated the effect of coloured plastic overlays on dyslexic children's reading. The children who were given their preferred overlay colour showed improvement in reading rate and accuracy. Those given clear overlays or non preferred coloured overlays remained essentially unchanged.

In another study, Cheetham and Ovenden[22] (1987) assessed 225 dyslexic students who responded to treatment with tinted lenses. Their patients ranged in age from five to 58 years with an average age of 17.1 years. Although their patients reported that tinted lens wear led to more stable print and better separation of words and sustained concentration on reading tasks they concluded that tinted lenses facilitated reading in poor readers but ". . . did not cure the reading problem". They also stated that it was rare to see a student responding simply to a tint. They suggested that further research is necessary to answer the two questions; does the tinting work, and if so, how does it work?

Winter[23] (1987) tested 15 dyslexic primary school children with Irlen lenses. The children were tested on timed letter identification tasks. No improvement was found for speed or accuracy of performance.

One study from America using tinted lenses on remedial high school students and a matched control group showed significant improvement for timed reading scores and finding a place on a printed page when wearing tinted lenses.[24]

In a study assessing the effect of wearing tinted lenses for 12 months on 44 dyslexic subjects ranging in age from nine to 14 years, Robinson and Conway[25] showed improvement in reading accuracy and comprehension which was more likely to occur in the first six months. They hypothesised that this improvement could be due to an improvement in visual clarity affecting re-reading (i.e. improved ability to see print on a page by altering the background colour of the page thus reducing the contrast between the print and the background), which thus enhanced spontaneous gains in comprehension. Alternately, improvement may have been due to a placebo effect created by the tinted lenses. They did not formally assess visual clarity or measure visual clarity improvement.

In one study[26,27] in which the author participated, reading disabled children were assessed by a special education teacher using the Neal Analysis of Reading and were then prescribed tinted lenses. After at least one term at school there was no statistically significant change in reading.

In a recent review article, Wilsher and Taylor[18] suggested that ". . . dyslexics should differentiate between visual preference (and other psychological factors), affecting the way they see the page after treatment with tinted lenses, and whether they actually improved reading ability. Most people (both normal and dyslexic) may prefer to see the world through rose-coloured glasses but this does not mean that the effects experienced are any more than placebo". They went on to suggest that the psychological effects could be explained in terms such as the attention factor; the patient felt someone was trying to help, the novelty value of seeing the page in a new light, the reduction in anxiety because the previously 'hidden' handicap could now be seen by everyone and would be taken seriously, the belief that the new treatment would help, the increased motivation to read now that they had a new treatment that they thought was going to help after years of failure and the 'glamour' of wearing the glasses.

Similar conclusions may be drawn from another study.[26] After three months of wearing tinted lenses reading disabled children's reading performance had shown no statistical improvement but, according to their parents, their self esteem had greatly improved.[27]

As previously mentioned, Robinson and Conway[25] discussed the possibility of placebo effects on the outcome of their study. They suggested that the fact that reading gains in their study occurred in patients wearing either intermediate or optimal tints (despite Irlen's claims that only optimal tints should have the desired effect), could suggest a placebo effect. They suggested that the improved scores were also associated with improved attitudes to school and school tasks. They cited research which suggested that success at school could depend on motivation. If the children were no longer experiencing persistent academic failure, motivation may have been increased.

The finding of subjective reports of improvement after wearing non optimal coloured lenses was indirectly supported by another study on dyslexic patients.[26] Results showed that when asked to choose the colour of print overlay which they preferred to read through, the subjects chose one colour for their preferred print overlay and most chose a different colour for their tinted lenses. This suggests that the colour may not be critical.

These findings conflict with Irlen's[8] suggestion that only the optimal tint would filter out the wavelength of light that was causing the retinal problem found in "scotopic sensitivity syndrome". Thus the finding of improvement with non optimal tinted lenses must beg the question of the placebo effect of the tinted lenses.

In a number of the patients in one study,[27] non tinted (clear) lenses were selected as the optimal tints in some cases. Two of the subjects prescribed these lenses reported that they felt better about themselves wearing the lenses and they felt that their reading had improved. Despite this their reading accuracy and comprehension did not improve when assessed with the Neal test. As the clear lenses do not filter out any wavelengths of light or reduce the contrast between the page and the print the use of these lenses raised the possibility of a placebo effect from tinted lens wear.

In the same study there were a large number of the children who virtually gave up wearing the lenses. The parents stated that they lost interest in the lenses. Despite this (and the finding that there was no improvement in reading accuracy and comprehension after at least three months of tinted lens wear) some of the parents reported that the children were "doing better" when they wore the lenses. One wonders at the extent to which the parents wanted the treatment to work for their children and the subsequent effect that this had on their reports of their children's performance in the lenses. In turn, perhaps to please their parents, a number of the children who gave up wearing their lenses reported that the lenses had "helped their reading but they just did not want to wear them".

In a study into contrast sensitivity in normal and dyslexic children,[28] the hypothesis that dyslexic patients' ability to discriminate fine detail was improved by wearing tinted lenses was investigated. If the dyslexic children were better able to resolve fine detail this may be the reason behind the reports of their improved ability to see the print on the page and thus to read when wearing tinted lenses. In order to test this hypothesis normal contrast sensitivity for 325 children and 25 dyslexic children of the same age without tinted lenses was first established. The effect of tinted lenses on contrast sensitivity in the same groups of normals and dyslexics was established. The study clearly demonstrated that this population of dyslexics had normal contrast sensitivity for their age.

Wearing tinted lenses made a white page appear the same colour as the tint, reduced the contrast between the print and the page and reduced the amount of light entering the eye across the spectrum.[29] Despite this the results demonstrated that the ability to see the range of spatial frequencies, 1.5 to 18 cycles per degree at the contrasts below 100% was unaffected by wearing tinted lenses in normals and dyslexic children. As normally sized printed text (black letters on a white background at or just under 100% contrast) fell into the range tested[30] it may be assumed that sensitivity to print was unaffected by the reduction in contrast induced by tinted lenses wear. (Statistically tinted lens strength and or colour had NO effect on contrast sensitivity (ANCOVA $p>0.05$) in either the normals or the dyslexics.)

A number of papers have reported tinted lenses improved visual clarity thus reading improved after tinted lens wear.[8,9,25] They suggested that vision was improved despite the fact that none of the researchers did formal quantitative tests of visual function on the subjects in their studies before or after tinted lens wear. Their suggestions were based on subjective evidence from the patients (i.e. the subjects said that they could see better).

The finding from the study on contrast sensitivity with tinted lenses did NOT support the visual anomaly theories of dyslexia or the "scotopic sensitivity" theory, however, it did not conflict with some of the other theories. If, as strong anatomical evidence suggest,[4-6] dyslexia was the result of abnormal brain micro architectural affecting the linguistic processing areas one would not have anticipated that tinted lenses would have had an effect on the ability of dyslexics to see fine print on a page or to read.

CONCLUSIONS

The finding of no improvement in reading after tinted lens wear had been supported by a number of authors.[18,23,26] However, there is a body of literature that has reported improvement in reading or reading related skills after tinted lens wear or the use of tinted print overlays.[8,9,11,20-22] Others reported mixed success with the treatment with some cases showing improvement and others showing no improvement.[19]

As well as the debate in the literature as to whether or not the treatment works there is a debate as to why tinted lenses may work. The hypothesis put forward included:

(1) the correct tint filtered out the wavelengths of light to which the person was uniquely sensitive (scotopic sensitivity syndrome);

(2) the tinted lenses improve the clarity of vision when looking at normal black print on a white page;

(3) the placebo effect of the lenses.

Analysis of the tinted lenses[29] has revealed that tinted lenses only filter out some of the light at any given wavelength. The stronger the tint the more light is filtered out but even with 75% strength some light at ALL wavelengths passes through the lens. At 15% very little light is filtered out. Thus the hypothesis that tinted lenses filter OUT a band of light may be questioned.

The hypothesis that tinted lenses improve the clarity of vision when looking at normal black print on a white page was not supported in the only study that has quantitatively assessed the effect of tinted lenses on vision and contrast sensitivity.[28] The study concluded that any change in reading ability after wearing tinted lenses was not the result of any effect of the lenses on the patients ability to see the print when the contrast between the page and the black letters was reduced by the tinted lens. The other papers that reported improved clarity did not report on any formal assessment of vision at all.

The possibility that the reported change in reading ability with tinted lenses may be related to psychological factors must not be ignored. Certainly by wearing tinted lenses there is a visible identification of the learning difficulty. It may be suggested that the problem has been taken from within and put on the nose.

To date there is no anatomical or physiological evidence that dyslexia is the result of visual anomaly. Although some psychophysical evidence pointed to the visual system it was frequently not substantiated between studies. The majority of the literature pointed towards anatomical anomalies affecting the linguistic processing areas of the brain. As yet there is no treatment for dyslexia which has been both effective and caused 'fast' improvement. If the problem stems from abnormal brain architecture, there may never be a 'cure' for the problem.

The treatment of dyslexia using tinted lenses is a non invasive therapy that cannot physicially harm the patient. However, it must be made clear to parents that there is no substantiated evidence that the dyslexic children have a visual problem and even if tinted lenses help they certainly do not cure reading problems.

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DYSLEXIA IS A DAB WORD

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Abstract

Dyslexia is a word which is used inappropriately by the general public and has acquired a number of undeserved connotations. A brief historical review is presented, and modern theories concerning the cause of specific primary dyslexia are presented.

Key words: *Dyslexia, learning disability, reading disability, dysgraphia.*

Consider for a moment what it would be like not to be able to read public notices, to be intimidated by a page of print, or to be cut off from our immensely rewarding literary heritage. These problems are part of the daily experience of a dyslexic.

The disorder now known as dyslexia was first described by Kussmaul in 1877 as "word blindness", but the word "dyslexia" was coined by Berlin in 1887. It was the Scottish Ophthalmologist Hinshelwood[1] who first differentiated alexia from dyslexia in 1895.

Dyslexia must be differentiated from the other four learning disabilities — educational deprivation, intellectual impairment, disorders of special senses (deafness, blindness), and emotional disorders. The group of disorders known as dyslexia can be further sub-divided into specific primary dyslexia, hyperkinetic syndrome, dysphasia, dysgraphia, and dyscalculia. Because it has been loosely defined in the past, and because it has acquired connotations of dim wittedness, laziness, and delinquency in some quarters — dyslexia is a bad word. The proposed alternatives include dyssymbolia, specific reading disability, word blindness, and strephosymbolia — but all are too narrow or too oblique in meaning.

The remainder of this discussion concerns specific primary dyslexia, which is defined as, "a disorder manifested by difficulty learning to read despite conventional instruction, adequate intelligence, and sociocultural opportunities. It is dependent on fundamental cognitive disabilities which are frequently of constitutional origin".[2]

Dyslexia occurs in languages which progress from right to left as well as those which progress from left to right, but the lack of logical orthography in English means that dyslexics are identified more easily. It affects 15% of school age children, 50% of young unemployed, and 70% of juvenile offenders.[3]

Some factors argue a genetic cause — an incidence in males eight times that in females, higher incidence in those with dominant left hand or left eye, 100% concordance in identical twins, 33% concordance in dizygotic twins, and up to 88% incidence in relatives of those affected.

Other factors argue an acquired cause — a 50% higher incidence in the second half of large

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sibships, and a high incidence of major stress events in early life.

A large number of epiphenomena are described, including EEG abnormalities, colour agnosia, clumsiness, left right confusion, other abnormalities of language development, poor attention span, slowness and variability of performance when using other symbols. Like other syndromes there is a large variability between cases, but a common pattern unites them. Unfortunately some investigators have been distracted by these epiphenomena — and spurious new syndromes have been defined, such as minimal cerebral disfunction.

Medical attention has been predictably focused on the problem of "where is the lesion?" In the early 1900's the angular gyrus was suspected and indeed a number of acquired neurological conditions manifest reading difficulties. Lesions around the left angular gyrus due to posterior cerebral infarction, aneurysm, metastasis or meningioma, can cause alexia without either agraphia or the usual right homonomous hemianopia. This is due to disconnection of the occipital cortex from Wernicke's area. Bilateral lesions in the visual association areas cause prosopagnosia, simultanagnosia, or optic ataxia, depending on the site of the lesions.[4] While these may all produce reading difficulties, none are like dyslexia.

