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CONTENTS

ASSOCIATE PROFESSOR IN ORTHOPTICS

In February, 1991, the appointment was announced of Elaine Cornell, DOBA Dip App Sc (Cumb) MA (Macq), to the newly created position of Associate Professor of Orthoptics at Cumberland College of Health Sciences, The University of Sydney.

Her achievement in gaining this selection has brought credit not only to herself but to the profession in Australia.

Born and educated in Australia, Elaine was awarded the John Pockley prize, for the top student in NSW and Victoria, on completing her orthoptic studies in Victoria in 1964.

Her clinical experience in Australia and overseas has been wide and academically she has had a long association with both the Victorian School of Orthoptics, including Head of School 1970-74, and with Cumberland College of Health Sciences. At CCHS she was first appointed lecturer in the School of Orthoptics in 1977 and became a Senior lecturer in 1990. The 9 months spent on secondment to the Community Relations Division to develop the Alumni Branch gave her broader administrative and communication experience within the College.

She has been closely involved in assisting the development of the orthoptic student training and conversion courses as the course was upgraded by stages first to an Associate Diploma (1976), then to a Diploma of Applied Sciences (1981) and finally to a Bachelor of Applied Science (Orthoptics) with an Honours programme. She has also been involved in Continuing Education and Post-graduate Education programmes for both Orthoptics and other professions.

During this time she has published extensively and obtained grants for a number of research projects. She has presented many papers at professional conferences conducted by the OAA, IOA and RACO, including her presentation as the OAA invited lecturer for the Patricia Lance Lecture in 1988. Her special interest has been in neuro-ophthalmology.

In more recent years Elaine has been a member of a select team from CCHS conducting work-

shops in Singapore, Indonesia and Fiji and in 1990 was the WHO consultant to Beijing on visual disability rehabilitation.

Such activities clearly demonstrate her academic leadership in teaching and research programmes. Her new academic status has also led to her appointment as Head of the School of Orthoptics at CCHS.

As a member of the Orthoptic Association of Australia Elaine has contributed to our professional body in many capacities including that of Honorary Treasurer, Editor and State and School representative. Currently she is President of the OAA, NSW Branch, and a NSW State representative on the OAA Council and Orthoptic Board of Australia.

The Association is indeed proud of her appointment to a level which is of similar standing to only 2 other such appointments overseas, both in the USA, and which is certainly a first in Australia.

Margaret Doyle

Australian Orthoptic Journal 1991; 27: 1-11

The Patricia Lance Lecture, Melbourne 1990

FIVE YEARS OF TINTED LENSES FOR READING DISABILITY

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University of Sydney, Dept Clinical Ophthalmology Save Sight and Eye Health Institute

The Patricia Lance Lecture is fitting recognition for all the work Miss Lance has done for Orthoptics both in Australia and overseas.

Miss Lance instilled in all her students the importance of the need for clinical research, and I hope that this presentation will live up to the standards she encouraged in us.

I am very honoured to be the first of Miss Lance's students to be asked by the Council of the OAA to present the Patricia Lance Lecture and I am going to discuss my research into the use of tinted lenses in the treatment of reading disability.

Tinted lenses were introduced to Australia in 1985 by Helen Irlen, a psychologist from the USA.

As there was no scientific backing for the therapy and no clinical evidence supporting Irlen's claims of a cure for reading disability, many thought that the treatment may die a natural death like so many of the other miracle cures for reading disability that have appeared over the years.

It would appear however that the reverse has happened. Irlen clinics have been established, under a franchise arrangement, in every capital city in Australia as well as in a number of country areas. Irlen clinics have also been established in centres throughout the world, (including New Zealand, UK, USA, Hong Kong, Canada and the Netherlands) once again, under a franchise arrangement.

Although testing of Irlen lenses has revealed that they are Solar CR39 lenses tinted with commercially available dyes there is an inordinate expense involved in their prescription. According to a report from the Australian Institute of Health in February, 1990[1] the charges being levied by the Irlen franchises for consultation and lenses are as follows:

First hour — $65 to $75
Second hour — $65 to $75.

During the first hour the patients are screened to assess their eligibility for tinted lenses. If they are eligible they return for a second one hour visit to have the tint colour and strength established. To have the tinted lenses made up it costs $60 for plano lenses or $105 for prescription lenses.

According to Irlen, to be eligible to be helped by Irlen lenses the patients must have 'scotopic sensitivity syndrome' which is a specific visual dysfunction associated with sensitivity of white light and a preference for certain wavelength bands. Irlen states that this sensitivity and thus reading ability depends on an interaction between rods and cones.[2]

The terminology is misleading as the scotopic system is not used when reading. Also, as we read in photopic conditions the rod receptors are relatively inactive, thus no interaction between rods

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and cones is necessary for reading. Irlen's explanation of a rod cone interaction is meaningless.

Irlen claims that her lenses filter out the narrow band of light which causes the visual symptoms commonly associated with reading disability. Analysis of the lenses revealed that, as expected, they did not entirely filter out any wavelength of light.[3]

Irlen also claimed that "...by filtering out the offending wavelength the lenses improve contrast and make the print clearer and bigger".[4] Results of my research, which will be discussed later in this paper, do not support Irlen's claim that tinted lenses improve the clarity.

The claim that tinted lenses make the print bigger has only been substantiated in cases where a plus correction has been added to the lenses.

Since 1985 I have been involved in a number of studies aimed at assessing the effect of tinted lenses on vision and contrast sensitivity in normal and reading disabled children.

In the first study conducted in Adelaide as part of the Speld South Australia tinted lens study group I tested a group of reading disabled children with and without tinted lenses.[5] These children also had their reading assessed by the remedial teacher at the Flinders Medical Centre. The results of this study demonstrated that reading did not improve in any of these children after three or six months of tinted lens wear.[6,7]

At the same time a number of other studies reported on the effect of tinted lenses on reading ability. Some authors reported that tinted lenses did not alter reading ability[6-10] while others reported that they did.[2,11-17] One paper reported mixed results.[18] The papers with the positive results usually attempted to give a reason for their findings. Two papers reported that the tinted lenses had improved the patients' ability to see. This intrigued me as neither of these papers formerly assessed visual acuity. All the reports were based on subjective comments from the patients.[12,16]

Our study was the only one to assess vision. I found that visual acuity and contrast sensitivity were not altered by the use of tinted lenses. Thus there was no quantitative change in vision.[19]

In a subsequent study I conducted in Sydney, vision and contrast sensitivity were assessed on 325 primary school children with and without various colours and strengths of tinted lenses.[6,20]

The results of this study demonstrated once again that vision and contrast sensitivity were not altered in normals with the use of tinted lenses irrespective of the tint colour or the strength of the tint used.

In both the Sydney and Adelaide studies the Vistech Vision Contrast Test System (VCTS) 6000 at 1/3m and 6500 at 3m were used thus contrast sensitivity was assessed at 1.5, 3, 6, 12 and 18 cycles per degree (cpd). The high spatial frequency (narrow stripes), 12 and 18 cpd represent vision needed for reading. The wider bands are commonly associated with more gross visual tasks such as face and object recognition.

The results of my first two studies provided no visual evidence that would explain why tinted lenses should improve the ability to see print on a page. As a result I began to strongly suspect that the lenses may have been causing a placebo effect or that there was a 'visual preference' effect at work.[6,21]

The placebo effect of the tinted lenses was that wearing tinted lenses gave the children a REASON or excuse for their inability to read and the tinted lenses acted as an attention factor. This, in turn, boosted their self esteem and gave them a reason to try to learn to read. Some of the literature both for and against tinted lenses also commented on the possible placebo effect.[12,16,18,22]

Visual preference could also be a factor. Almost everybody has a preference for looking at the world through one or other colour and strength of tint. The tinted lenses alter the appearance of objects and it is possible that this alteration could be MISINTERPRETED as an improvement in vision. Wilsher and Taylor[22] suggested that dyslexics should differentiate visual preference and other psychological factors affecting the way they see the page from actual improvement in visual performance.

Despite my feelings about the placebo and visual preference effects of the tinted lenses I was still curious because discussions with so many of

the tinted lens wearers and their parents revealed that they were convinced that tinted lenses improved things (without necessarily affecting their reading ability in many cases). I was not certain if a placebo effect could be so widespread. As a result I decided to look at the question of why we traditionally used tinted lenses or sunglasses, what their benefits were, and if they really affected our ability to see.

The use of sunglasses to reduce glare is not new. In the 15th century an Ophthalmologist called Turberville prescribed silk veils which were worn by his patients to reduce post operative photophobia.[23] This technique was the predecessor of sunglasses.

Sunglasses are now extremely popular as demonstrated by the 1974 statistics from the USA. In that year there were over 100 million new pairs of sunglasses purchased in the USA giving the manufacturers over $300 million in profits.[24] Sunglasses are big business and people like wearing them.

The wearing of sunglasses affects the light entering the eyes in the following manner. They affect

1. the intensity of the light
2. the spectral distribution
3. the environmental factors which operate on light by altering the background illumination.

However there is debate in the literature as to whether sunglasses alter vision.

Results of testing yellow tinted sunglasses for example showed, in one study of 98 subjects,[25] that despite cutting down the intensity of light and altering the spectral distribution, the sunglasses had no effect on vision or contrast sensitivity. One interesting point reported in this paper was that although the lenses did not improve the ability to see, over 50% of the patients reported a subjective improvement in vision and contrast while wearing the yellow tint. This was thought to be secondary to improvement in visual comfort. These findings were supported by a number of other publications using other coloured sunglasses.[26,27]

Although these studies did not statistically demonstrate that sunglasses altered visual acuity and contrast sensitivity in normal indoor laboratory lighting such lenses may alter visual performance under specific lighting conditions or in the outdoors.[28-30] These outdoor conditions include bright sunlight, glare from the ocean, glare from roads, and glare from fog and snow. (Glare is the result of light, the intensity of which is greater than that of the background to which the observer is adapted).

BRIGHT SUNNY DAY

Although the human eye only sees light from about 380 nm to 760 nm, short wavelength ultraviolet light also enters the eyes. High levels of short wavelength spectral light together with longer wavelength ultraviolet light cause the visual annoyance associated with glare. When looking at a scene on a sunny day, firstly with sunglasses consisting of an ultraviolet filter and a coloured filter, and then without, it is apparent that sunglasses minimally alter the appearance of the scene. This occurs because the sunglasses reduce the amount of short wavelength light hitting the eye. Also, visual comfort is enhanced as it is not necessary to 'screw up' the eyes to look at the scene.