Galaburda et al in 1985[5] reported lack of the usual asymmetry in the planum temporale — and subsequent reports have added weight to the notion of failure of right hemisphere regression in dyslexics. This work supplants previous notions of delayed myelination in dyslexics.

The notion of a visual defect in dyslexics has persisted despite the recent ascendancy by theories supporting a primarily central or language problem. Pavlidis[6] reported saccadic abnormalities in dyslexics, and despite several excellent studies refuting his findings, the notion of saccadic problems has found a place in medical folklore. While dyslexics do demonstrate abnormal saccades when reading — a situation which requires higher processing — they perform quite normally when using a target such as flashing LED's in an array. A small number have abnormal saccadic intrusion when tracking a target at pursuit velocities of 5° per second.[7] Two more recent findings may be of note, especially when considered together. Lovegrove[8] reports that dyslexics have abnormalities in their transient ganglion cell function. It is known that during reading, saccades of approximately 25 m-seconds intrude between fixations of around 250 m-seconds. Each fixation absorbs a letter string of some 20 spaces — forming a precategorical mental icon in short term visual memory. However, the after image of the sustained cells is some 300 m-seconds, so it intrudes on sebsequent fixations. Transient retinal elements — Y cells and some W cells — have shorter retina to cortex conduction times and shorter after images.

Geiger and Letvin[9] reported that dyslexics have greater ability to detect a letter flashed in their extra foveal field, whereas non dyslexics detect a letter flashed in their foveal field better than dyslexics. It is known that the fovea has mainly sustained, or X type ganglion cells, while the extra foveal retina has mainly transient elements, with better temporal but poorer spacial resolution. Could it be that dyslexics fail to make the central adaptations necessary to enable rapid sequential fixations, and instead employ the strategy of using extra foveal retina, with its transient elements? In so doing, normal saccadic function, which serves to foveate the fixation target, would have to be altered, thus explaining the abnormal saccades.

Whatever the real answer may be, it is still safe to say that we do not yet know the site of the lesions.

Dyslexic patients usually present with poor school performance at second or third grade level, and may demonstrate certain characteristic features which help to confirm the diagnosis. There may be a positive family history, some difficulty with colour naming, crossed hand eye dominance, some history of retarded language development, left right confusion, a tendency to read letters on the chart which are not there, and skip other letters which are usually read, and some difficulty with compound cooperation such as is required for slit lamp and visual field examination. It is important to ensure that the child

has had an adequate educational opportunity, that he has had normal developmental milestones and does not have any hearing or visual impairments. Assuming emotional disturbances have been excluded, the diagnosis can be confidently made at this time. A record of reading age, handwriting, and a standard drawing test, is useful.

What is the management of dyslexia? In the past, both perceptual training and visual training have been advocated, and both have been systematically proven to have no benefit in the treatment of dyslexia. In 1971 the American Academy of Ophthalmology and the American Academy of Paediatrics made a strong statement to this effect. The use of coloured glasses has recently come into vogue, and was introduced into this country in 1985 following a report on the 60 Minutes television programme. Since that time no scientific evidence has been produced to support the use of this modality, and the Royal Australian College of Ophthalmologists, the American Academy of Ophthalmologists, and the American Optometrist Association have all made statements to this effect. In Western Australia those prescribing coloured lenses follow either the Irlen technique, and pay a five thousand US dollar per year franchise for the right to prescribe these lenses, or else the Meares technique. These lenses are aggressively marketed within the local schools, and it is fair to say that a great deal of time and money is being diverted from conventional treatment modalities by these coloured lens prescribers. The only treatment which confers proven advantage to dyslexic children is a very thorough and systematic educational programme, supplemented by additional remedial tuition. This is arranged after assessment by the local school guidance officer who is under the control of a district guidance officer, who is in turn controlled through the Education Support Branch of the Education Department of Western Australia.

There are also private clinics available.

The role of the Ophthalmologists and Orthoptists in the management of dyslexia is to treat any coincidental visual problem, make a firm diagnosis, advise the family of where best to seek assistance, and offer an informed opinion regarding the controversial treatment modalities. We must all make an effort to keep in touch with developments in this field as these families are extremely vulnerable to unscrupulous practitioners who are able to profit out of their misfortune.

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NORMAL CONTRAST SENSITIVITY IN 200 CHILDREN AGED SEVEN TO 13 YEARS

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Abstract

The Vistech VCTS 6000 and 6500 tests are designed to accurately assess contrast sensitivity under a specified illumination. A normal range of contrast sensitivities was provided by Vistech for both tests based on results from 300 subjects ranging in age from 10 to 70 years. As contrast sensitivity varies with age it was hypothesised that the normals given may not have accurately reflected contrast sensitivity for children. Two hundred children between the ages of seven and 13 years (with 6/6, N5 vision and no strabismus) were assessed with both tests. Results show that subjects in this age group are more sensitive to low contrast for high spatial frequency gratings than the test normals suggested.

Key words: *Contrast sensitivity, Vistech chart, normals, children, CSF.*

INTRODUCTION

As objects in the visual world are not usually made up of black outlines on a white background like letters on a conventional visual acuity chart, visual acuity measurement tells the examiner nothing about the visibility of objects larger or smaller than the letters on the chart or objects that are not 100% contrast. As a result contrast sensitivity tests are being used to assess the ability to recognise subtle shadings on low contrast backgrounds. As the targets (gratings) vary in size and orientation and in the number of repeats of the pattern (spatial frequency) as well as contrast, much more information can be gained about visual capabilities.

Contrast refers to the differences between the maximum and minimum luminance of the grating. It is defined by Michelson as

$$c = (L_{max} - L_{min})/(L_{max} + L_{min})$$

where c is contrast, L_{max} is maximum luminance and L_{min} is minimum luminance. Michelson contrast ranges from zero (no contrast) to one (100% contrast or black on white).[1]

Contrast sensitivity at high spatial frequency (narrow gratings) is needed to see fine detail and small print and it has been demonstrated that high spatial frequency resolution is related to above average Snellen acuity. Low and middle spatial frequencies have been correlated with the ability to see large low contrast targets. Recognition of human faces depends heavily on low spatial frequencies.[2-4] Patients with defective low to middle contrast sensitivity and normal high contrast sensitivity can therefore see the bottom line of a vision chart (which indicates normal vision) but may not see large objects on a low contrast background.

Assessment of contrast sensitivity is an important diagnostic tool for assessing visual deficit in a number of conditions. These include amblyopia[5-7] refractive error and astigmatism,[8] glaucoma,[9] cataract,[10] macular disease,[11]

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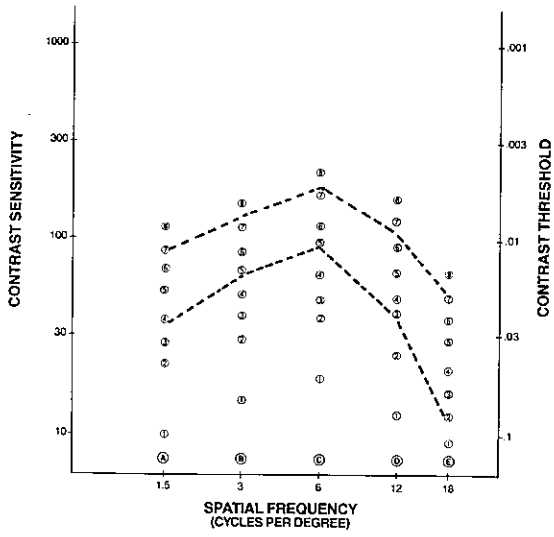

Figure 1: VCTS evaluation form (reproduced with permission from Vistech Inc).

multiple sclerosis[12] and optic neuritis,[13] corneal oedema[14] and cerebral lesions.[15]

There are a number of different techniques available for the assessment of contrast sensitivity, the most recently introduced are the printed photographic test stimuli. These techniques are cheaper and quicker to administer and simpler to design than the tests that use visual display unit (VDU) stimulation.

The first of the printed photographic tests was developed by Arden and produced by the American Optical Company and were therefore known as the AO plates. Many investigators have criticised the AO plates because they were designed for screening thus they only tested up to 6.2 cycles per degree. In order to test contrast sensitivity in patients with vision that ranged from normal to near blindness, stimulus contrasts that ranged from near normal threshold values (0.003) to a maximum of 1.00 were needed. As the contrast range did not extend to the very high spatial frequencies many patients with visual problems performed normally on the AO plates.[16]

Ginsburg[17] (1984) developed a set of plates known as the Vistech VCTS system. The Vistech VCTS 6000 and 6500 contrast sensitivity tests measure the subjects ability to detect bands or gratings of various spatial frequencies and different contrasts under specified illumination at 1/3 m and 3.05 m, respectively. The minimum contrast at which the grating can be seen is known as the contrast threshold and contrast sensitivity is the reciprocal of the contrast threshold.

The Vistech tests come with evaluation forms (see Figure 1) and an overlay (represented by dotted lines) showing the normal range of contrast sensitivity in patients tested binocularly.[18] This normal range is based on a sample of 300 people ranging from 10 to 70 years of age measured under specific luminance conditions.[19,20] (For each row the mean was assessed and the upper and lower boundaries of the normal range represented the 5th and 95th percentiles.)[18] According to the Vistech manuals the contrast sensitivity of normal individuals age 50 years and younger with no visual complaints, should fall in the upper half of the normal range.[19,20]

Many authors have demonstrated that contrast sensitivity increases throughout childhood then decreases with old age; mostly at mid and high spatial frequencies.[4,21-30] Another author suggested that contrast sensitivity did not alter with age.[31]

Diagnosis of reduced sensitivity in the Vistech tests is based on the lower limit of the normal range as indicated by the overlay. As the age range that these normals were based on was a predominantly older population that varied greatly in age it was considered that the Vistech normals may not accurately reflect the normal contrast sensitivity for school children between the ages of seven and 13 years. This paper reported the findings of contrast sensitivity using the VCTS 6000 and 6500 on 200 children in that age range.

METHOD

Two hundred and twenty five children who attended the same primary school were selected to form the normal population. A random computer allocation of the order of all testing procedure was done for all 225 children, with

each child being issued with a case number as they came into the room for testing. Classes were selected at random throughout the testing time.

Visual acuity assessment, cover test and Lang stereo tests were performed in random order on each child. Of the 225 children, 200 had monocular visual acuity of 6/6 (Snellen's linear chart), N5 (reading chart) with each eye without optical correction and no strabismus, and were thus included in the study group to have their contrast sensitivity tested. The mean age of the children was 10.34 years and none of the children had had previous exposure to the tests.

Contrast sensitivity testing procedures were exactly the same as those used by Vistech.[19,20] The order of testing (ie VCTS 6000 or VCTS 6500 performed first) was randomised. Both tests were conducted binocularly using uniform and constant illumination between 30-50 ft-L. Light intensity readings from one area of the chart to another and from the VCTS 6000 to the VCTS 6500 were within 5° of pointer movement of this range. As long as the readings were in this range, results could be compared with the Vistech population normals.

Both Vistech tests had 40 sine wave grating targets. Each target consisted of a number of bands (sine waves) which either pointed upwards (0°) or were tilted 15° to the right or left of the vertical position. Subjects were asked to identify the orientation of the bands in each target they could see in each row in turn. The orientation of the bands was randomised along each row to help control for guessing. The mean luminance of the targets was 90 cd/m² and the mean luminance of the surround was 125 cd/m².

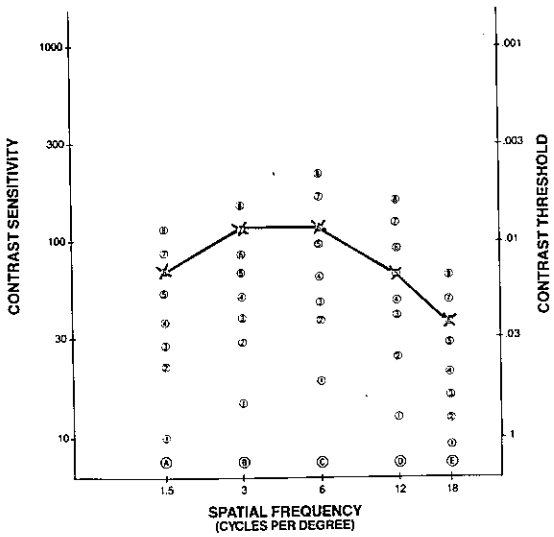

Figure 2: VCTS evaluation form showing normal CSF curve.