Thus sunglasses can alter the appearance of the world and enhance visual comfort without necessarily altering visual acuity in bright sunlight.

SNOW and FOG

The effect of glare is very noticeable when there is more ultraviolet light such as at altitude or where light is preferentially reflected into the eyes for example from the snow or in conditions of fog.

Fog is made up of droplets of water and it is the size of these droplets which determine the manner in which the light is scattered and thus the intensity of the glare. Normally short wavelength blue and violet light is scattered more than red and green wavelengths. As a result, yellow lenses which absorb short wavelength light, will minimize the effect of the scatter of the light in fog and improve visibility. Thus sunglasses have a physiological effect on our ability to see in fog.[31]

In other low contrast conditions where there are high levels of ultraviolet light, such as in blizzards and snow, yellow and yellow green tinted sunglasses or goggles will enhance visual ability by absorbing more short wavelength light.

Some studies claim that Polaroid lenses will give most improvement in visual ability in these low contrast conditions.[30] They also work just as well on reflected glare such as the glare from water.

Polaroid sunglasses are very effective for reflected glare from flat surfaces. This is because the light is preferentially reflected off surfaces and is generally reflected in a horizontal plane. Polaroid lenses oriented vertically absorb most of these reflected light rays and work no matter what the ambient light levels.

Much of the literature concluded that sunglasses only altered vision due to an increase in apparent contrast and brightness to mid spatial frequencies especially in extreme environmental glare conditions.[30, 32] Thus, in out-door conditions sunglasses enhance visual comfort and alter the appearance of the world and can sometimes alter the ability to see at mid spatial frequencies (not at high spatial frequencies).

There are a number of ocular conditions in which sunglasses do alter the ability to see in normal viewing conditions and in mildly increased glare. Two such conditions are colour vision anomaly and rod monochromatism.

(i) ABNORMAL COLOUR VISION: Tinted lens filters alter the purity of a colour so we perceive the colour slightly differently. This effect is extremely mild in patients with normal colour vision but it can have marked effects on patients with poor colour vision. Farnsworth[33] tested patients with a variety of colour vision defects. He found, for example, that yellow lenses affected colour appreciation markedly in patients with moderate to moderately severe colour defects and red tinted lenses markedly reduce vision in patients with severe colour vision loss. He concluded that patients with colour vision loss should only be prescribed mildly tinted lenses.

Tinted lenses can also be used to enhance the appearance of objects to enable colour defective patients to pass tests like the Ishihara. Red lenses brighten the reds compared to the other colours enabling the patient to distinguish between the number on the plate and the background.

(ii) ROD MONOCHROMATISM: This is a condition in which the cones do not function normally rendering the child partially sighted and colour deficient. These children are extremely photophobic and virtually blinded by very bright light.

Recent work has demonstrated that dark red tinted lenses (especially contact lenses) which absorb long wavelength light will reduce the reflected glare from the background. This improves the contrast between the background and the image for rod monochromats.[34]

As there are some conditions in which sunglasses actually alter vision I decided to investigate if Irlen's hypothesis that reading disabled patients were more affected by glare than normals[4] was correct and, if so, did tinted lenses improve their ability to see, as they do for rod monochromats.

I investigated the effect of tinted lenses on vision and contrast sensitivity in conditions of increased glare in reading disabled children and children who read normally.

This study was conducted in the Division of Orthoptics at the Lincoln School of Health Sciences, La Trobe University.

Two groups of patients were studied

Group 1. 22 reading disabled patients aged 8-19 years who had been prescribed tinted lenses at the Irlen tinted Lens clinic in Melbourne. (Nineteen children were 16 years or under).

Group 2. 129 school children aged 12 to 16 years.

To be included in the study the patients had to have monocular visual acuity of 6/6 or better (Snellen's) and N5. Stereopsis had to be normal using the Lang Stereotest and strabismus was excluded.

All the children were tested with and without tinted lenses using the Vistech Multivision

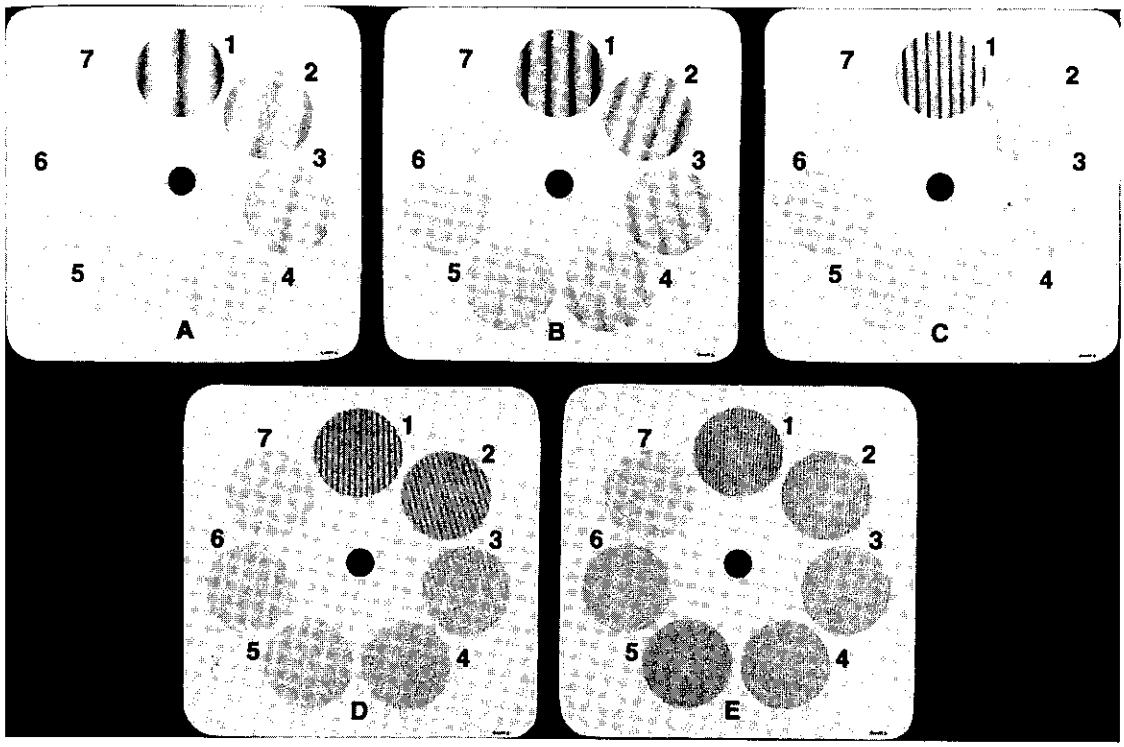

Figure 1a: MCTS Contrast Sensitivity Targets.

Contrast Test System (MCTS) test. Contrast sensitivity was assessed under normal photopic lighting conditions both with and without additional peripheral glare. Testing was done at 1/3m.

The gratings used are the same as those used in the VCTS tests, namely circular gratings of 1.5, 3, 6, 12 and 18 cycles per degree. The children were asked to look at each circular target in turn and inform the examiner of the orientation of the stripes (see figures la and lb). The minimum contrast at which the stripes could be detected was recorded for each spatial frequency (strip width).

Each of the normals (group l) had contrast sensitivity assessed binocularly at 1/3m with one randomly selected colour and strength of tinted lens. Tint colours used included green, yellow, blue, pink, amber, grey and clear with strengths of 25%, 35% and 50%.

Patients in group 2 were tested with and without the lenses prescribed by the Irlen lens clinic using the Irlen technique.

The test was firstly performed under normal photopic lighting conditions with and without tinted lenses and then with the peripheral glare lights turned on with and without tinted lenses. The results were recorded and later averaged for the whole group and transposed onto the evaluation form supplied with the test (see figure 2). The horizontal axis gave the stripe width or spatial frequency and the vertical axis gave the contrast sensitivity.

RESULTS

In this study only 22 tinted lens wearers from the Irlen Lens clinic were examined so, at best it can be described as a pilot study giving preliminary results only. (The full statistical analysis of the results is the subject of another publication).[35]

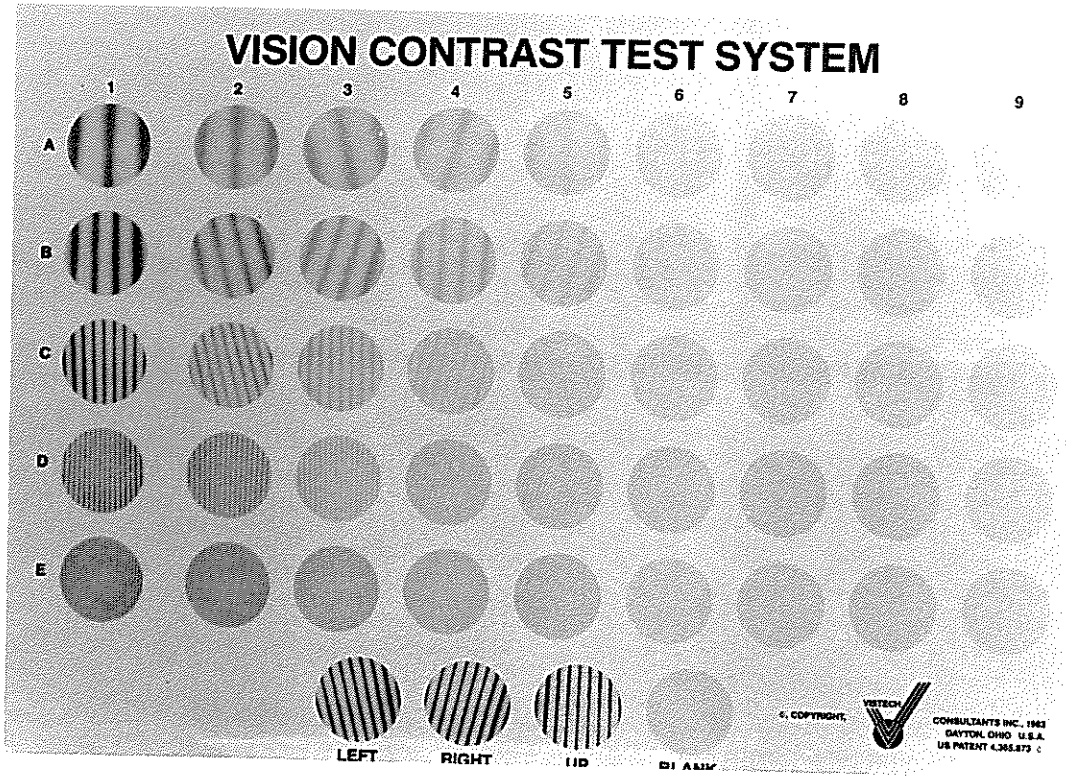

Figure 1b: VCTS Contrast Sensitivity Targets.

Effect of tinted lenses:
Analysis of variance (ANOVA), at the $p<0.05$ significance level, showed that the score was a statistically significant alteration with the use of tinted lenses in Rows A, D and E in the normals.[35] In Row A the score minimally increased with tinted lenses and in Rows D and E the score minimally decreased when tinted lenses were worn (see figure 2a). The same findings occurred using a Wilcoxon matched pairs signed-ranks test and using a Friedman two-way ANOVA.[35] These results differed from my results gained when using the Vistech VCTS 6000 and it conflicted with other reports in the literature on the effect of tinted lenses on contrast sensitivity.[5,19,20,25,27,30] (The pattern of the alteration of score with tinted lenses was confusing as there was a tendency for scores to decrease in Row A and increase in Rows D and E).

In the Irlen clinic reading disabled group, tinted lens wear did not have a statistically significant effect on contrast sensitivity (Wilcoxon test; see figure 2b). This result was in keeping with the other reading disabled patients I have assessed.

Effect of glare (without tinted lenses):
Using an ANOVA, a Wilcoxon test and a Friedman analysis in the normals peripheral glare produced no statistically significant effect on contrast sensitivity in Rows A, D and E however in Row B and C the response to the contrast sensitivity test improved (see figure 3a). This improvement was statistically significant.

In the Irlen clinic reading disabled group, contrast sensitivity improved using peripheral glare without tinted lenses in Rows A, D and E (see figure 3b). This improvement was NOT

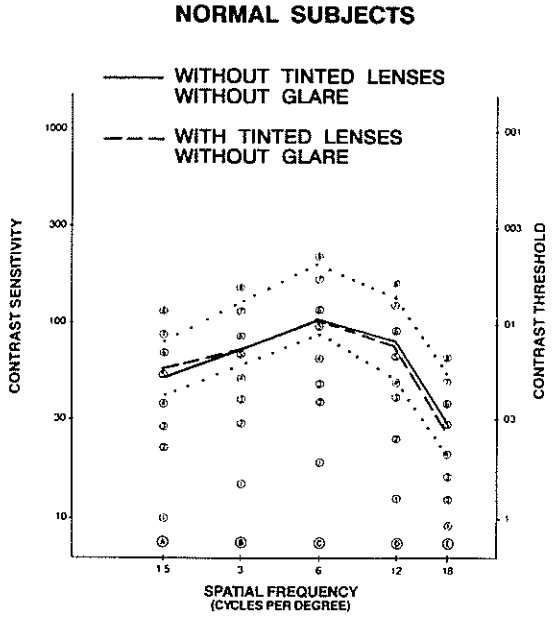

Figure 2a: The Effect of Tinted Lenses on Contrast Sensitivity (NO Glare); Normal Subjects.

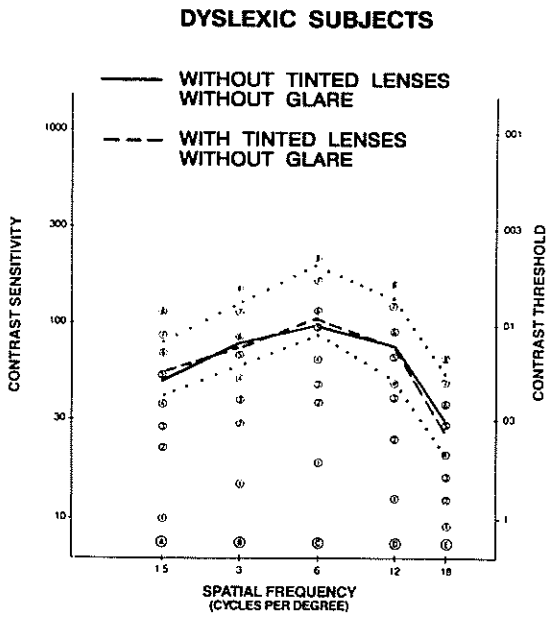

Figure 2b: The Effect of Tinted Lenses on Contrast Sensitivity (NO Glare); Irlen Lens Clinic Patients.

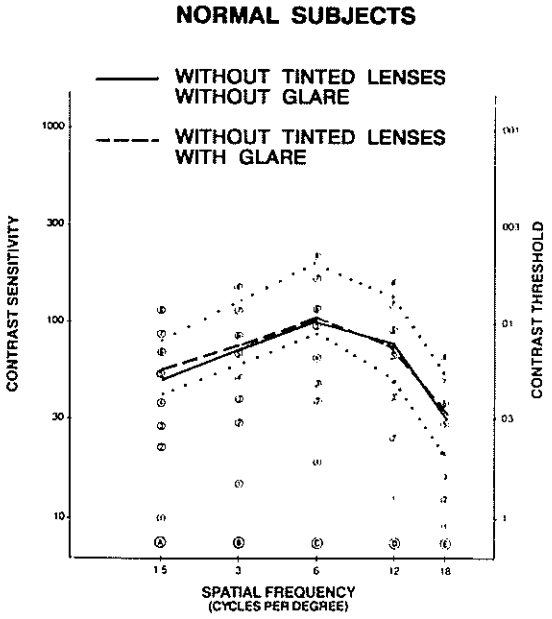

Figure 3a: The Effect of Glare on Contrast Sensitivity (NO Tinted Lenses); Normal Subjects.

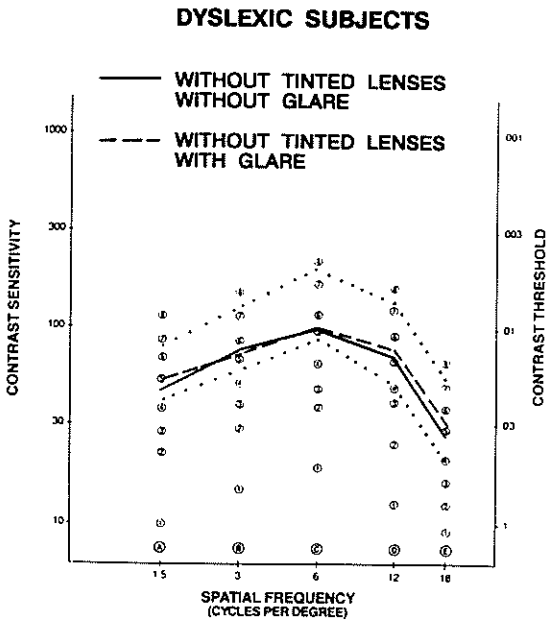

Figure 3b: The Effect of Glare on Contrast Sensitivity (NO Tinted Lenses); Irlen Lens Clinic Patients.

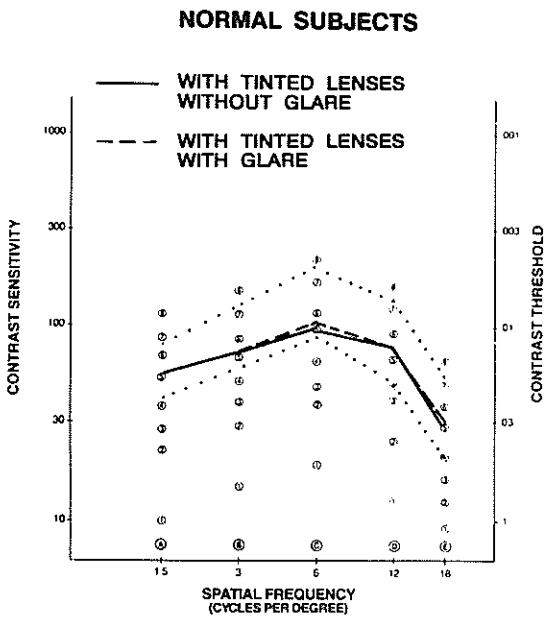

Figure 4a: The Effect of Glare on Contrast Sensitivity while Wearing Tinted Lenses; Normal Subjects.

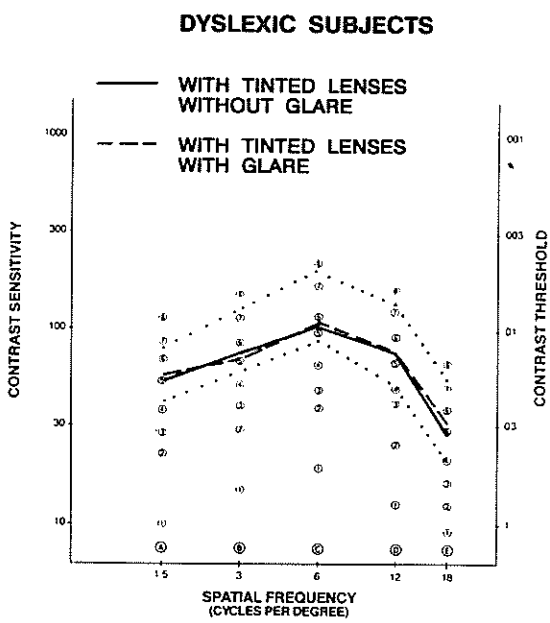

Figure 4b: The Effect of Glare on Contrast Sensitivity while Wearing Tinted Lenses; Irlen Lens Clinic Patients.

statistically significant using a Friedman ANOVA assessment.

A similar finding occurred with tinted lenses (see figures 4a and 4b). In both groups there was a tendency for the score to improve when the test was performed with glare (both with and without tinted lenses) but this was not statistically significant.

DISCUSSION

The finding of an alteration in score (both up and down) with the use of tinted lenses in the normal subjects cannot be explained. Further analysis of a larger sample would be needed to clarify this finding.

The finding of improvement in some scores with peripheral glare (which occurred with and without tinted lenses) may be accounted for in terms of pupillary response. The glare causes the pupils to constrict thus improving visual resolution.

In all cases, both reading disabled and normals, the combination of tinted lenses and glare produced no statistically significant effect. Thus if any child was minimally affected by glare, tinted lenses had no effect on the response.

However the findings of importance in the Irlen Lens Clinic patients were that peripheral glare did not cause a deterioration in contrast sensitivity and that tinted lenses did not affect contrast sensitivity.