The targets were divided into rows A, B, C, D and E each consisting of eight targets. All the targets in a given row had the same spatial frequency (band width) and a different contrast. The spatial frequencies were row A; 1.5 cycles/degree, row B; 3 cycles/degree, row C; 6 cycles/degree, row D; 12 cycles/degree and row E; 18 cycles/degree.

The highest contrast targets (with a contrast threshold of 0.1) were found at the left hand end of each row (in column 1). Targets became progressively lower in contrast across to the right of the chart. In each row targets were numbered one to nine with the lowest contrast targets being in column eight. In column nine there were no bands.

Results of each test were recorded on Vistech contrast sensitivity evaluation forms with the target number marked with "x" in each column corresponding to the lowest contrast target orientation correctly identified by the subject in each row. The "x's" were connected by a line forming a curve known as the contrast sensitivity function (CSF) curve (see Figure 2). The curve was the graphic representation of spatial contrast sensitivity as a function of the spatial frequency of the gratings. On the evaluation forms the horizontal axis represented spatial frequency with the lowest spatial frequency row (row A) being on the left hand end of the axis. The vertical scale on the left gave the log of the contrast sensitivity. The vertical scale on the right gave the log of contrast threshold (one/contrast sensitivity).

To be able to directly compare the results of the contrast sensitivity tests to the Vistech norms the normal range was assessed using percentiles. The 5th and 95th percentiles were calculated for each row of the tests.[32]

The null hypotheses were that the CSF curve would not be affected by whether the child was

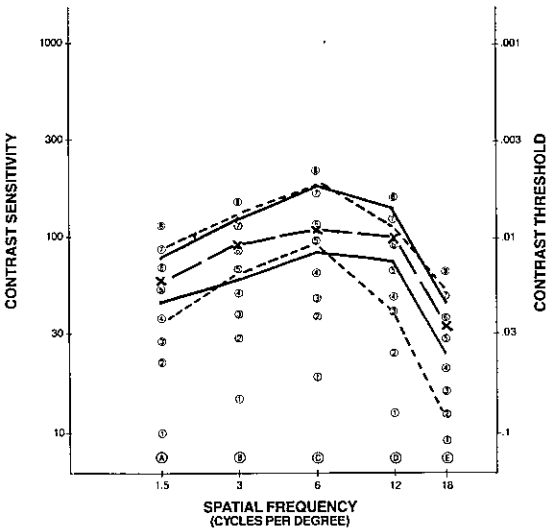

Figure 3a: Normal contrast sensitivity function curve, 5th and 95th percentiles (black lines) for children aged seven to 13 years for VCTS 6500 (3.05 m).

------- VCTS Normal limits
——— Normal limits seven to 13 years
X Mean scores
X--X--X CSF curve

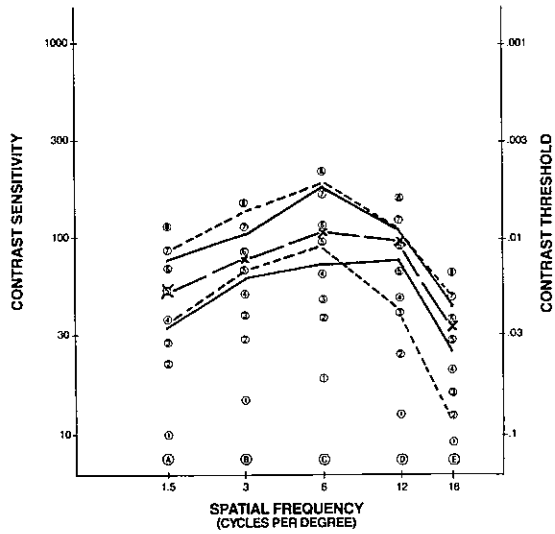

Figure 3b: Normal contrast sensitivity function curve, 5th and 95th percentiles (black lines) for children aged seven to 13 years for VCTS 6000 (1/3 m).

------- VCTS normal limits
——— Normal limits seven to 13 years
X Mean scores
X--X--X CSF curve

assessed firstly at near or distance test or by the sex of the child (ie the independent variables).

To ascertain whether these independent variables would effect the scores analysis of variance (ANOVA) was used. This analysis was performed using Statsoft CSS Complete Statistical Package; Statsoft, 1987. The significance level used was 0.05. (If $p < 0.05$ then it was not very likely that the result had arisen by chance.)

RESULTS

For every child the minimum contrast seen for each different spatial frequency (that is each different row) was recorded on an evaluation form.

The mean score was calculated for each row as were the 5th and 95th percentiles (see figures 3a and 3b). Scores that fell within this range were considered to be within normal limits (see Tables 1 and 2). From the mean scores a normal contrast sensitivity function curve was constructed. This normal range of scores could be directly compared to the normal ranges of the Vistech form.

These results have clearly demonstrated that for children between the ages of seven and 13 years, contrast sensitivity for high spatial frequencies (narrow bands, rows D and E) was much higher than the range suggested by the Vistech normals. Thus children appreciated these targets at much lower contrasts than was previously realised. As a result the shape of the contrast sensitivity function curve for children differed from the shape of the curve for the 10 to 70 year old subjects tested by Vistech. These findings agreed with the findings reported for 325 children aged six to 12 years.[33]

The normal range for mid spatial frequencies (row C) showed a mixed result. The lower limit of the normal range for both the tests were slightly lower than the Vistech normal range. The upper limit of normal was the same as the Vistech normals.

The maximum sensitivity to contrast (peak contrast) occurred at six cycles per degree for both the VCTS 6000 and the VCTS 6500. This maximum sensitivity is similar to the maximum sensitivity in adults on the VCTS tests.

TABLE I
Scores, Means and 5th and 95th Percentiles of Cases Achieving Each Score for the VCTS 6500 (3.05 m)

Score*	Row A	Row B	Row C	Row D	Row E
4	3	1	4	0	9
5	134	38	26	22	67
6	53	129	142	134	110
7	10	30	27	42	13
8	0	1	1	2	1
Mean	5.350	6.005	5.975	6.120	5.645
5th percentile	4.6	4.7	4.7	5.1	4.5
95th percentile	6.5	7.2	7.2	7.3	6.9

*Score refers to the target number of the lowest contrast target seen by the subject.

Results in this study were obtained testing subjects binocularly. Ross et al[26] reported only minimal improvement in contrast sensitivity in adults when tested binocularly rather than monocularly.

Analysis of variance revealed that sex had no effect on the score in any of the rows ($p>0.05$) and, in addition, the order of testing had no statistically significant effect on the scores (analysis of variance showed $p>0.05$ in all rows).

DISCUSSION

The study has clearly demonstrated that for children aged between seven and 13 years old, contrast sensitivity to high spatial frequency gratings is much higher than has been previously reported. This finding has been supported by a separate study conducted by the author in which another population of 325 children between the ages of six and 12 years old were assessed.[33]

A number of other studies which have assessed the contrast sensitivity in adults[4,21-30] have demonstrated a higher sensitivity at the high spatial frequencies in young adults.

Using VDU screens, a number of authors have demonstrated an improvement in contrast sensitivity with age.[22,27-30] In the other series of 325 normal children conducted by the author, results demonstrated that contrast sensitivity improved with age up to 10 years old. In this study the effect of age on contrast sensitivity within the study population was not investigated as there was an uneven distribution of cases in each age group.

Arden[31] reported that age does not influence results in a study carried out on patients aged from 11 to 70 years using the AO plates monocularly. Findings from most studies do not agree with Arden's, however, this could be explained by the fact that the AO test only measures contrast at relatively low spatial frequencies from 0.2 up to 6.4 cycles/degree. The Vistech test measured contrast sensitivity with higher spatial frequency gratings (up to 18 cycles per degree). The increased sensitivity in the seven to 13 year age group occurred only in the high spatial frequency gratings which were not used in the AO test.

The peak sensitivity in this study was found to occur at six cycles per degree for both the VCTS 6000 (1/3 m) and the VCTS 6500 test (3.05 m). A similar finding has been reported by

TABLE 2
Scores, Means and 5th and 95th Percentiles of Cases Achieving Each Score for the VCTS 6000 (1/3 m)

Score*	Row A	Row B	Row C	Row D	Row E
4	29	3	13	0	8
5	144	67	62	12	53
6	26	115	102	176	132
7	0	15	22	11	7
8	1	0	1	1	0
Mean	5.00	5.71	5.68	6.005	5.735
5th percentile	3.8	4.6	4.2	5.3	4.6
95th percentile	6.2	6.8	7.1	6.7	6.5

*Score refers to the target number of the lowest contrast target seen by the subject.

several other researchers using the Vistech tests on children and adults.[29,34] However, when using VDU display techniques to assess peak CSF a number of authors[27,35,36] found the peaks occurred at lower spatial frequencies around four cycles per degree.

CONCLUSIONS

The finding of increased contrast sensitivity for narrow stripes in children between the ages of seven and 13 has obvious clinical application. Children who had a low score in the high spatial frequency gratings were previously thought to be within normal limits. Following this study it is apparent that they are not within normal limits. Contrast sensitivity at high spatial frequency (12 to 18 cycles/degree, rows D and E) is affected in a number of conditions commonly affecting children. These include amblyopia, refractive error and astigmatism. As these conditions can cause unnecessary and permanent visual loss it is most important that appropriate norms be used when testing contrast sensitivity in this age group.

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Australian Orthoptic Journal 1989; 25: 17-19

THE USE OF THE PLANIMETER TO MEASURE FIELDS OF BINOCULAR FIXATION

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Abstract

The fields of binocular fixation of 20 normal subjects were plotted on an arc perimeter and the area of the fields was then measured with a planimeter. A planimeter is a cartographic device used to measure areas of two dimensional closed, irregular areas. The results showed that the planimeter gave an accurate quantifiable measure of fields of binocular fixation in normal subjects.
Key words: *Fields of binocular fixation, fields of binocular single vision, planimeter.*

INTRODUCTION

In Lyle and Wybar's "Practical Orthoptics in the Treatment of Squint" the average size of the binocular field of fixation (field of binocular single vision) is described as 50 degrees in up gaze, 50 degrees in direct down gaze with a reduction of approximately 15 degrees in dextro and laevodepression because of the nose, and 100 degrees in lateral gaze.[1] However, it has been generally accepted that irregularities of facial features, eg a large nose or deeply set eyes may alter the standard size of the binocular field. Consequently, quantifying the extent of a field of fixation has not been possible. The measurement has simply described a reduction of the field of fixation by comparing it to the "normal" or by a comparison made from one visit to another. In order to accurately obtain a quantitative measure of the size of a field of fixation, a cartographic device called a planimeter was used to measure the area as plotted by a perimeter. The planimeter is used by surveyors to measure areas of two dimensional, closed, irregular shapes. It consists of a base, a pole arm and a tracing arm on which is a clear plastic magnifying cylinder through which the outline can be viewed and traced. On the other end of the tracing arm is the scale which records the area traced as shown in Figure 1. The area is measured in either units of 0.1 cm^2 or .01 $inches^2$. To measure the area, the planimeter is set up on the recording chart, the outline of the field is traced and the area read directly from the vernier unit on the tracing arm, as shown in Figure 2.

The use of a planimeter in ophthalmology is not new.[2] It has been employed mainly in the quantitative analysis of sizes of scotomata in visual field testing both with patients with glau-

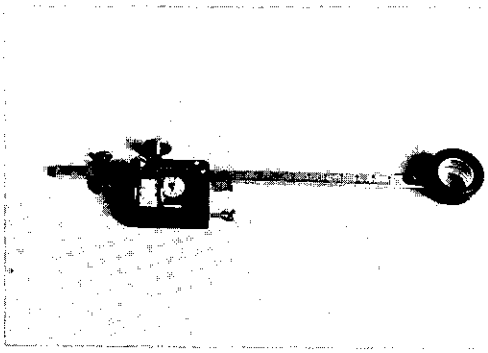

Figure 1: The planimeter, an instrument used by surveyors to measure areas of two dimensional, closed, irregular shapes.

coma and retinitis pigmentosa.[3-6] Those in favour of measuring field loss in terms of area, have argued that irregularities of some types of field loss can be accurately calculated and a quantitative measure of progression is then possible.[7]

To the authors' knowledge, planimetry has not been used to calculate areas of fields of fixation. The aim of this pilot project was to evaluate the cartographic method of obtaining a quantitative measure of the size of binocular fields of fixation.

SUBJECTS

Twenty normal subjects were chosen all with uncorrected right and left visual acuity of not less than 6/9, as measured on a standard Snellens chart at six metres; N5 each eye as tested with the Moorfields Bar Reading book at one-third metre; a full range of ocular movements; near stereopsis as evidenced by gaining 80 seconds of arc on the Titmus Test and full visual fields. None had any history of ocular problems.

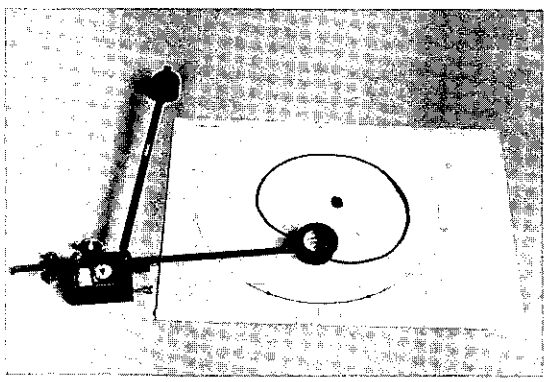

Figure 2: Use of the planimeter to measure the area of the binocular field of fixation.

METHOD

The authors have noted that it is possible to confuse the limits of the field of fixation with a field of vision. This can occur when the subject loses fixation but can still "see" the target while it remains within the visual field. Therefore to minimise this possibility, the Ellis (arc) perimeter was used with an accommodative target equal to Snellens N12 attached to the white 5 mm moving target. Three letters of N12 size were placed on the target of the perimeter. The rationale for using the accommodative target was that a blurred image would indicate the exact position when foveal fixation was lost and therefore the limit of movement. The subjects were instructed to place the chin centrally on the chin rest, with both eyes to follow the target from the central position without moving the head. The subject was asked to indicate at what point blurred vision or diplopia of the target occurred. This information was charted on the relevant binocular field of fixation chart. The planimeter was then set on the chart, the outline of the field was traced and a reading in square centimetres was taken.

RESULTS

The extent of the normal binocular field of fixation as described by Lyle and Wybar[1] is 50 degrees in elevation, 50 degrees in depression and 100 degrees laterally. When the contours of this field are measured by the planimeter, the area is 34.20 square centimetres.

The frequency distribution of fields of binocular fixation for the subjects in this study is shown in Figure 3 and as can be seen, there is a range of normal fields of fixation.

The mean, standard deviation and standard error are shown in Table 1. While the mean of 34.26 is almost exactly that of Lyle and Wybar, it has a negative skew, and so the median (36.45) more closely represents the obvious middle of the distribution. The negative skew is caused by the extreme values of this sample. No record was kept which could explain these extremes and so

TABLE
Range of Area of Fields of Binocular Fixation

Mean	Standard Deviation	Standard Error
34.26	7.40	1.64
Minimum	Maximum	Range
18.50	46.50	28.00

more subjects need to be examined to ascertain whether the skew is real in the sense that it represents the state of the population of fields of fixation, or merely measurement/sampling error.

DISCUSSION

The advantage of more accurate measurement in orthoptic investigation eliminates errors of clinical judgement and resultant variations between different clinicians. The monitoring of recovery of extra-ocular muscle paralysis, it is postulated, may be better carried out through this method than other methods, eg Hess Charts which the authors have noted frequently do not correlate with patient symptoms and clinical findings. This is a preliminary investigation to evaluate the use of the planimeter as a quantitative measure of the binocular field of fixation. The authors will now proceed to investigate the binocular field of fixation in subjects with known extra-ocular muscle paralysis, in order to assess its value in measurement of progressive recovery.

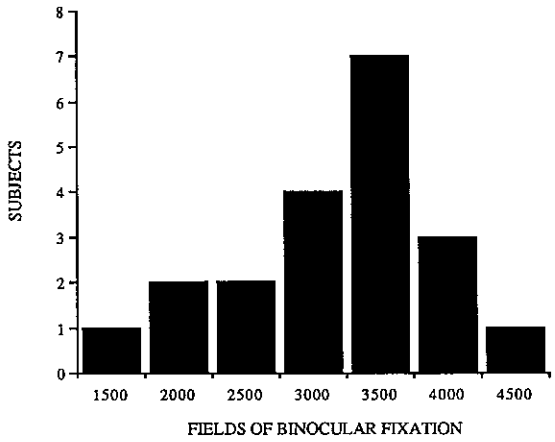

Figure 3: Frequency distribution of fields of binocular fixation.

The fact that some subjects fall outside the "normal" field of fixation is well known clinically and has been shown in this study. Because there is a range of apparently normal fields of fixation, the results should perhaps be compared intra subject rather than inter subject over a period of time.

CONCLUSION

The measure of the field of fixation by planimetry is a quick and easy measure, providing a numerical value which closely relates to the accepted standard normal field of binocular fixation. Clearly this has uses in measurement of both uniocular and binocular fields of fixation.

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ACCOMMODATION MEASUREMENT — CLEAR OR BLURRED?

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Abstract

The method of measuring accommodation using the Royal Air Force Gauge is ambiguously described in the literature. A study was undertaken to measure accommodation in 12 subjects. Each subject's accommodation was measured using two different methods. There was found to be no statistically significant difference ($p < 0.05$) between the measurements obtained using the two different methods.

Key words: *Air Force Gauge, accommodation, focusing.*

INTRODUCTION

The Royal Air Force Gauge (RAFG) is probably the most frequently used instrument for measurement of accommodative amplitude in Orthoptic practice. As such, it provides baseline measurements which aid the Orthoptist in decisions regarding existence of normal/abnormal function and whether treatment affects any change in accommodative amplitude. The amplitude of accommodation is the maximum physical increase in convexity of the crystalline lens of the eye, by contraction of the ciliary muscle, in response to a blurred retinal image. In the emmetropic population this response is specifically elicited by the proximity of a near object.

The RAFG or RAF Near Point Rule was developed at the request of the British Royal Air force by Allied Instrument Manufacturers (circa 1950) and accepted by Air Commodore Neely.[1]

There are varied descriptions regarding the exact method by which a measurement of accommodative amplitude should be made using the RAFG. Most authors instruct examiners to move the printed target from a distant point, towards the eye and record in centimetres, the position where the print becomes blurred. However, notable variations within the parameters of this instruction are as follows: ". . . it is brought up to the eye until it appears blurred",[2] or "The patient reports the moment when . . . print becomes too blurred to read".[3] Other instructions include: "The patient is asked to say when he first notices the letters becoming blurred",[4] or "The distance at which the patient reports blurring of the letters is measured".[5] Probably the most ambiguity is demonstrated by the following: "As the accommodation card is approximated to the eye its distance can be read off in centimetres and translated into dioptres of accommodative power",[6] and, finally: ". . . a test chart . . . is moved from or toward the eye until the closest point is found at which it still can be seen clearly".[7]

It was decided to investigate whether there were any statistically significant differences between accommodation measurements obtained using two different instructions/methods.

METHOD

Subjects were selected from patients attending for visual and Orthoptic screening at Lincoln Institute Orthoptic Clinic, between 12/2/1985 until 9/7/1985, and were arbitrarily appointed to one of two groups, according to their order of presentation at the Clinic. The first six were assigned to Group 1, and the second six to Group 2. Eleven subjects had no ocular abnormality except one with corrected ametropia. One subject in Group 1 was being treated for convergence insufficiency. Ten subjects were female and two were male. Ages of subjects ranged between 13 years to 45 years ($\bar{x} = 22$ years).

A RAFG was used to measure accommodation in all 12 subjects using two different instructions/methods. All subjects viewed N_5 print during measurement.

INSTRUCTION 1

"Tell me when you can read the print."

This instruction was given to subjects whose measurement was being recorded after moving the target from its most proximal position (at the nose), outwards.

INSTRUCTION 2

"Tell me when it's too blurry to read."

This instruction was given to subjects whose measurement was being recorded after moving the target from a distal point, towards the nose.

Each subject in Group I received Instruction 1 first. Immediately following this they were measured using Instruction 2. Each subject in Group II received Instruction 2 first. Immediately following this they were measured using Instruction 1. All subjects in both groups had measurements of the right eye taken first, followed by the left eye, then finally, binocularly.

When the subjects indicated appropriately (according to the Instructions), the actual measure of accommodation was recorded in centimetres from the RAFG measuring scale. If the measure was noted between two extremes on the scale, the distance recorded was the number indicated on the scale between the two extremes. All subjects were measured by the author. All measurements were recorded in the same Clinic room, with the same level of illumination.

RESULTS

T-Test Analysis, Non Directional ($p < 0.05$)

	Number (n)	Mean ($\bar{x}$ cms)	Standard Deviation (SD)	Degrees of Freedom (DOF)	Crit T	Observed T
Group I (Instruction 1 followed by Instruction 2)						
Right eyes						
Instruction 1	6	11	1.67	10	2.228	.06
Instruction 2	6	11.16	5.47	10	2,228	.06
Left eyes						
Instruction 1	6	9.66	1.74	10	2.228	.81
Instruction 2	6	10.83	3.08	10	2.228	.81
Binoc						
Instruction 1	6	10.16	.32	10	2.228	.25
Instruction 2	6	10.5	3.19	10	2.228	.25
Group II (Instruction 2 followed by Instruction 1)						
Right eyes						
Instruction 2	6	12	1.26	10	2.228	.77
Instruction 1	6	11.33	1.74	10	2.228	.77
Left eyes						
Instruction 2	6	11.66	.24	10	2.228	.66
Instruction 1	6	12	1.26	10	2.228	.66
Binoc						
Instruction 2	6	10.66	1.34	10	2.228	2.226
Instruction 1	6	12.33	1.27	10	2.228	2.226

In both groups there was no statistically significant difference ($p < 0.05$) in measurements recorded using Instruction 1 compared to Instruction 2.

DISCUSSION

The two instructions/methods of measurement were apparently asking for slightly different responses from subjects. Instruction 1 asked subjects to report when they could read N_5 print and therefore assumed they were using a maximum accommodative effort. Instruction 2 asked subjects to report when N_5 print was too blurry to read and therefore assumed their accommodation had failed. However, statistically there was no significant difference in measures of accommodation in each subject group, whether being measured for maximum accommodative effort (Instruction 1) or failure of accommodation (Instruction 2).

Clinically then perhaps we can assume that maximum accommodation and failure of accommodation are closely related. Clinicians should be reassured that measures of accommodation taken using either of the above instructions will not differ significantly from each other within a population where ocular pathology is minimal.

Further investigation of accommodation measurement should aim to validate the RAFG as a reliable instrument of measurement. This may take the form of comparing RAFG measures with those obtained when inducing accommodative effort by the use of concave spherical lenses. This present study should also be expanded to examine whether there are significant differences between methods of measurements employed to measure populations where abnormal accommodative function exists.

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PERSISTENT INCOMITANCE OF LONG STANDING FOURTH NERVE PALSIES

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Abstract

The length and course of the trochlea nerve causes it to be one of the most vulnerable of the cranial nerves. A retrospective study of the long term muscle sequelae of 32 fourth nerve palsies, both congenital and acquired, was undertaken to examine the development of concomitance with time. Particular reference has been made to the increasing dysfunction of the contralateral superior rectus muscle, compared with that of the primary palsy.

Key words: *Trochlea nerve palsy, fourth cranial nerve palsy, superior oblique muscle, superior rectus muscle, concomitance, incomitance, muscle sequelae.*

INTRODUCTION

When a fourth nerve palsy occurs, one would expect the greatest deviation to be in the direction of gaze of the affected muscle.

However, following a fourth nerve palsy the contralateral antagonist superior rectus muscle may become markedly restricted and the field of greatest deviation and separation of images is found in that direction of gaze. That is, the muscle sequelae which usually results in concomitance in the upper field, can change with time.