If Irlen's suggestion that the glare from the page affected the patient's ability to see the page was correct, one would have anticipated that the contrast sensitivity recordings from the reading disabled children would have been reduced with the introduction of glare. This did not occur with or without tinted lenses.

If Irlen's other suggestion that tinted lenses enabled the patients to see more easily was correct one would have anticipated that the scores (with and/or without glare) would have altered with tinted lenses. They did not.

Once again the data I have collected has given NO explanation for the claims of the effects of tinted lenses on vision.

This finding is probably to be expected when taking into account recent findings on the

aetiology of reading disability reported in the literature.

The literature is providing more and more evidence that reading disability is more likely to be associated with linguistic processing anomalies, and that the visual system in dyslexics is perfectly normal.

In 1987 Vellutino[36] suggested that reading disability may be the result of dysfunction during storage and retrieval of linguistic information rather than a consequence of a deficit in the visual system.

He tested a group of children; half of whom were dyslexic. All the children were asked to write pseudo words then Hebrew words from visual memory. The results showed that the dyslexic children performed as well as the normals. This suggested that when complex, word like symbols lacked any linguistic association and had no meaning or sound the visual recall of these symbols was no more difficult for the dyslexics than the normals.

Eye movement disorders have also commonly been associated with reading disability.[37-42] For years the literature has debated whether such anomalies are a cause or a result of the reading deficiency, and this debate has not been resolved.

The offending eye movements are usually reported to be saccadic movements (return sweeps) and micro saccadic movements which are controlled in the frontal cortex; area 8.

Despite this, most of the eye movement treatments for reading disability include pursuit movements (watching a ball swinging on a pendulum or drawing circles on a blackboard) and fixation movements (jumping up and down while reading letters on a chart) which are controlled in the occipital cortex areas 17, 18 and 19, convergence and binocularity exercises.[43] The control centres for these two are yet to be found.

It seems odd that treatment of movements mostly mediated in the occipital cortex or other parts of the brain will help an anomaly in saccadic movements which are initiated in the frontal lobe.

Much of the literature has suggested that eye movement anomalies reported in reading disabled are present in all children not just poor readers. For example, a study conducted by Polatajko[44] in 1987 showed that there was no statistically significant difference in OKN, smooth pursuit and refixation saccades between normal and dyslexic children. He concluded that these eye movements were not fully developed in any children. Similar findings have been reported by a number of other authors.[45-48]

Anatomical evidence has cast further doubt on the theories of reading disability being caused by some anomaly of the visual system. Using a brain activity mapping (BEAM) technique, Duffy et al[49] showed qualitative differences in the functioning of the speech, language and linguistic areas of the left brain in dyslexics.

This was further supported by Galaburda.[50] He conducted autopsies on the brains of dyslexic patients and found that the arrangement of neurones was distorted in the language related areas particularly in the left cortex. He suggested that this may well explain the linguistic anomalies found with dyslexics.

The other major question that has to be addressed in reading disability is the effect of motivation. It is thought that a large percentage of reading anomalies may be secondary to lack of motivation. Once again the eyes are not to blame.

CONCLUSIONS

At the OAA conference in 1985 when I first spoke about tinted lenses my major objections to the Irlen treatment were as follows

1. Irlen was claiming that reading disability had an ocular basis but there was no evidence for this anywhere in the literature. There is still no evidence for this.
2. Irlen was claiming that tinted lenses were curing reading disability by cutting out a narrow band of spectral light.
   Analysis of Irlen lenses has revealed that they do not cut out bands of light.
3. Irlen claimed in 1985, and still claims, that tinted lenses enabled a balanced interaction between rods and cones which in turn enabled the patient to read.
   There is absolutely no evidence that rod cone interaction is necessary for normal reading,

and there is ample evidence to suggest that reading is normal in the absence of rod function.

4. Irlen had not tested her lenses on a population of normal readers to see if their reading ability was also altered.
   This still has not been reported in the literature.
5. The schedule Irlen used to determine which children would benefit from tinted lenses was a secret hence could not be subject to normal scientific scrutiny. It is still a secret.
6. Irlen had not discussed the possibility of a placebo effect of the lenses.
   This has now been suggested widely in the literature as a major factor in the success of the tinted lenses.
7. Irlen had not tested whether or not tinted lenses altered vision in any way despite claims that the lenses improved the child's vision and thus ability to see the print on the page.
   I have tested 744 normal and reading disabled children and vision and contrast sensitivity have NOT been altered by the lenses in any reading disabled children and in the majority of the normals.
8. Irlen lenses were expensive and there were substantial monetary gains to be made by the franchise holders.
   This situation remains unaltered.
9. In 1985 Irlen claimed that her treatment worked in isolation.
   In 1990 she concedes that visual examination and remedial teaching are also needed.

Almost all of us have had pleas for help from the parents of children with reading difficulties. Reading is the milestone of education and it is almost always seen as an essential prerequisite for success in life.

The studies on the effect of tinted lenses on reading performance remain inconclusive. Despite the evidence to suggest that tinted lenses do not alter vision or contrast sensitivity in reading disabled children there is no question that they do enhance 'visual comfort' and alter the appearance of the world, and that some people prefer to 'see the world' through tinted lenses. It is yet to be proven if this alteration in 'visual comfort' and appearance has any true effect on the ability to read, or whether any alteration in reading is secondary to a motivational or placebo effect.

Determining the effect of the lenses on the ability to read is not the responsibility of the orthoptic profession. It should be left in the hands of the reading experts and the psychologists. Our responsibility lies in visual and ocular assessment of reading disabled children.

The community as a whole associates the ability to read with the eyes. We must attempt to dispel the rumours that eye defects cause reading disability and to dispel the claims that tinted lenses improve vision.

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THE EFFECT OF ACUTE PHYSICAL ACTIVITY ON LEVELS OF STEREOACUITY

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Abstract

The effect of acute physical exercise on levels of stereoacuity was examined in 8 subjects. The subjects' ocular state was assessed by testing distance and near vision, contrast sensitivity, the near and distance deviation, convergence, near fusion range and stereopsis. This was followed by a bout of moderate exercise on a Monark bicycle ergometer and on completion the ocular tests were repeated. There were 8 subjects in a control group who underwent the same ocular assessment but did not participate in the exercise.

Key words: *Stereopsis, contrast sensitivity, vision, ocular muscle balance, fusion range, moderate exercise.*

INTRODUCTION

While it may be generally accepted that sportspersons must have good visual acuity for successful athletic performance, stereopsis is also considered to be necessary. In basketball, for example accurate stereopsis is considered to be an important factor in a player's ability to shoot for the basket[1], and Jolly and Jolly[2] found that successful competitive tennis players scored significantly higher on a stereoacuity test when compared to a normal population. They hypothesised that the natural selection which operates in sporting activities may be influenced by visual standards. The effect of accurate stereopsis has been questioned by Beals et al (cited by Sherman)[1], who suggested that the level of dynamic visual acuity (DVA) may be more important than depth perception.

DVA and kinetic visual acuity (KVA) are defined as "visual acuity for the moving target".[3] The distinction between DVA and KVA is the "difference in the direction of movement of the test object". DVA is tested when the test object is moved in horizontal and vertical directions. KVA is tested when the test object is moved from far to near. Suzumura[4] studied the effect of acute exercise on DVA and KVA. He found that KVA was decreased following an acute exercise bout. These results are in agreement with Watanabe.[3] Watanabe's study revealed that while KVA is decreased following an exercise bout, static visual acuity (SVA) remained unchanged immediately after exercise, but increased 7-9 minutes later and was maintained at that level until 21 minutes post exercise. This increase was statistically significant. He suggested that different physiological mechanisms may be involved in SVA and KVA in terms of visual perception. These results were not compared to those of a control group.

Visual acuity is only one component of stereoacuity. The other components which affect

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TABLE 1
Number and age of subjects

	No.	Mean Age	Range	Female	Male
Exercise group	8	33 yrs	22-48 yrs	4	4
Control group	8	32 yrs	17-48 yrs	6	2

the level of stereoacuity are ocular muscle balance and the fusion ability. As visual acuity has been found to alter with exercise, the aim of this pilot study was to investigate the effect of physical activity on stereoacuity, by examining stereopsis and its components, namely, vision, contrast sensitivity, and ocular muscle balance including convergence and fusion.

METHOD

Subjects

Sixteen subjects were randomly assigned to either the control or the exercise group. Subjects' details are summarised in Table 1. All subjects were non-orthoptist employees of the Lincoln School of Health Sciences. None had any previous ocular history or history of cardiorespiratory disease which limited their ability to participate in moderate physical exercise. Subjects were fully acquainted with experimental procedures and all signed a written consent form prior to participation in the study. The study was approved by the University's Ethics Review Committee.

Method of Visual Assessment

A number of visual tests were performed both prior to and following the experimental treatment. The tests chosen were those which gave a quantitative measure of function where possible. They were visual acuity at 6 metres and at 1/3 metre, contrast sensitivity at near, measurement of the near and distance deviation by prism cover test, ocular muscle balance, fusion range and stereoacuity.

Visual Acuity

This was tested uniocularly with a Snellen's Chart at 6 metres. The same chart was used for the pre and post tests. The maximum level possible was 6/4. No subject wore glasses for distance. Near vision was tested uniocularly at 1/3 metre using the Moorfields Bar Reading book. Reading glasses were worn by one subject in the control group.

Contrast Sensitivity

Contrast sensitivity was tested with the Vistech vision contrast sensitivity test system (VCSTS), Model 6000 designed for use at near. This test was chosen as it is considered to be a more sensitive test of visual function than a visual acuity test. The test was administered uniocularly. Reading glasses were worn by one control subject. The results were recorded by noting when a grating was visible and the orientation correct. Subjects were encouraged to attempt the grating beyond that at which they had originally stopped, as suggested in the manufacturer's instructions.

Measurement of the Deviation

Measurements were taken to quantify the amount of the deviation and any change post exercise. The type of deviation (ie whether the deviation was latent or intermittent) was not considered to be relevant. Measurements were taken by the prism cover test at 1/3 metre and at 6 metres while the subjects fixed on an accommodative target. The size was assessed as the strength of prism below that where a reversal of movement was noted.