To better understand the muscle sequelae of paralytic strabismus, one must be mindful of Hering's Law of equal innervation (1879). "When a nervous impulse is sent to a muscle causing it to contract, an equal impulse goes to its contralateral synergist in order to maintain parallalism of the visual axis. When an underacting muscle requires extra innervation to bring about contraction, the innervation received by the contralateral synergist is equal but excessive, causing it to overact".[1]

Hering's Law must be considered in conjunction with Sherrington's Law of Muscle action. "When a muscle contracts, its direct antagonist relaxes to an equal extent, allowing smooth movement to take place. When there is underaction of the muscle which persists with time, the unopposed action of its antagonist results in contraction of that muscle".[1] For these reasons both eyes become affected when an ocular palsy occurs.

Therefore following a fourth nerve palsy the initial manifestation of the abovementioned laws is an overaction of the yoke muscle, the

contralateral inferior rectus muscle, overaction of the ipsilateral antagonist inferior oblique muscle, and finally, secondary inhibitional palsy of the contralateral antagonist, the superior rectus muscle. Our understanding of concomitance is that the angle of deviation becomes the same in all directions of gaze, and is due to innervational changes involving the muscles of the non-palsied eye.

METHOD

Thirty-two cases of fourth nerve palsies, 25 acquired, seven congenital or longstanding, seen over the past six years, were reinvestigated to observe any change in muscle sequelae with time.

The term PALSY is used in this study to describe the limitation of movement of the superior oblique muscle. We classify a case as "congenital" when there is no known history of trauma, no neurological disorder or serious illness, and when the patient is not in the age group of possible vascular episodes. For this study the diagnostic procedure was:

- cover tests with and without abnormal head posture at near and distance fixation.
- ocular motility, observed by at least two members of staff.

Particular attention was given to identify any bilateral anomaly of the superior oblique muscle. Cover tests and reversal of diplopia were assessed, on down gaze and to the right and left sides.

- Bielschowsky Head Tilt Test (BHTT).
- Hess Chart.
- Diplopia chart with subjective estimation of image separation noted.
- Angles measured on the synoptophore in the primary position and in the field of action of the affected superior oblique muscle. Measurements were taken with each eye fixing, 20° on up gaze and down gaze, on the affected side.

Measurements can be obtained from the Hess Chart, by calculating the displacement of images at 50 cm. This procedure elicits the ocular motility pattern and is reproducible at a constant distance with constant accommodation.

However, we consider that with longstanding vertical anomalies, the vertical fusion range is often abnormally large,[9] and therefore, that some fusional control may mask the true degree of vertical deviation.

CLASSIFICATION

Knapp[2] classifies cases of primary fourth nerve palsy according to the direction of greatest deviation. His list of six classifications comprises all possibilities, variations and combinations of muscle sequelae, which assists in decisions relating to surgical correction. Scott[3] also describes eight classifications of fourth nerve palsies for the same reasons.

Our clinical observations determined three categories of primary palsy of the superior oblique muscle.

Type 1. Primary palsy of the superior oblique with greatest deviation and separation of images in the area of action of the palsied muscle.

Type 2. Primary palsy of the superior oblique with greatest deviation and separation of images in the area of action of the contralateral antagonist superior rectus muscle.

Type 3. Primary palsy of the superior oblique with any combination and/or variation of sequelae other than that of groups 1 and 2.

FINDINGS

Symptoms: Presenting symptoms in order of frequency were:

- Difficulty with near tasks.
- Diplopia, intermittent or constant.
- Torsion.

As reported by Seaber,[4] and Moore and Stockbridge,[5] torsion as a presenting symptom was found to be a rare occurrence. Only 3% of this series complained of a tilted image.

Bilaterality: Mansour and Reineke,[6] in an extensive literature study, cite 22 authors who found that the incidence of bilateral fourth nerve palsies is low, i.e. only 17% of a total of 895 cases. These statistics support our findings of 9.6% with a bilateral defect, in spite of Jampolsky's comment[7] "that all fourth nerve palsies should be considered bilateral until proven to be otherwise".

However, Keishner[8] makes reference to Urist's

description of cases of bilateral fourth nerve palsies which were masked as a bilateral entity until post operative examination. It then became obvious that there was also a fourth nerve palsy on the other side. Only one case of this study showed this result, although there were no bilateral signs prior to surgery.

Position of maximum deviation: According to the Hess Charts, 43% of this series showed either recovery, concomitance, or greatest limitation in the field of action of the superior oblique muscle.

However, 57% showed an increasing limitation in the field of action of the contralateral superior rectus muscle. Of this group, 67% of Hess Chart findings were confirmed by the synoptophore measurement being greatest in the same area of gaze.

Wright and Hansotia[10] in reviewing 23 cases of fourth nerve palsies relied on the BHTT and diplopia charts only, for diagnosis of the primary palsy. In our experience these tests alone are not conclusive for such a diagnosis. We consider all tests mentioned in this study to be essential. Goodier[11] states that the BHTT as a differential diagnostic test between a superior oblique palsy and a superior rectus palsy of the other eye, can produce misleading results. She reports finding positive responses with primary palsies of the superior rectus muscle, but has relied on the Hess Chart solely for the original diagnosis.

Ninety per cent of this series exhibited a positive BHTT response, as described for diagnosis of a primary superior oblique palsy. Ten per cent of the series gave a negative response, i.e. no increase in the deviation when the head is tilted to either side.

Figure 1 shows a typical case of a subject showing marked contraction of the ipsilateral inferior oblique muscle, and a positive BHTT response.

Clinical observation of ocular movements revealed that 57% showed the greatest deviation in the direction of the contralateral superior rectus muscle. The deviation was most marked when alternate cover test was performed in that field of gaze.

This observation was confirmed by the patient's subjective estimation of image separation in *all* cases. We found this anomaly in both congenital and longstanding cases of fourth nerve palsies.

Four of the original 32 cases had recovered. Of the remaining, six were categorised as type 1, 18 were categorised as type 2, and four as type 3 (three of whom had bilateral palsy).

DISCUSSION

An electromyographic study of paralytic strabismus by Sakuko and Tsutsui[12] has shown loss of relaxation in the direct antagonist during the action of the agonist, i.e. loss of reciprocal innervation.

If this is so, and the findings applied to a superior oblique palsy, then the direct antagonist inferior oblique could become constantly contracted, and would exaggerate the inhibition of its yoke muscle, the superior rectus of the other eye.

Meyer, Ladatscher and Zonis,[13] after biopsying tissue from 10 overacting antagonist inferior oblique muscles (in cases ranging from six-18 years of age), found that most muscles were in different stages of atrophy. "Contraction bands and asymmetrically contracted sarcomeres, disruption of the sarcomere pattern, and alterations of the Z bands were encountered". A control biopsy of a normal non-overacting inferior oblique was also done, taken from the site of an enucleation, and there was no abnormality reported.

Spencer and McNeer[14] also reported structural alterations in overacting inferior oblique muscles.

Other research in the past decade regarding such muscle fibre changes[15-17] adds support to Sakuko and Tsutsui's findings that anatomical changes may occur in an antagonist inferior oblique muscle.[12]

Certainly other authors have recognised that deviation changes can increase in the upper field of gaze with a fourth nerve palsy. Several authors note that there is diagnostic confusion between an isolated primary palsy of the superior rectus and a primary fourth nerve palsy of the other eye.

Duke Elder[18] states "Congenital cases of superior rectus palsies are frequently associated

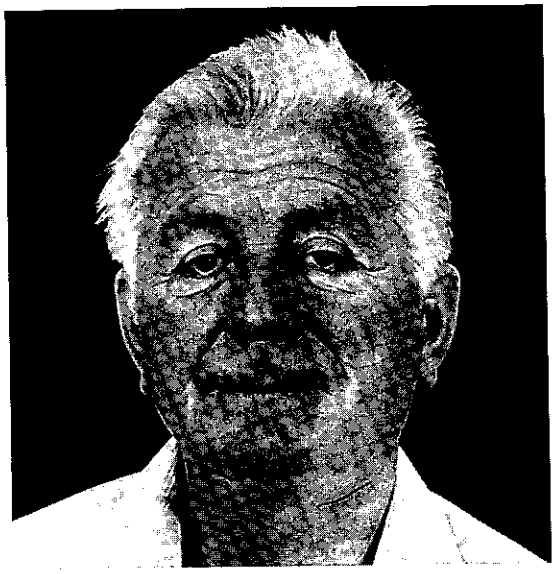

Figure 1 (i): No deviation in the primary position.

Figure 1 (ii): Looking right (fixing left eye).

Figure 1 (iii): Looking right (fixing right eye), showing marked overaction of the left inferior oblique muscle.

Figure 1 (iv): Bielchowsky Head Tilt Test, showing a positive response to a left superior oblique palsy.

with ptosis, usually in the same eye but occasionally in the other. Acquired cases are rare, and when they occur they are usually due to a paresis of the superior division of the oculomotor nerve and are therefore accompanied by ptosis . . ."

We postulate the following reasons for the development of this incomitance in the upper field, following a fourth nerve palsy.

1. From the initial sequelae, innervational changes according to Hering's Law, result in an overaction of the contralateral inferior rectus muscle and those of Sherrington's Law cause inhibition of the contralateral superior rectus muscle.
2. Sherrington's Law results in an overaction of the ipsilateral inferior oblique muscle, which is also advantaged due to decreased tonus of the palsied superior oblique muscle. As a consequence of Hering's Law, the yoke muscle to this inferior oblique is inhibited, i.e. the contralateral superior rectus muscle.
3. The EMG studies and muscle tissue biopsy reports show anatomical changes in the antagonist inferior oblique which result in a constant state of contracture of the muscle. This further exaggerates the inhibition of the contralateral superior resctus muscle.

Therefore, following a fourth nerve palsy, there are resulting significant anatomical and innervational changes to the ipsilateral inferior oblique muscle, which in turn, produce a significant underaction of the contralateral superior rectus muscle.

CONCLUSION

Our observations show that congenital or long-standing fourth nerve palsies are not uncommon, and that the deviation may become greater in the upper field with time. This is found in the field of the contralateral superior rectus muscle, as a result of innervational and anatomical changes of its yoke muscle, the inferior oblique on the originally palsied side. It is important to identify this phenomenon, to eliminate any dilemma that there may be a primary superior rectus palsy.

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BLURRED VISION WITH HEAD MOVEMENTS — TESTING THE EFFICACY OF THE VESTIBULO-OCULAR REFLEX

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Abstract

The vestibulo-ocular reflex (VOR) maintains clear vision via a stable retinal image during head movements. Disorders of the labyrinth, cerebellum or brainstem may reduce the efficiency of this mechanism and patients may present with a main complaint of blurred vision, viz oscillopsia. The clinical test of visual acuity assessment during head movement is outlined and illustrated with a Case Presentation of a subject with a defective VOR. The neuroanatomical substrate is briefly discussed.

Key words: *Blurred vision, vestibular, visual acuity, VOR.*

INTRODUCTION

An interesting clinical dilemma arises on occasion when a patient presents complaining of blurred vision. The reasoning process that ensues is one of a process of elimination and then confirmation. Common aetiologies encountered include: static or dynamic errors of refraction, prismatic effects, sensory media defects such as corneal problems, cataract, retinal distortions or raised intraocular pressure, minimal diplopic separation of images in small angled strabismus, accommodation or convergence anomalies, acquired nystagmus, decompensating heterophoria, tearing, or visual field loss.

The mechanism is not always a purely optical distortion of the retinal image, and the perceptive abilities of patients often label quite distinct disorders such as lack of visual field or closely associated diplopia as blurriness. Visual symptoms resulting from a disturbance of the vestibular system, such as oscillopsia, will often be labelled similarly.[1] Whilst vertigo is an illusory sensation of motion of self or of the environment, oscillopsia is an illusory to and fro movement of the environment. This is often reported as vision becoming blurred with movement of the head or body.[2] The key to diagnosis of a vestibular aetiology relating to a complaint of blurred vision is a history of visual disturbance which only occurs during head movements.[1] Whilst it is important to determine the characteristics of the blur itself, its precipitation and relief features, it is imperative to evaluate its relationship to head and body movements.