Ocular Movements and Convergence

Ocular pursuit movements were assessed to detect any gross abnormality; no attempt was made to objectively measure pursuit or saccadic velocities. The convergence near point was measured with the RAF near point rule. The subjects were asked to maintain single vision while fixing on the vertical line with the dot as it was moved towards the nose. The convergence near point was assessed at the position where the subject indicated that the target had formed a double image. This was performed three times and an aggregate measurement was taken.

Fusion Range

The near convergence and divergence ranges were measured with the prism bar while the

subject fixed on an accommodative target. The maximum convergence range was recorded at 45△, this being the largest prism on the bar.

Stereoacuity

Stereoacuity was tested in the near position using the TNO stereotest. The level recorded was the maximum level at which both plates were appreciated correctly.

PROCEDURE

The exercise group followed the first set of visual tests with a 15 minute bout of moderate exercise. Subjects cycled on a Monark bicycle ergometer at a work load selected to elicit a heart rate representing 70 to 75% of their predicted maximal heart rate as described by Astrand et al[5]. Heart rate was continually monitored throughout the exercise period using a heart rate monitor (PE 3000 Sport Tester). The control group followed the first set of visual tests with a 15 minute rest period. The second set of visual tests followed either the exercise or the rest period for the exercise and control groups respectively. This study employed a 2 x 2 design with one between-subjects factor (group) and one within-subjects factor (pre-post test). Analysis of differences between groups was performed using an independent groups t-test on the differences between pre and post-test scores in each group for each of the dependent variables except visual acuity. The significance level for each test was 0.05.

RESULTS

Visual Acuity

The majority of subjects in both groups had 6/6 vision or better in each eye. One subject in the exercise group had 6/9 vision in the left eye only. In each group, 2 subjects recorded a reduction of one line in one eye between the pre and post tests. Clearly there was no difference between the groups on pre and post testing.

Near Vision

All subjects in both the exercise and control groups had N5 vision at near. There were no differences at the post test.

TABLE 2
Measurement of the near deviation

	Exercise Group		Control Group	
Subjects	Pre	Post	Pre	Post
1	0	+1△	−10△	−10△
2	−10△	−4△	−8△	−14△
3	−4△	−4△	−1△	−1△
4	+14△	+18△	−16△	−12△
5	−6△	−4△	−4△	−6△
6	−6△	−4△	−14△	−10△
7	−2△	−1△	−1△	−1△
8	−2△	−2△	0	−1△

Contrast Sensitivity

Both groups were similar at the pre-test, and were similar for both left and right eyes. At the post-test, the control group had higher mean values at each of the five contrast levels while the exercise group showed a minimal increase in the lower and mid contrast levels. The contrast sensitivity results are shown in Figures 1 and 2. They are represented cumulatively, that is the values of each eye were totalled and the mean values are represented on the tables.

Measurement of the near deviation

The near prism cover test results are shown in Table 2. In the exercise group, 6 subjects showed a change in the deviation. Subjects 2, 5, 6 and 7 recorded slightly less divergent deviations, while subjects 1 and 4 recorded a slight increase in the convergent deviations. Only 2 subjects remained unchanged. In the control group, 3 subjects measured differently post test. Subjects 2 and 5 showed an increase in the divergent deviation. Subject 4 demonstrated an increase in the convergent deviation. The remaining 4 subjects were unchanged. While this change in the deviation of the experimental group is interesting it was not statistically significant.

Measurement of the Distance Deviation

The results of the distance measurement show no change between the pre and post tests in either group, except from one subject in the exercise group whose convergent deviation increased from 1△ to 2△. This was not statistically significant.

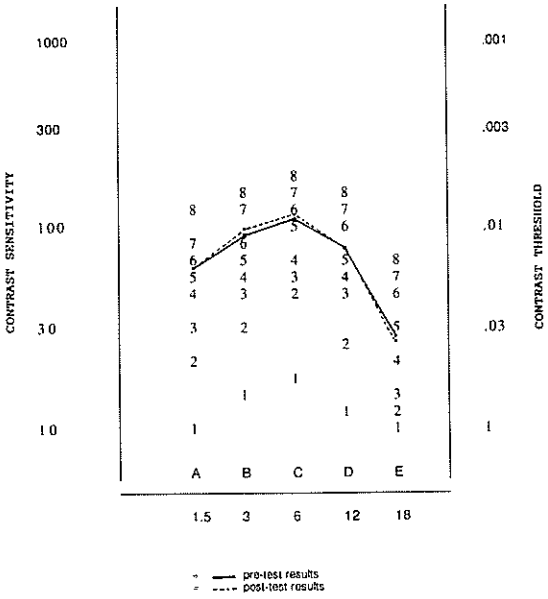

Figure 1: Cumulative results of both eyes of Experimental Group.

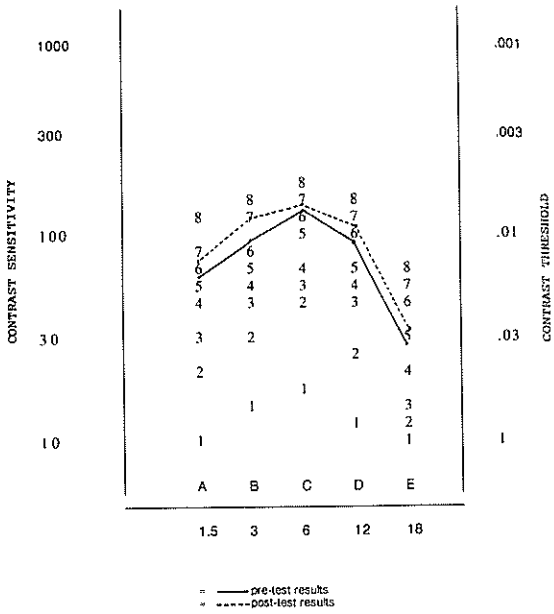

Figure 2: Cumulative results of both eyes of Control Group.

Ocular Muscle Balance

No subjects in either the exercise or control groups had any gross defect of smooth pursuit movements at the pre test. The equipment was not available to measure pursuit or saccadic velocities, but on gross assessment there were no changes post test. At pre test, the mean convergence near point of the exercise group was 6 cms, which was the same at the post test. The mean of the control group at pre test was 7cms and it too remained unchanged at post testing.

Fusion Range

Four subjects in the exercise group showed mild (average of 4△) increase in the fusion range, while one subject showed no change and one, a decrease of 1△ . The same changes were noted in the control group. These differences were not statistically significant. The fusion ranges are shown in Table 3.

Stereoacuity

The results of testing stereoacuity with the TNO pre and post testing is shown in seconds of arc in Table 4. The average score of the two groups was quite different at the pre test which was largely due to control subject 5. Without this subject, the groups showed less discrepancy at pre test and show similar decreases at post test. These differences were not statistically significant.

DISCUSSION

The effect of exercise on visual acuity.

There was no statistically significant difference between the results at pre and post testing. The mild reduction of vision of the 4 subjects cannot be explained except to suggest that it was most likely to be the effects of test-retest reliability.

The effect of exercise on contrast sensitivity.

The importance of the raised values is not clear. It may be that the raised values of the control group indicated merely random error as the group sizes were small. It may also be due to a problem with test-retest reliability, that is, on post-test the control group was more familiar with the test and so scored higher. It is not possible to ascribe a cause for these observed differences given the limitations of the study.

TABLE 3
Fusion Range

	Exercise Group				Control Group			
	Pre-test		Post-test		Pre-test		Post-test	
Subject	Conv	Div	Conv	Div	Conv	Div	Conv	Div
1	+45△	− 6△(51△)	+45△	− 6△(51△)	+30△	− 8△(38△)	+35△	− 6△(41△)
2	+45△	−10△(55△)	+45△	−12△(57△)	+45△	−10△(55△)	+45△	−12△(57△)
3	+35△	−13△(48△)	+45△	−12△(57△)	+35△	−16△(51△)	+35△	−16△(51△)
4	+45△	−11△(56△)	+45△	−14△(59△)	+20△	−14△(34△)	+20△	−14△(34△)
5	+25△	−16△(41△)	+25△	−15△(40△)	+45△	−12△(57△)	+40△	−18△(58△)
6	+18△	− 6△(24△)	+20△	−10△(30△)	+45△	−18△(63△)	+45△	−18△(63△)
7	+45△	−10△(55△)	+45△	−12△(57△)	+16△	−12△(28△)	+13△	−15△(28△)
8	+45△	−14△(59△)	+40△	−10△(50△)	+18△	−12△(30△)	+22△	−12△(34△)

(The figures in brackets are the total fusion range)

None of the differences were statistically significant.

The effect of exercise on the deviation.
While the changes in the deviation measured at near are not statistically significant, it is of clinical interest that there was an apparent increase in the esophoria and a decrease in exophoria amongst the exercise group, which was not evident in the control group. A possible explanation for these observations is that an increase in body temperature causes an increase in conduction velocity in muscles. This may be sufficient to cause an increase in convergence particularly when the eyes are already in that position, that is, the medial recti are in a state of contraction. This would explain why the increase was evident at near only.

The effect of exercise on ocular pursuit and vergence movements.
While there were some changes in convergence near point results, none of the differences were statistically significant. They may be due to the subjectivity of the test. Subjects were encouraged to maintain single vision for as long as possible, and it was impossible to ascertain if the effort to converge was equally applied at the pre and post tests. This possibly could be overcome by examining a larger group.

TABLE 4
Stereoacuity

	Exercise Group		Control Group	
Subjects	Pre	Post	Pre	Post
1	60″	60″	60″	60″
2	30″	15″	120″	60″
3	30″	30″	30″	30″
4	60″	60″	60″	30″
5	30″	30″	480″	240″
6	60″	30″	60″	60″
7	30″	30″	15″	30″
8	60″	60″	120″	60″

The effect of exercise on the fusion range.
As there was an apparent increase in the convergent deviation of the subjects in the exercise group at near, it might be expected that there would have been a corresponding increase in the fusion range at near. This was not found to be so. When the results of these subjects' fusion range was compared to the near deviation, in cases 2, 6 and 7, there was an increase in the fusion range and in case 5 there was a decrease. While this is interesting, the major increase was in case 3 who did not show any alteration in the near deviation. Unfortunately, convergence was only assessed to 45△ on the prism bar. It is conceivable that if convergence had been measured to its maximum, any increase would have been evident. However, there was no increase on convergence as measured on the RAF gauge except in 2 cases and that was only by 1cm in each case.

The effect of exercise on stereoacuity.
As there was no statistically significant difference found in any of the tests of the various aspects of visual function which combine to result in a person's ability to perceive depth, it

was predictable that stereoacuity was not altered by exercise. However, it may be interesting in future experiments to examine differences in eso and exo disparity by appropriate changes in presentation of the tests, as it may change with alteration of the near deviation.

CONCLUSION

In conclusion, there were no statistically significant differences between either the exercise or control groups on pre and post testing for visual acuity, contrast sensitivity, ocular muscle balance, fusion range or stereoacuity. It is interesting, however, to speculate on the small differences which were found. Most may be due to subject error, to test-retest error, or to a learned response in cases of minor differences recorded in vision testing, convergence, fusion range and stereoacuity testing. If there had been a larger number of subjects some of these errors may have been minimised.

The results of the contrast sensitivity did not demonstrate as marked a learning curve tendency as in the control group. A larger group would help to explain whether this is test-retest error or whether contrast sensitivity is affected by acute exercise.

While the differences in the results of the measurements at the pre and post tests were not statistically significant, the fact that 6 of the 8 subjects in the exercise group showed an increase in the deviation in a convergent direction is interesting and may be an indication of increased conduction velocity in the medial recti muscles. More studies are necessary to prove this hypothesis.

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THE OCULAR MOTOR DEVELOPMENT OF INFANTS

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Abstract

The aim of this longitudinal study was to record the development of ocular motor responses of infants observed in the routine clinical environment during the first year of life. The fixation, smooth pursuit and saccadic responses to different stimuli at various age groups are outlined. The responses to the fusion reflex test using a 10 and 20 dioptre prism are also described.

Key words: *Infant, ocular motor development, fixation, smooth pursuit, saccades, convergence, binocular response.*

INTRODUCTION

There have been many laboratory studies of infants' ocular motor behaviour such as those by Kremenitzer et al,[1] Hainline,[2] and Barten et al[3] studying the characteristics of fixation, smooth pursuit and saccades. One study using clinically based assessment techniques by LaRoche and Anderson[4] of 40 normal neonates demonstrated the preferential response of infants for the human face, reporting that 83% of the infants looked at and followed an examiner's face, with none responding to a penlight. They also reported that 73% of the neonates showed some response to OKN strips. This study by LaRoche and Anderson[4] assessed the infants only in the first days of life.

Two previous studies on the development of binocular function have shown conflicting results. Coakes et al,[5] using an 8 dioptre and a 15 dioptre prism, reported that a response was observed to a smaller prism at a younger age than to a larger prism. This pattern of development is contradictory to that reported by Aslin[6,7] who suggests the hypothesis that infants require a larger retinal disparity as a stimulus to motor fusion than do adults.

The aim of this longitudinal study was to record the development of ocular motor responses of infants observed in the routine clinical environment during the first year of life.

METHOD

Systematic sampling of infants born at Monash Medical Centre, Clayton Campus was carried out. Parental consent was gained for the one hundred and one infants tested. To be included in the sample infants were to be full-term with uncomplicated delivery and normal neonatal assessment. Table 1 gives a description of the sample showing the range of normal infants.

The infants were assessed during the first week, then at 1, 3, 6 and 12 months. Fixation was observed using the examiner's face, a penlight, a visual object and a sound-making visual toy to gain the infant's attention. The examiner's face was used as a fixation object only until a reliable response was obtained from other stimuli, so was not recorded beyond 1 month of age.

The following aspects of ocular movement were assessed both horizontally and vertically;

- smooth pursuit was assessed using the same

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TABLE 1
Description of sample (N = 101)

Variable	Mean	Standard Deviation	Range
Gestation (weeks)	39.7	1.05	37-42
Birth weight (grams)	3327	429	2212-4670
Apgar score: 1 min	8.4	1.02	4-10
Apgar score: 5 mins	9.6	0.53	8-10
Maternal Age (years)	27.9	5.46	17-40

stimuli as those used to gain ocular fixation.

- saccades were assessed using the examiner's face, a penlight, a visual toy and an auditory stimulus to gain a refixation response.
- optokinetic nystagmus using an OKN drum.
- doll's head vestibular responses were assessed only at the neonatal and 1 month examination.
- convergence using a visual object.

The binocular response was assessed using a 10 and 20 dioptre base-out prism to observe the fusion reflex. A visual object with a penlight was used as the fixation target.

Visual acuity was assessed using Teller Acuity Cards. Each of the infants had a retinoscopy and fundus and media examination performed under cycloplegia with Cyclopentolate 1%.

RESULTS

The number of infants tested in each age group is recorded in Table 2. Sixty nine infants were able to be fully assessed at the first visit, four infants fell asleep during assessment and the remaining twenty eight were asleep at the time of testing and unable to be wakened. Of the ninety two infants returning for the 1 month assessment, four were unable to be wakened. At all other testings, all infants were awake.

The Teller Acuity results fell within the normal range as recommended by other authors.[8,9] Retinoscopy results showed a refraction of mean

TABLE 2
Number of Infants Tested

Mean Age	Standard Deviation	Range	Number Tested
4 days	1.36	2-7	73
30.9 days	3.02	26-46	92
13.7 wks	1.28	11-18	85
26.3 wks	1.95	22-34	83
52.4 wks	2.04	47-60	74

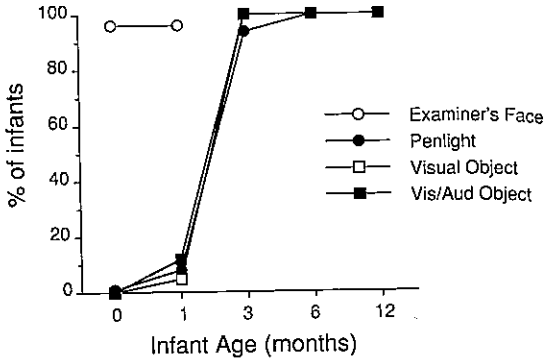

Figure 1: Fixation response.

spherical equivalent of RE + 3.00 dioptres (SD ± 1.01) and LE + 3.00 dioptres (SD ± 1.02), with a mean cylindrical error of RE 0.27 dioptres (SD ± 0.48) and LE 0.24 (SD ± 0.43), with no significant difference between right and left eyes using a paired t-test. The range of refractive error extended from 0 to + 6 dioptres of spherical equivalent and 0 to 2 dioptres of cylindrical error.

Figure 1 demonstrates the fixation responses to the different stimuli. It can be seen that the neonates responded only to the examiner's face, 96% giving a fixation response. Only one infant was observed to fixate the penlight. At 1 month similar results were obtained, but with more infants fixating the visual/auditory object (12%) and the penlight (8%). At 3 months there was a 100% response to all stimuli except the penlight which gained a fixation response in 94% of the infants.

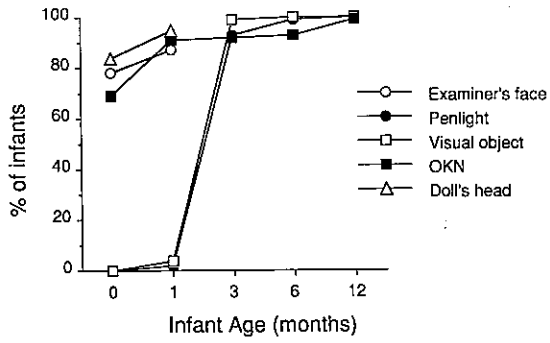

Figure 2: Horizontal pursuit/following response.

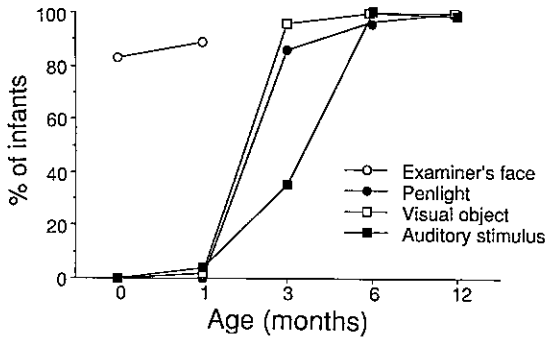

Figure 3: Horizontal refixation/saccadic response.

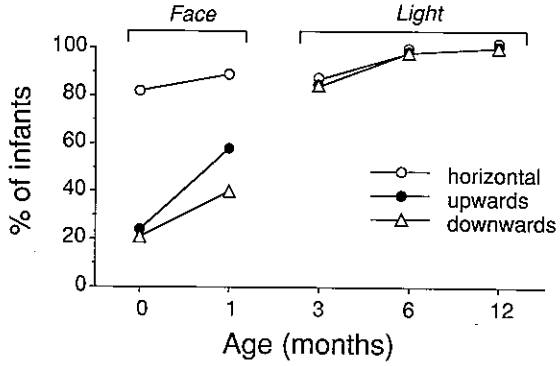

Figure 5: Refixation/saccadic response.

The responses to horizontal pursuit movements (Figure 2) and horizontal saccades (Figure 3) show a similar pattern. Of the neonates, 78% were observed to make a horizontal refixation of the examiner's face with no response to any of the other fixation objects, except the OKN drum. At 1 month of age, 87% demonstrated a horizontal following movement of the examiner's face and 89% a refixation movement. Only 2% showed a following movement to the penlight, none demonstrating a saccadic refixation response, with a slightly higher incidence of response to the visual object. Optokinetic nystagmus was demonstrated in 69% of neonates and 91% of 1 month infants. At 3 months of age 99% of infants demonstrated pursuit movements and 96% demonstrated saccadic refixation to a visual object. These figures were a little less when a penlight was used as the fixation object.

A comparison between the horizontal and vertical responses of pursuit and saccadic movements is shown in Figures 4 and 5, respectively. It can be seen that as neonates and at 1 month, horizontal movements are observed more frequently than vertical, with upwards movements more frequent than downwards movements. At 1 month, 87% of infants can be observed to make a horizontal following movement of the examiner's face, 59% an upwards and 42% a downwards movement. 89% of 1 month infants demonstrated a horizontal refixation saccade, 58% an upwards and 40% a downwards refixation.

Convergence was observed in all of the infants at 3 months. The fusion reflex is illustrated in Figure 6 and the results are compared to those found by Aslin.[6,7] In this study, using a 10 dioptre and a 20 dioptre prism, a positive

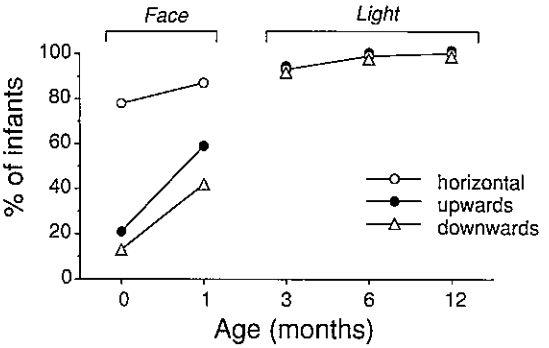

Figure 4: Pursuit/following response.