THE VESTIBULO-OCULAR REFLEX

The vestibulo-ocular reflex is part of the central nervous system in which connections between the balance sensors within the ear, the brainstem, the cerebellum and the visual system function to stabilise gaze and ensures clear vision during head movements.[3] The cervico-ocular reflex is similarly involved with reflexes in neck and spinal cord movement and the optokinetic ocular reflex is effective in sustained body rotations.[4] At 70 milliseconds, visual retinal processing is too slow to maintain steady images in response to natural rapid head movements, whereas the latency of

the vestibulo-ocular reflex is only 16 milliseconds.[2,4] Total compensation of a prompt slow phase gaze movement to head rotation is not achieved, and in fact would be undesirable due to retinal stabilisation and fading of the image. A horizontal head turn to the right produces an equal eye movement in the orbit to the left, so that the summated movement does not change and the image of the world does not move significantly on the retina. During instances of sighting an object using a combination of eye and head movement in the same direction, this vestibulo-ocular reflex is suppressed, probably by the pursuit system.[2]

The stimulus to the reflex is head movement, which creates a change in the position of the fluid within the balance sensors of the ear. The impulses that are triggered follow a pathway through the brainstem to the extraocular muscles. The balance sensors involved are the labyrinthine apparatus or semicircular canals and otoliths. Impulses are transmitted by the VIII cranial nerve to the VIII cranial nerve nuclei, where communication occurs between both sides of the brainstem. (Figure). After synapsing in the VIII cranial nerve nuclei, impulses travel to the contralateral VI, IV and III cranial nerve nuclei to supply the extraocular muscles. Each semicircular canal within a labyrinth is directly linked with a pair of synergistic extraocular muscles. Simultaneous impulses travel to the cerebellum, which has projections back to the VIII cranial nerve nuclei as a monitoring system. Labyrinthine, brainstem or cerebellum dysfunction may lead to a malfunction of the vestibulo-ocular reflex, and some drugs will also decrease the vestibulo-ocular reflex gain. This gain of eye velocity to head velocity is ideally matched at 1.0.

Clinical or laboratory testing of the vestibulo-ocular reflex can be used to determine if this system is intact, and if it is efficient. Electrodiagnostic techniques, chair rotations or caloric testing (water stimulation of the semicircular canals within the labyrinthine system of the ear) give detailed investigations. Clinical methods include ophthalmoscopy with head movements,[2] watching for corrective saccades[2,4] and testing visual acuity with head movement.[3] Various

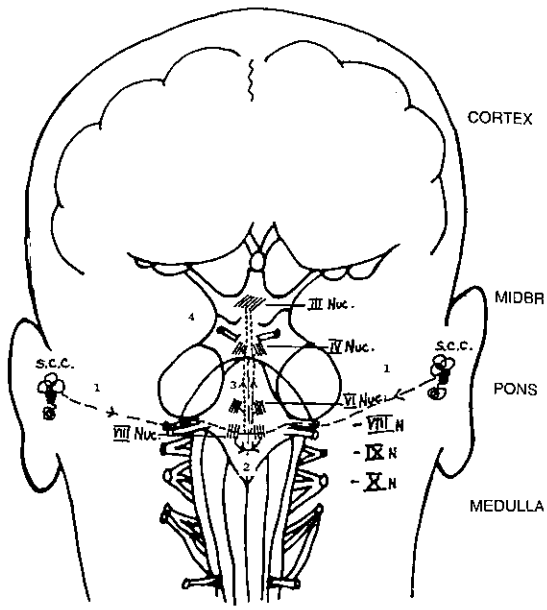

Figure 1: Pathway for the vestibulo-ocular reflex: (1) Stimulus from semicircular canals to VIII cranial nerve nucleus; (2) Communication between both VIII cranial nerve nuclei; (3) Contralateral innervation to VI, IV and III cranial nerve nuclei; (4) Innervation to extraocular muscles.

methods have been postulated for testing vision whilst a patient's head is rapidly turned. The most recent sensitive test shows a monitor with a triggered visual display of square wave gratings at intervals of 50 milliseconds.[5] Whilst this determines the degree of lack of retinal stabilisation as compensated for by the vestibulo-ocular reflex, the clinical test performed by an orthoptist of visual acuity assessment with head movements is sufficient in determining an influence on the visual system by vestibular dysfunction.

METHOD

The clinician firstly ascertains the optimum visual acuity of the patient viewing binocularly. This does not imply binocular single vision, but purely the visual functioning of the patient under normal viewing conditions with both eyes open. Snellen's Charts, Logmar Charts, E Charts[3] or the usual clinical methods may be employed. Having informed the patient of the nature of the

test, place open hands firmly but gently on either side of the head and perform small horizontal oscillations. The patient is to attempt to continue to read their optimum visual acuity levels. If this is not possible, the next best levels are indicated by the patient. The amplitude of the head movement does not need to be large, as it is the movement itself that induces the reflex. The speed of movement should approach that of natural rapid head movements, being two-three cycles per second.[2] If the movement is slower than this, optokinetic or cervico-ocular reflexes may be compensating for vestibular dysfunction and no defect will be seen. At speeds approaching natural head movements, only the vestibulo-ocular reflex is fast enough to generate compensatory eye movements, and if this system is impaired, a defect will be found. This defect will manifest as a reduction in visual acuity during head movements.

Three or more lines decrease in vision levels from head stationary to head movement readings indicate vestibular dysfunction.[3] However, suggestions have been made that any decrease in visual ability due to head movement may be indicative of pathology. Whilst there appears to be a large variability in the efficacy of normal vestibulo-ocular reflexes to compensate for head movements, in order to detect a degradation in vision levels due to retinal smear, the head rotations in normal subjects need to be at speeds three times faster as compared with those suffering vestibular disease.[5] It is therefore unlikely that a head rotation performed in a clinic would be fast enough to elicit poor efficacy in a normal patient, but be of sufficient speed to detect poor compensation as a result of vestibular disease.

The procedure is repeated with vertically induced head movements[1] and the results noted. No additional clinical equipment is required to perform this test and the time taken usually does not exceed 30 seconds.

CASE HISTORY

The following case illustration highlights the value of clinically testing the vestibulo-ocular reflex and shows its significance. MJ, aged 32, presented with a history of closed head injury five months previously, following an assault. Surgical evacuation of a left occipital extradural haematoma and resuscitation from respiratory and cardiac arrest formed part of his previous treatments. Formal eye assessment did not occur until this period of time post injury. He had initially spent one week comatosed with cerebral oedema and moderately dilated ventricles. The patient's main complaint was of blurred vision and diplopia. Visual acuity measured 6/9 in each eye, both improving to 6/5 with a pinhole. Cover testing revealed an intermittent alternating esotropia of the divergence weakness type with small left hypertropia seen with distance fixation. Ocular movements revealed the L/R to be greatest in left gaze positions, being the areas of most troublesome diplopia. Hess charting showed a small left lateral rectus paresis. Vertical saccades and pursuit were intact, but saccades were undershooting on left gaze, and cogwheeling occurred to both sides. Convergence and accommodation were intact and normal.

Pupils were reactive to stimuli of light and accommodation, but the resting state of the left pupil was greater than that of the right. Binocular functions were present and normal. Visual fields of the right eye were full, and a small relative superior arcuate defect was seen for the left eye. Intra ocular pressure was normal. A small congenital posterior lens opacity was seen in the left eye on dilation. Whilst signs appeared to indicate probable partial resolution of ocular paresis(es) and mild disruption to eye movement systems, the signs were not consistent with the symptom of the blurred vision. Further questioning revealed that the problem occurred with movement, as in travelling in a car, and disappeared when the patient was stationary. Results to clinical testing of the vestibulo-ocular reflex showed a decrease in visual acuity from BEO 6/9 with head stationary to BEO 6/60 with head movement.

DISCUSSION

Impairment of the vestibulo-ocular reflex can be quite debilitating. The patient in the case illustrated had a visual reduction from 6/9 to 6/60,

which simultaneously reduced the quality of his life with any head movement. The significance of performing this simple test can be many fold. The most obvious one is the diagnostic value to the clinician in localising a symptomatic visual problem to a vestibular dysfunction. Thereafter, appropriate referral can follow. Patients are also relieved and grateful if they can be told that they have a real and organic problem and do in fact require further investigation. This empathy and reassurance is most therapeutic to the patient. Irrelevant and fruitless investigations will be avoided as outlined elsewhere,[6] thereby reducing patient anxiety, discomfort and expense. Differentiation is aided, in the medico-legal sense, between a recent or longstanding problem. The onset of the symptoms will be quite abrupt, the timing well described by the patient and the reduction in vision seen easily with head movement testing. Orthoptic reporting will be made more comprehensive. The sequelae of vestibular problems may be that compensation occurs or that a level of stability is reached and maintained without further adaptation. In cases following vestibular neurectomy, clinical compensation was no further improved after one year as compared to one week post-operative.[4] Compensation may be similar in other disease processes. Visual therapies may not be directly applicable, but awareness of the problem by the patient may encourage some adaptive procedures. Improved knowledge in testing procedures and their underlying bases will benefit discussions between orthoptists and the relevant specialists involved, such as neurologists, ear, nose and throat specialists and ophthalmologists.

SUMMARY

When confronted with a history of blurred vision with head movement, the testing of the efficacy of the vestibulo-ocular reflex to prevent retinal slip during head movement can be performed during an assessment of the integrity of ocular muscle movement and the eye movement systems. It is a test that is very quick to perform, requires no additional clinical equipment and provides significant information on the relationship of the vestibular system to the ocular system, and can indicate when referral for further investigation may be required.

ACKNOWLEDGEMENTS

Thanks are extended to Dr Justin O'Day for introduction to this clinical test, Dr Michael Halmagyi for his information and background discussions, Dr B. D. Cooke for access to patients at the Eye Clinic of the Austin Hospital, Melbourne and Ms Helen Williams for assistance in collating and typing the manuscript.

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UNIOCULAR CENTRAL FIELD LOSS: A CASE STUDY

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WILSON HERIOT, FRACS, FRACO

Abstract

A sixty year old male is presented with unilateral central field loss and severely reduced stereoacuity who was taught to view eccentrically in an attempt to improve his stereoacuity. Although stereoacuity was improved by this treatment it also resulted in a loss of binocular visual acuity.
Key words: *Eccentric viewing, stereoacuity, stereopsis.*

Stereopsis is the result of the stimulation by dissimilar images of slightly disparate retinal images. The two images appear as one and are appreciated with depth. The ability to fuse the images is dependent upon the retinal points being located within Panum's area, where each retinal area has correspondence with a similar area in the contralateral retina. It is a complex entity which will vary with the distance of the object to the observer, and with the length of exposure to the stimulus. Monocular cues may also be used to imply depth perception.

The phenomenon of stereopsis is often associated with the central retina and is assumed to be absent with the absence of central retinal function. Recently a dual stereoscopic system has been suggested with a means of assessment which will distinguish between 'global' or peripheral, fusion with stereopsis; an entity which has been clinically observed, and central fusion, with high grade stereoacuity.[1]

If stereoacuity, as with visual acuity, is most sensitive within the central retina, then unilateral central field loss could be expected to severely reduce stereoacuity, with binocular visual acuity remaining at the level of the unaffected eye. As stereopsis can be demonstrated from both central and peripheral areas, it is reasonable to suggest that, if such a person could be taught to view eccentrically, then some level of stereopsis could be restored.

CASE STUDY

A.D., a sixty year old male, presented with a juxta foveolar macular hole in the left eye.

The condition was not considered to be progressive. Functionally he was not concerned with the area of field loss except for the marked loss of stereoacuity. The nature of his employment required very fine levels of stereoacuity, and he was now unable to perform many work tasks.

His visual acuity in the right eye was 6/6, central acuity in the left eye was 6/60, however

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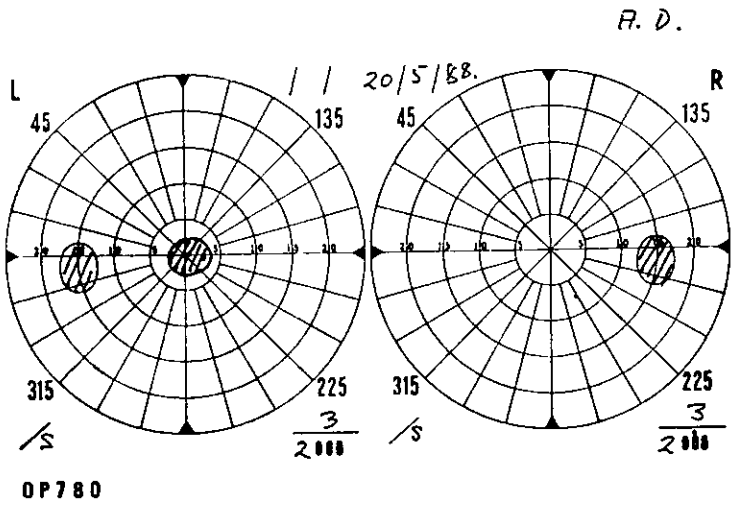

Figure 1: Visual field showing a 3° central scotoma in the left eye.

this could be improved to 6/18 by viewing 3° eccentrically to the right. The visual field of left eye (see Figure 1) showed a 3° central scotoma. His stereoacuity, as measured on the Titmus test was to 1,000", but was not demonstrable on either the Frisby or the TNO tests.