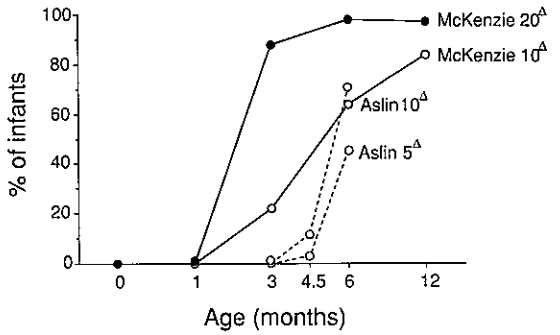

Figure 6: Percentage of infants giving positive prism fusion response.

response was observed in 22% and 88% of 3 month infants respectively, and in 64% and 98% of 6 month infants respectively.

DISCUSSION

The visual acuity, retinoscopy and ophthalmoscopy examination show that the visual development of these infants is within normal range.[8,9]

It can be seen that the fixation responses of the neonates were similar to the results found by LaRoche and Anderson,[4] showing that the majority of infants responded to the examiner's face by fixation, following and refixation, with only one infant showing any response to the penlight. At 1 month of age the results are similar, with only a small number of infants responding to stimuli other than the examiner's face.

Optokinetic nystagmus was elicited in a large proportion of the neonates, again showing a similar response to the study by LaRoche and Anderson.[4] Kremenitzer et al[1] also state that OKN is a more potent stimulus than a single target in the responses of neonates. Of note is the stability of the frequency of a response to optokinetic nystagmus from 1 month of age.

The infants at 3 months show a dramatic change in response, with almost all the infants demonstrating pursuit and saccadic responses to each of the different stimuli. It is interesting to note that a "noisy toy" did not show an increased frequency of response to a "quiet toy". At this age an auditory stimulus did not result in a refixation saccade in most infants, only 36% looked towards the sound in a horizontal direction and 11% vertically. These figures are in contrast to the developmental sequence suggested by Erhardt[10] who states that 3 month infants will turn their head and eyes towards the side of a sound.

The difference in responses of horizontal and vertical ocular movements is of interest. As detailed previously the horizontal responses, both pursuit and saccadic, appear to develop before the vertical responses, with horizontal responses much more frequently demonstrated than vertical movements. This is reported by Hainline,[2] but the frequencies are not cited. Of interest also is the apparent development of upwards responses prior to downward responses. By 3 months of age these differences are no longer observed.

Pursuit movements were observed and were qualitatively assessed as smooth or jerky. All of the neonates and 1 month infants were recorded as having jerky pursuit movements. At 3 months, 39% of the infants still showed consistently jerky pursuit, whereas 60% showed an asymmetrical response where the movement was smooth on following a target from the periphery to the midline, but jerky on following from midline to periphery. This phenomenon is reported by Erhardt[11] Faragher and McLean[12] as a developmental pattern, but no physiological reasons are given to explain the observation. By 6 months of age, 98% of the infants demonstrated smooth following movements and this increased to 100% by 12 months. Various studies, both qualitative such as Barten et al,[3] and quantitative such as Kremenitzer et al[1] and Hainline,[2] report that the latency for a pursuit movement in infants is increased and that smooth pursuit movement is interspersed with saccadic movements.

Another phenomenon reported as a developmental stage by Erhardt[10,11] is the midline jerk. On observation for this phenomenon at each age group, a midline jerk was not observed when the infants were smoothly following and maintaining their fixation of the target. At the neonate, 1 and 3 month age groups when jerky pursuit movements were still observed, there appeared no difference at the midline.

Saccadic movements in young infants are reported as being hypometric and consisting of saccadic steps.[2,7] This study would support the opinion that up until 6 months of age saccades appear hypometric, often observing 2 or 3 saccades to obtain fixation, and accompanied by head movements. At 6 and 12 months of age the majority of saccades qualitatively appear accurate.

As a binocular motor fusion response was clearly demonstrated in the majority of infants at 3 months of age using a 20 dioptre prism, the results of this study appear to support Aslin's hypothesis[7] which suggests that a stimulus greater than 10 prism dioptres is required to elicit a

motor fusion response in infants prior to 4 months of age. It is of clinical interest that in 3 and 6 month infants a small fusion response is difficult to observe because of the large amounts of head, eye and body movements that occur when attempting a fusion reflex test, whereas a 20 dioptre prism gives a more easily observed and conclusive result.

CONCLUSIONS

In summary, it would appear that the best stimulus to use for assessment of a neonate or 1 month infant in the routine clinical environment is the examiner's face, where a reliable fixation or following horizontal movement could be observed in at least 80% of the infants. At this stage, no other stimuli, excepting optokinetic nystagmus and dolls head testing, will gain a reliable ocular motor response. Of clinical importance also is the delay in the development of vertical compared to horizontal eye movements.

In contrast, by 3 months of age infants are very attentive visually and will observe any fixation object. At this stage fixation, pursuit and saccadic responses, both horizontal and vertical, are expected from all infants, though a penlight may not be of interest to some infants and may not gain a response. At 3 months of age pursuit movements may still appear jerky and saccades inaccurate, but a response is obtained. All these infants demonstrated full convergence, and a fusion response can be elicited in the majority using a 20 dioptre prism.

At 6 and 12 months all the responses are demonstrable, pursuit movements appearing smooth, saccades accurate, convergence full and almost all demonstrating a positive 20 dioptre fusion response.

ACKNOWLEDGEMENTS

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A REVIEW OF THE FARNSWORTH MUNSELL TYPE COLOUR VISION TESTS

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Abstract

This paper outlines the use of the Farnsworth Munsell (FM) type tests. It discusses the FM 100 hue, the FM D-15, the City University, the Lanthony Desaturated D-15, the Roth 28 hue and the Farnsworth F2 Tritanopic Plate tests. With the exception of the 100 Hue test these tests cannot be used in isolation to screen for congenital or acquired colour vision loss.
If a patient is found to have congenital colour blindness (ie congenital anomalous trichromatic colour vision) the Farnsworth tests can be used to give a qualitative assessment of the defect. The FM 100 Hue test is the only one to give a quantitative assessment of the defect. (The 100 Hue can also be used as an isolated screening test although it is an extremely time consuming procedure). For suspected acquired defect the Roth 28 test can be used as a starting point but it should always be followed by a 100 Hue test as it is very important to quantify the defect to monitor any progress.
The paper suggests appropriate clinical investigation protocols for screening and evaluating colour vision deficiencies.

Key words: *Farnsworth Munsell, FM 100 hue test, FM D-15 test, City University test, Lanthony Desaturated D-15 test, Roth 28 hue test, Farnsworth F2 Tritanopic Plates, colour vision screening.*

INTRODUCTION

The Farnsworth Munsell (FM) type tests examine the subject's ability to discriminate between hues which differ by a small amount when viewed under constant illumination. In the Munsell system colour is described in terms of hue (the wavelength of the light), chroma (the saturation or strength of colour) and value (the proportion of black and white light). There are a number of tests in the FM type test series including the FM 100 hue, the FM panel D-15 test, the City University colour vision test, the Lanthony desaturated D-15 test, the Roth 28 hue test and the Farnsworth F2 Tritanopic Plate test. All the above-mentioned tests (except the F2 Tritanopic Plate) use coloured targets of different hues which are selected from the range of 100 coloured papers originally manufactured by the Munsell Colour Company. (The Lanthony Desaturated Test uses desaturated coloured papers).

Any colour deficiency can be classified using the FM type colour vision tests because colour defective patients normally have greatest difficulty with those parts of the spectrum which are complementary to their deficiency. For example a subject who has a red green deficiency will have greatest difficulty with subtle shades of blue or yellow as both these colours are 'made up' from a mixture of small proportions of red and green. These small proportions of red and green are poorly perceived by subjects with red green deficiency.

This paper discusses the FM tests with a view to suggesting appropriate clinical investigation protocols using the Farnsworth Munsell and

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other colour vision tests for patients with suspected colour vision anomalies.[1]

FARNSWORTH MUNSELL 100 HUE TEST (FM 100 hue)

The FM 100 hue has been designed to detect all types of colour vision abnormality from the mildest red green defect to total achromatopsia. According to Farnsworth[2] its primary uses are, firstly, to separate persons with normal colour vision into classes of superior, average and low colour discrimination and secondly to measure the axes or zones of colour confusion in patients with defective colour vision.

The FM 100 hue test is a test of hue discrimination consisting of 85 caps of perceptually equal differences in hue. When placed in the correct order in a circle the 85 caps form a perfect hue circle of the visual spectrum. The hue circle is divided into 4 parts (one part per box) for the testing. Each has an additional fixed or pilot cap at either end of the box and 22 or 21 loose caps. The 4 boxes render it impossible to make errors across the hue circle so patients cannot confuse reds with greens or blues with yellows.

Subjects with normal colour vision arrange the caps in order of hues between the pilot caps in each box with a few errors but patients with anomalous colour vision make errors in the boxes that are complementary to their deficiency. Contrary to popular belief there is NO time limit on performing the test.[2] In every FM test the caps have a hue spot in the centre which is 1.2cm in diameter and subtends 1.5° at the nodal point when viewed at 50 cms.

Once completed each box is shut and inverted then reopened. A number is seen underneath each cap. The order in which the patient has arranged the caps is recorded. The score for each individual cap is calculated by summing the difference between adjacent caps. For example if the cap order was '2', '4', '8', '5', '3', '6', '7' etc the score for cap '4' would be as follows

The absolute difference between the preceding cap '2' and cap '4' is 2.

The absolute difference between cap '4' and the following cap '8' is 4.

The sum of the absolute differences is $2+4=6$ hence the score for cap '4' is 6.

With this method of scoring the minimum score for any cap is 2. For example if the cap order was '8', '9', '10', '11', '12' etc the difference between adjacent caps is 1. The sum of the absolute differences is $1+1=2$. This corresponds to the lowest score marked on the vertical axis of the score sheet.