AD was taught to eccentrically view by occluding the right eye as, initially, the desire for central fixation was too strong to permit eccentric viewing with both eyes open. Alignment of the left eye eccentrically was practised during occlusion until, after several treatments, eccentric viewing was able to be maintained when the occlusion was removed. This practice was continued for five sessions of 30-45 minutes each. At the completion of the fifth session stereoacuity could be measured to 200" on the Titmus test, 440" on the Frisby test and 240" on the TNO test.

DISCUSSION

Disruption of the central 3° of retina was sufficient to grossly reduce this man's stereoacuity. Training in eccentric viewing subsequently showed that his stereopsis could be substantially improved. Functionally, however, this improvement was less significant as the level of visual acuity correspondingly decreased from 6/6 to 6/18, and he did not find the gain in stereoacuity at the expense of visual acuity to be worthwhile. It is of interest to note that acceptable levels of stereoacuity can be achieved within 5° of central field. This has implications in cases of bilateral central field loss which have not been previously considered.

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DUANE'S RETRACTION SYNDROME — A REVIEW OF 39 CASES

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Abstract

Thirty nine cases of Duane's Retraction Syndrome who presented to the Eye Clinic at the Children's Hospital over a 12 month period are reviewed. Many of their features are consistent with those of previous authors, although, there was a high incidence of associated abnormalities, which may be explained by the specialist paediatric setting in which they were seen.

Key words: *Paediatrics, associated anomalies, aetiology, lateral rectus.*

Duane's Retraction Syndrome is not a rare condition, and its forms may be as varied as its reported aetiology. It was described by Stilling in 1887 and Turk in 1896. Duane in 1905 provided a more complete definition; and there are currently varied explanations as to its cause.

It was originally described as a musculofascial anomaly, being thought to be due to replacement of normal contractile substance within the lateral rectus with fibrous tissue. Strachan[1] describes increased stiffness in the forced duction test of the lateral rectus of all 15 cases examined. An inability of the lateral rectus to generate a normal force pattern was also reported, lending support to the early theory of fibrotic muscle tissue.

Burian and Von Noorden[2] postulate that it is the result of an innervational disturbance of nuclear or supranuclear origin, with abnormal substitute innervation of the paresed lateral rectus occurring via a branch of the third cranial nerve.

More recent study based on electromyography by Prieto-Diaz suggests the main feature of the syndrome is co-contraction of the horizontal eye muscles.[3] Electromyography distinguishes the normal firing of the medial rectus from the abnormal innervation of the lateral rectus, which show silence on abduction but firing on adduction. Co-contraction of the lateral rectus and, at times co-contraction of the superior or inferior rectus when the patient attempts adduction, produce the retraction of the globe and the palpebral fissure narrowing. Kommerell and Bach[4] believe that the co-contraction occasionally of superior and inferior recti leads to the noted 'A', 'V' and 'X' patterns associated with Duane's syndrome. A case of twitch abduction on attempted upgaze has been reported as a new type of Duane's syndrome with 'V' incomitance; it is thought to be similar to abducting twitches on attempted up/down saccades which can occur following aberrant regeneration after a third nerve palsy. Kommerell and Bach's explanation for this is that the burst impulse in nerve fibres misdirected from a vertical to the medial rectus, pulls the eye away from its direct vertical trajectory.

Retraction is often inconspicuous and may be revealed only by electromyography, as discussed by Huber, Esslen, Kloti and Martenet.[5] Some

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cases described as a sixth cranial nerve palsy are quite possibly Duane's syndrome. In most cases of Duane's, the absence of innervational input to the lateral rectus when the patient attempts abduction, is the cause of that inability to abduct. (It must be noted too, that part of the inability to abduct may be due to restriction which is secondary to a stiff medial rectus.)

Hotchkiss et al describe a clinicopathological study of a case of bilateral Duane's syndrome where the intracranial and orbital pathology show absence of both abducens nuclei and nerves,[6] with the lateral recti being partially innervated by branches of the third cranial nerves. Similarly, Miller describes a unilateral case where the lateral rectus was innervated by the inferior branch of the third cranial nerve; there was an absence of the sixth cranial nerve and no cell bodies of motor neurons in the sixth nerve nucleus.[7]

The diverse anomalies associated with Duane's syndrome (Pfaffenbach, Cross and Kearns,[8] Ramsay and Taylor,[9] O'Malley, Helveston and Ellis[10]) suggest that Duane's syndrome may be of teratogenic origin, with the teratogenic stimulus occurring toward the middle of the first trimester of pregnancy. The congenital anomalies of ear deformity, deafness, facial hypoplasia, facial paresis and thalidomide embryopathy which have appeared concurrently with Duane's syndrome, share a common critical time of embryological development. If Duane's syndrome could be of teratogenic origin then the presence of crocodile tears in some cases could shed light on the site of the lesion.

Thirty nine cases of Duane's syndrome presented to the Eye Clinic at The Children's Hospital over a 12 month period from 1987-1988. Of these, 44% were male, and 56% were female.

Involvement of the left eye only occurred in 61% of cases, the right eye only in 18%, 21% were bilateral. Seventy seven per cent were classified as Type 1, 10% Type 2, and 13% Type 3 (according to Duane's original classification). These findings are consistent with other published studies, as can be seen in Table 1.

In this series a strabismus (most commonly an intermittent esotropia) was the most common reason for presentation, occurring in 41% of cases. Other reasons included 'poor eye movement' (23%), referral for suspected sixth cranial nerve palsy (10%), apparent ptosis (12%), and pain on eye movement, frequent blinking, blocked tear duct and presentation for a general check up due to a family history of unrelated eye problems.

Duane's syndrome usually occurs sporadically but it has been reported that there is some family history in about 2% of cases.[10] In this series there was one case where a male infant's father also had the syndrome. Another female child had one male sibling with the Märcus Gunn Jaw Winking Syndrome and another male sibling with a intermittent exotropia and a significant 'V' pattern. Their parents were first cousins.

The syndrome is a benign entity and treatment is not usually required. Seventy four per cent of this series had good binocularity, usually with a slight compensatory head posture (77% of the whole group had such a head posture). Of the group, 59% were orthophoric or had a well controlled heterophoria, 13% had intermittent esotropia, 3% intermittent hypertropia, 21% constant esotropia and 5% constant exotropia. O'Malley, Helveston and Ellis[10] reported

TABLE
Comparative Incidences (in %) of Features Associated with Duane's Retraction Syndrome

	Right Eye only	Left Eye only	Bilateral	Male	Female	Type		
						I	II	III
This Study	18	61	21	43	56	77	10	13
O'Malley, Helveston and Ellis[1]	16	66	18	48	62	87	11	2
Parks[13]	22	60	18	35	65			
Pfaffenbach,[8] Cross and Kearns	22	60	19	43	57			

'orthotropia' in 31% of their cases, esotropia in 53% and exotropia in 16%.

In this series, 23.1% required correction of some anisometropia and/or astigmatism and in 89% of these cases the greater refractive error was found in the eye affected by Duane's syndrome. Tredici and Von Noorden in a series of 72 cases of Duane's syndrome found 17% had anisometropia of greater than one diopter,[11] and O'Malley et al found a 16% prevalence of the same.[10] Consideration must, however, be given to the approximate 10% of the normal population having anisometropia of greater than one diopter.[12]

One third required some part-time occlusion for amblyopia. Of these, 46% had strabismic amblyopia; 8% refractive amblyopia; 31% a combination of strabismic and refractive amblyopia; and 15% stimulus deprivation amblyopia due to ptosis.

Cosmetic surgery was required in 18% of cases, this was generally to reduce an abnormal head posture. Eight per cent required and responded well to convergence insufficiency treatment.

Duane's syndrome may co-occur with other syndromes. The Klippel Feil Syndrome is said to occur in 3-4% of cases and Congenital Labyrinthine deafness in 7.5-11%.[8,13] When these problems occur together with Duane's syndrome it is called Cervico Oculo Acoustics Syndrome or Wildervanck syndrome. Similarly, Goldenhar's Syndrome may co-occur with Duane's syndrome,[13] and cases of thalidomide embyopathy have also been described by Papst.[14] A rarer co-occurrence is that of the gusto-lacrimal reflex (crocodile tears).[9]

Other ocular conditions such as optic nerve hypoplasia,[15] coloboma of the fundus, iris and lids, pupillary anomalies, persistent hyaloid arteries, cataract[2] and Marcus Gunn Jaw Winking[16] have also been reported in conjunction with Duane's syndrome.

Two of the cases in this series had Goldenhar's syndrome, one with cerebral palsy, deafness and facial hypoplasia, and the other with hydrocephalus, facial hypoplasia, dextrocardia, transverse liver, the left main bronchus on the right side and a malpositioned right ear. One child had middle ear malformation, and another, with bilateral Duane's had developmental delay, curvature of the spine and butterfly vertebrae. One case had neurofibromatosis. There were also one case each of Henoch-Schoelien Syndrome and Congenital Adrenal Hyperplasia.

Ptosis, 'A' and 'V' patterns, convergence weakness, latent nystagmus, chalazion and micropthalmos were also noted.

The high incidence of associated anomalies in this series is no doubt explained by the specialist nature of The Children's Hospital, which deals with the whole range of paediatric medicine. However, it does indicate the need for a thorough examination of any child presenting with this syndrome

ACKNOWLEDGEMENTS

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EARLY CHILDHOOD SCREENING

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Abstract

A screening programme at Early Childhood Centres has resulted in 12% of children seen by the orthoptist at the request of the nursing staff being referred for further ophthalmic consultation. The reasons for referral included squint, suspected reduced visual acuity, ocular muscle imbalance, external pathology or a significant family history of eye problems.

Key words: *Amblyopia, strabismus, infancy, screening.*

The early detection of ocular abnormalities is recognised as being crucial for early intervention of treatment if return to normal or near normal function is to be attained. This approach has been reinforced by the acknowledgement of a critical period for visual development.

Screening programmes have proven their validity in the detection of abnormalities. There is considerable debate pertaining to the most acceptable age at which vision screening should be performed. Much of the literature is related to screening children between the ages of three to five years. This in part, is due to it being the earliest age at which a reliable and accurate visual assessment can be performed (in a screening capacity) as the children are more co-operative, and better able to understand more accurate vision testing techniques.

Screening programmes have shown success in the detection of visual acuity defects, the referral rate is high, and indicates the competency and capability of the nurses conducting the assessments. There is, however, documentation of under-referral of strabismus. MacFarlane, Fitzgerald and Stark,[1] in reporting Queensland school screening in 1987, showed that approximately 70% of children with manifest strabismus would have been detected on the basis of decreased visual acuity. There was a 28% under-referral rate, and this was attributed to the reduced detection of manifest strabismus on cover test.

The presence of nursing staff carrying out screening programmes is essential, as they provide an invaluable service, however, assistance is needed in the detection of strabismus. It is the detection of strabismus which is of major importance in those children aged from birth to three years. A severe and untractable amplyopia is that associated with unilateral strabismus of early onset. It is logical to assume that the earlier the intervention, the better the prognosis for achieving and maintaining a near normal standard of vision.

Newmann, Friedmann and Abel-Peleg[2] in 1987, clearly demonstrated the importance of early detection and treatment of strabismus. The study comprised 78 children. This group was

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divided into two groups: those who had commencement of treatment prior to two years of age and those where treatment started after this age. Treatment included prescription of glasses is necessary, occlusion or penalisation of the fixing eye and corrective surgery if warranted. This treatment continued to the age of six to eight years. Results showed better end result vision if treatment commenced prior to two years of age.

A screening programme to Early Childhood Centres was commenced in 1988 by the Orthoptic Department of the Children's Hospital, Camperdown with the co-operation of the clinic sisters at these Centres. The programme was initiated as a result of frequent referrals to the Eye Clinic for infants suspected of having strabismus. On examination, however, a large proportion of these children were found to have pseudosquint related to epicanthal folds or facial asymmetry. The nursing staff were overwhelmingly enthusiastic to the proposal of assistance. It was also important that ongoing education be integrated into the programme, with the aim of educating the clinic sisters in the detection of ocular anomalies.

During each screening session, if time allows, the clinic sisters observe the examination and gain insight as to what procedures they can apply to their own assessments.

Within each screening area, there are four base clinics which are located for easy accessibility to parents of three to four surrounding clinics. The objective of the programme being to take the service to the people, and the success of this approach is reflected in the high attendance rate of 84% of eligible children.

Developmental assessment of infants at Childhood Centres is at one month, six weeks, three and six months of age. At these visits, visual performance is also assessed. If the clinic sister feels that the responses are developmentally inappropriate, the children are referred to the orthoptic clinic (these referrals usually result from poor visual attentiveness, asymmetrical corneal reflections, abnormal ocular motility or a family history of strabismus). Any obvious ocular abnormality is referred immediately for ophthalmological investigation.

The aim of the orthoptic examination is to not necessarily give a quantitative evaluation of an infants vision, but to qualitatively assess that vision is present and functioning adequately, and that there are no barriers to impede normal visual development such as squint or ptosis.

Orthoptic assessment includes cover test at 1/3 metre, three metres or six metres, visual acuity, binocular status, convergence, ocular movements and pupillary responses.

Following orthoptic assessment, referral to an ophthalmologist is advised if any of the following criteria are found: intermittent/manifest strabismus, reduced or unequal visual acuity, objection to occlusion of one eye, ocular muscle imbalance, family history of ocular pathology, "At Risk" premature infants without review or external ocular pathology.

In eight months of service, the programme has seen the assessment of 298 children aged between three months and five years, 68% were less than 12 months of age, 21% aged between 12 months and 3 years, 10% aged three to five years. Of those children seen, 12% were referred for further ophthalmological consultation (see Table).

The incidence of squint in a screening population is documented as ranging from 0.5% to 5%. The RAHC figure of 4% is comparatively high, however, the children included in this study are not indicative of a random sample, as those assessed were either suspected of having a squint or had a pre-disposition to ocular problems through their family history.

Of the 12 children referred due to the presence

TABLE
Ocular Anomalies Detected

	No	% of General Pop.
Constant/intermittent strabismus	12	4%
Ptosis	8	2.6%
Reduced visual acuity	4	1.2%
Family history	4	1.2%
Anisocoria	3	1%
Facial asymmetry	2	0.6%
Photophobia	2	0.6%
External punctum	1	0.3%

of strabismus, the majority (nine) were from the "less than 12 month old" age group. Five of these had constant esotropia, one three month old child had an intermittent esotropia, and three had intermitten exotropia. This particular deviation was masked by the epicanthal folds, and, as a result presented to the clinic as suspected esotropia.

Four children were referred for reduced visual acuity — all were aged between three and five years.

Orthoptic screening, as with any screening programme, does have its limitations, especially with regard to detection of refractive error. With the current screening programme, we are only able to successfully detect ametropia in those children of verbal age. We rely on more gross methods of visual assessment which only indicate significant deprivation in infants. With the advent of video-refraction and more portable methods of preferential looking techniques, it is hoped that this hurdle will be crossed, thereby making orthoptic screening of infants a more comprehensive examination.

This programme has proven to be an effective means of detecting ocular abnormalities, particularly strabismus, and particularly in the younger age groups.

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ABSTRACTS OF STUDENTS PAPERS

N.S.W.

The following are abstracts of research papers by third year orthoptic students at Cumberland College of Health Sciences, N.S.W. Copies of particular papers of interest may be obtained by writing to:

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IS THERE A ROLE FOR THE ORTHOPTIST IN THE DIAGNOSIS INTERVENTION AND TREATMENT OF OCULAR PROBLEMS IN THE ELDERLY UNDERGOING REHABILITATION? — **Katherine Ardzejewska**

The importance of an Orthoptic assessment in patients with cerebro-vascular disease prior to rehabilitation has long been established. Twenty elderly patients were screened at Governor Phillip Hospital. Sixty-five per cent of the sample had ocular problems that had been previously undetected. Seventy-seven per cent of these went on to receive benefit from intervention and or therapy, supporting the value of an orthoptic role in the diagnosis and rehabilitation of CVA patients.

A STUDY OF THE EFFECT OF OPERANT CONDITIONING BEHAVIOUR SHAPING ON PRISM BAR CONVERGENCE — **Suzann Farrelley**

The effectiveness of the application of Behaviour Shaping in facilitating improvement whilst training fusional convergence by the prism bar method was investigated in 16 subjects who had received no previous experience with prism bar fusion training. This was studied by dividing Behaviour Shaping into its components of Instruction and Positive Reinforcement and investigating which factor provided greatest improvement.

The effect of prior experience to prism bar fusion training was also investigated, by comparing Naive and Experienced groups.

It was found that instruction provided a higher standard of subject improvement than positive reinforcement, and that by providing instruction prior to positive reinforcement a higher standard of improvement was achieved.

COLOUR VISION DEFECTS IN CHRONIC OPEN ANGLE GLAUCOMA — **Nicolette Braaksma Kathryn McKay**

Acquired colour vision defects were investigated in 17 subjects with Chronic Open Angle Glaucoma (COAG). Both the City University and Farnsworth Munsell 100 Hue (F.M. 100) tests were used to determine which test was more effective. Testing procedures were standardized with all tests being performed upon a black background, using illumination from a 60 watt daylight blue globe. The order of presentation for the plates on the City University and F.M. boxes was randomised.

Colour vision defects were compared with established age related norms for the F.M. 100 test. It was revealed that 66% of abnormalities detected were age induced artifacts. The remaining 34% showed either a Tritanous or All Over Colour depression. There is no significant difference between the City University and F.M. 100 tests in detection of colour vision defects in subjects with COAG. No significant relationship was revealed between the subjects' Cup Disc ratio and defects detected by either test.

A COMPARISON BETWEEN THREE DIFFERENT METHODS WHEN TESTING VISUAL ACUITY IN NYSTAGMUS PATIENTS — **Gillian Enright**

Three different methods of testing the Visual Acuity in patients who had latent nystagmus or manifest nystagmus with a latent component were compared. The three tests included clear contact paper, a +5.00 diopter lens and an opaque occluder. The +5.00 diopter lens was found to give the best overall results.

THE CAMBRIDGE LOW CONTRAST GRADINGS — HOW SUITABLE FOR CHILDREN? — **Anita Petrovic**

Measuring contrast sensitivity is an important parameter of visual function, yet it is still largely ignored. It has been found to be a much more sensitive indicator of ocular disease than other methods currently in use. It has also proved a valuable tool in the assessment and monitoring of childhood amblyopia.

This study is aimed at discerning how suitable the Cambridge Low Contrast Gradings are for testing children. Forty-five children between the ages of three and ten years inclusive were tested with this new method. All subjects had normal visual acuity according to the Snellen Chart. Forty (88.9%) of the children were able to complete the test satisfactorily indicating that the test is suitable for children.

THE CHRONIC FATIGUE SYNDROME — WHAT ARE THE OCULAR SIGNS? — **Kathryn Radford**

Chronic Fatigue Syndrome (CFS) is a condition with

no known aetiology or treatment. Sufferers develop a variety of symptoms, especially profound lethargy and gross fatigability. Although there have been several references to ocular symptoms no study has investigated whether there are any specific ocular defects. A sample of fifteen CFS patients were given a full orthoptic investigation. The study revealed specific ocular defects in the areas of saccades, accommodation, convergence, absolute fusional amplitudes and pupil reactions. Future research knowledge gained from these areas may aid understanding of the progression of the condition and influence future ocular treatment. Knowledge of ocular symptoms may assist diagnosis in a previously undiagnosed person attending an eye clinic with ocular symptoms.

THE EFFECT OF CATARACT EXTRACTION AND INTRA-OCULAR LENS IMPLANT ON THE PRE-EXISTING DEVIATION — **Maria Repko**

The near and distance deviation was measured in twenty two subjects undergoing cataract extraction and intraocular lens implant surgery. Measurements were taken on the last pre-operative visit and the first post-operative visit using the prism bar cover test method.

The visual acuity was noted pre-operatively with one of the stipulations for inclusion in the study being that acuity was not less than 6/60 in the worse eye. It was found that there was a statistically significant change in the size of the heterophoria and the rate of recovery of the heterophoria improved in 64% of the subjects.

SACCADIC EYE MOVEMENTS: EVALUATION OF FIVE DIFFERENT TESTING PROCEDURES — **Joelle Gaeta**

In a clinical situation saccadic eye movements are tested routinely. The aim of this study was to determine whether a saccadic defect found on testing with visual targets can be reproduced by using different methods, and to compare the saccadic response to different stimuli in patients with specific visual defects.

Seventy five subjects, 50 of whom had recently suffered a cerebro vascular accident, and 25 'normals' matched for age, were assessed. Individuals performed saccades on five different tasks using: (1) visual targets, (2) examiners command, (3) auditory targets, (4) tactile cues, (5) OKN.

Defective saccades were more commonly detected in response to visual targets, and rarely did any other tests detect defects where none were found on visual targets.

RESPONSE OF MICROTROPES TO THE LANG STEREOTEST — **Kyaran Butler**

The aim of the study was to investigate the clinical characteristics of microtropia that determines its response to the Lang Stereotest. The tests performed on 36 cases of microtropia included evaluation of refractive error, distance vision, near cover test, near prism cover test, visuscope and the Lang Stereotest.

The results indicated that there is a positive response to the Lang Stereotest if the amount of anisometropia is greater than 0.5D and the subject has a low level of ametropia, and a negative response if the amount of anisometropia is les than 0.05D and the subject has a high level of ametropia.

KEEPING AN EYE ON THE MOTOR REGISTRY — **Dolly Nanda and Kalliopi Pilatos**

Testing procedures currently used by the Department of Motor Transport only assess visual acuity and do not adequately detect those with other visual deficits. This study indicated that the majority of people who had a visual defect were aware of their visual parameters or had some idea that a problem existed, but had not declared this on renewal of their licences. It is suggested that safe driving skills can be promoted by modifying the requirements and testing procedures currently used by the DMT.

COMPARISON BETWEEN THE MADDOX WING AND THE PRISM BAR COVER TEST IN MEASUREMENT OF HORIZONTAL HETEROPHORIA — **Katarzyna Stepniewska**

Clinical measurements from the Maddox Wing and Prism Bar Cover Tests have often been questioned. In this study of 42 patients the difference between the size of horizontal heterophoria using each method has proved to be statistically insignificant.

CONTRAST SENSITIVITY IN INTERMITTENT DIVERGENT SQUINT OF DIVERGENCE EXCESS TYPE — **Anne Davis**

Traditionally, the cause of many cases of intermittent distant extropia has been attributed to glare, or excess light, resulting in the classification 'sunlight deviations'. As yet, no physiological reason has been established for this type of strabismus. The aim of this study was to see if there is a relationship between contrast sensitivity and decompensation of intermittent distant exotropia.

The Vistech Contrast Test System was used monocularly and binocularly at 33 cm and 3 metres under suitable illumination. The subjects generally had lower sensitivity to contrast than a normal subject. Deficits were particularly noticed in the middle frequency range. Contrast sensitivity for distance was found to improve slightly when tested binocularly.

CONTRAST SENSITIVITY, VISTECH VERSUS AMERICAN OPTICAL SYSTEM — **Ngaire Stubbs**

The purpose of this study was to determine which

contrast sensitivity test, the American Optical Contrast System or the Vistech 6000, was the most effective in detecting contrast sensitivity variations in normal subjects. The tests were administered to 29 subjects who had normal visual acuity, and were free from ocular pathology. More 'defects' (according to the tests' guidelines) were found using the American Optical system (52% of all subjects tested) compared with the Vistech 6000 (25%). Thirty six per cent of the subjects were found to have a defect on either test.

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