The total error score is also calculated for the FM 100 hue test by adding the scores for each individual cap. As there are 85 caps with a minimum score of 2 per cap a perfect score is 170 (or $85\times2=170$). Some clinicians refer to a perfect score of '0'. To do this they subtract 2 from the individual score for each cap thus in the above example the score for cap '4' would be 4 (or $6-2=4$). Alternately they subtract 170 from the total error score. For example, a total

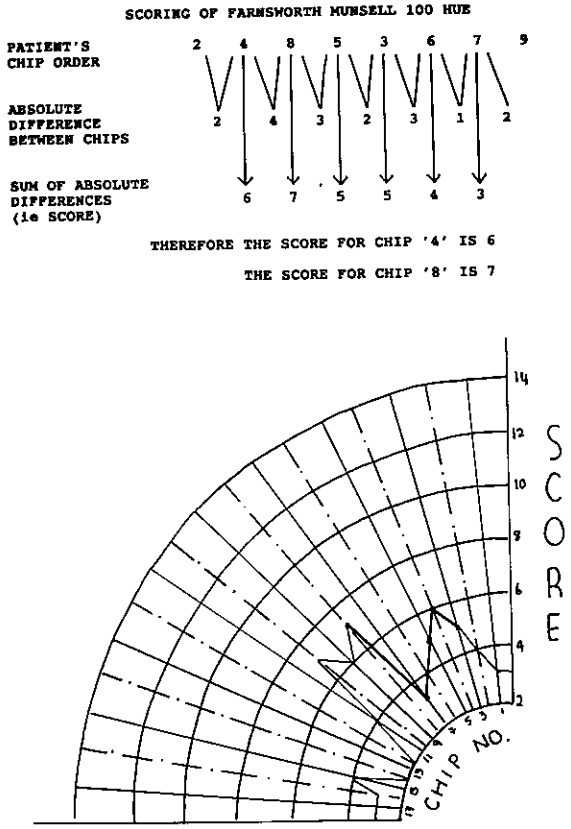

Figure 1: Scoring of the FM 100 hue test.

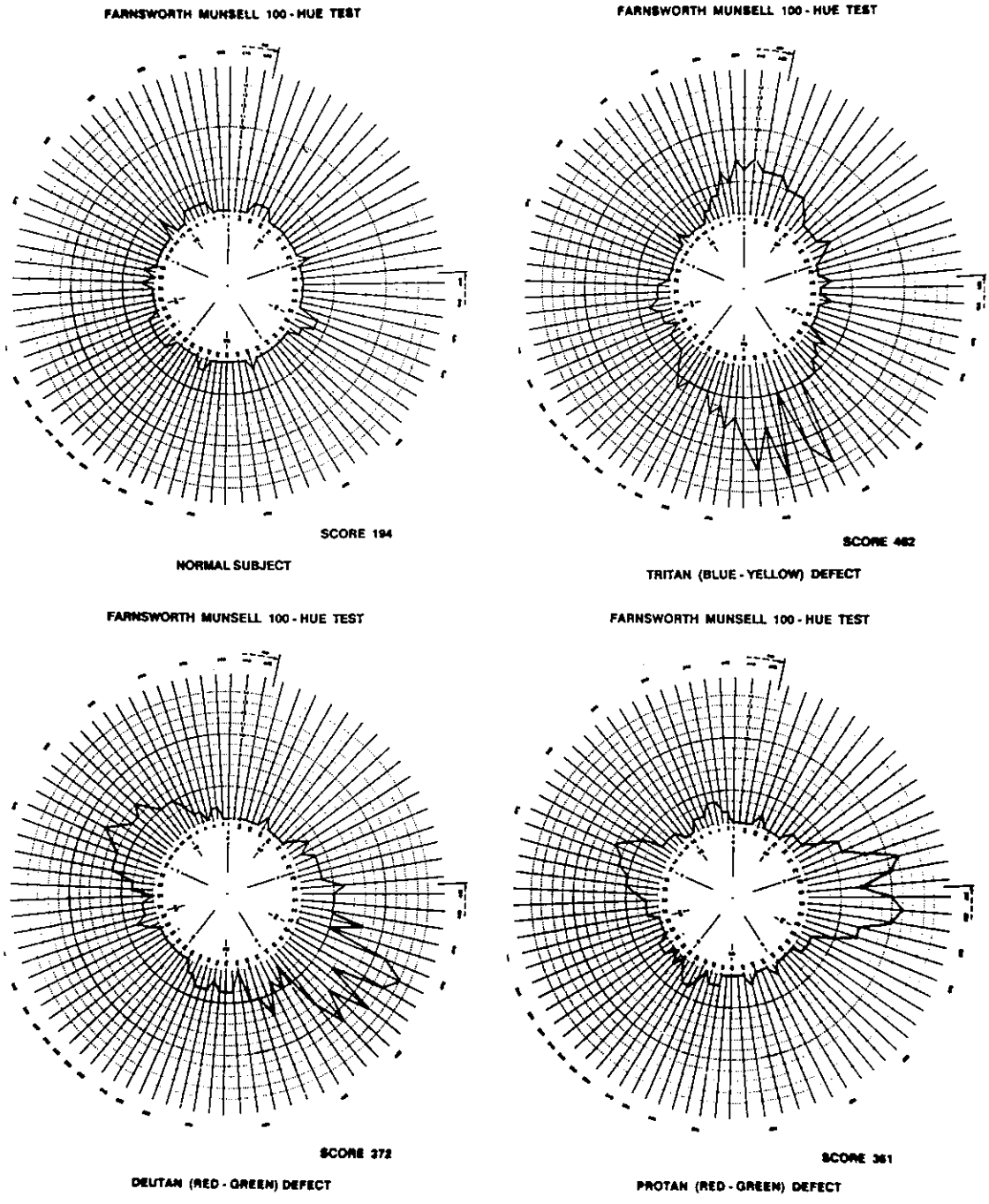

Figure 2: Typical FM 100 hue test error patterns.

error score of 275 is the same as a total error score of 105 (or 275 – 170 = 105).

According to the manual a perfect score is 170, superior discrimination occurs with a score of 171-186, normal discrimination occurs with a score of 190-270 and low discrimination occurs with scores above 270 and no particular zones of loss. The absolute error score increases with age showing the deterioration in colour appreciation which occurs with increasing age. Optimal colour discrimination occurs between the ages of 16 and 35.[3] After 55 years of age, fine colour discrimination is impaired especially blue yellow or violet, blue green discrimination.

Although some clinicians only refer to total error score when discussing FM 100 hue test results, the score alone tells the examiner nothing about the type of colour vision anomaly. To be able to differentiate the various anomalies the clinician must plot the individual scores on the FM 100 hue test graph. The inner circle of the graph gives the cap number and the vertical axis at 12 o'clock gives the score (see figure 1). When the patient has normal colour vision the plot is around the inner circle. The areas of high score in patients with anomalous colour vision are usually clustered together along certain axes known as poles of confusion (see figure 2). When there is a clustering of errors along poles in two regions which are almost opposite then the type of colour vision defect can be diagnosed.

The position of the mid points of the pole regions is also of diagnostic value. Protans or so called red 'blind' patients (protanomalous or protanopic) have a midpoint between caps 62 and 70 thus this is their greatest area of trouble. These are the bluish-purple to purple caps in boxes 3 and 4. Protan patients also have problems distinguishing the yellow and yellow-green hues from caps 16 to 22 in box 1 but they score well in the red and green areas (see figure 2).

Deutans or so called green 'blind' (deuteranomalous or deuteranopic) have mid points between 56 and 61. Thus they have most confusion trying to distinguish between blue and bluish-purple caps in box 3. Deutan patients make a few errors with orange-yellow caps between 14 and 18 in box 1 and mostly normal responses in the green and red areas of the spectrum (see figure 2). As a result the graph of a typical protan patient has more 'horizontal' poles than that of a deutan patient.

Tritans or so called blue 'blind' (tritanomalous or tritanopic) have a mid point between 46 and 52 thus they have most problems distinguishing between the greenish-blue caps in box 3. They also make mistakes distinguishing the different red hues between the red caps 84 and 4 (boxes 4 and 1) hence the more 'vertical' appearance of the graph. Tritans typically have no trouble distinguishing pure blue hues or yellow hues thus score well in those areas of the graph (see figure 2).

Patients who have a generalised loss of colour vision (due to an advanced retinal disease for example) will show errors right around the spectrum (see figure 3).

The FM 100 hue test is the most comprehensive of the Farnsworth Munsell type tests giving both differential diagnosis and score (thus progression) of the disease. The test can be used to screen for any type of colour vision loss. The major disadvantages of the test are the time it takes especially when a patient has an acquired

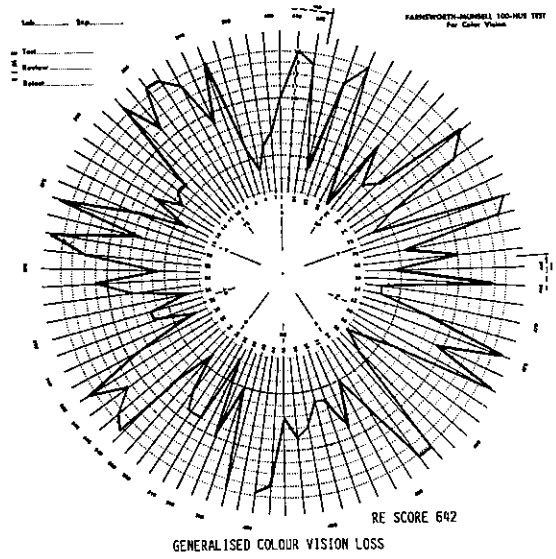

Figure 3: Generalised colour vision loss on the FM 100 hue test.

THE UNIVERSITY OF SYDNEY, DEPARTMENT OF CLINICAL OPHTHALMOLOGY
FARNSWORTH 15 HUE TEST

NAME SAMPLE RESULT AGE _____ SEX _____

DATE _____ HOSP. NO. _____

ANALYSIS

RED-GREEN DEFECT — DEUTERANOPE
YELLOW-BLUE DEFECT — TRITANOPE
PROTANOPE — NORMAL

RIGHT EYE	PATIENT ORDER	1	2	15	14	3	4	13	12	5	6	11	10	7	8	9
	CHIP NO.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
LEFT EYE	PATIENT ORDER	1	15	14	13	2	3	12	11	4	5	10	9	6	7	8

DEUTERANOMALOUS

PROTANOMALOUS

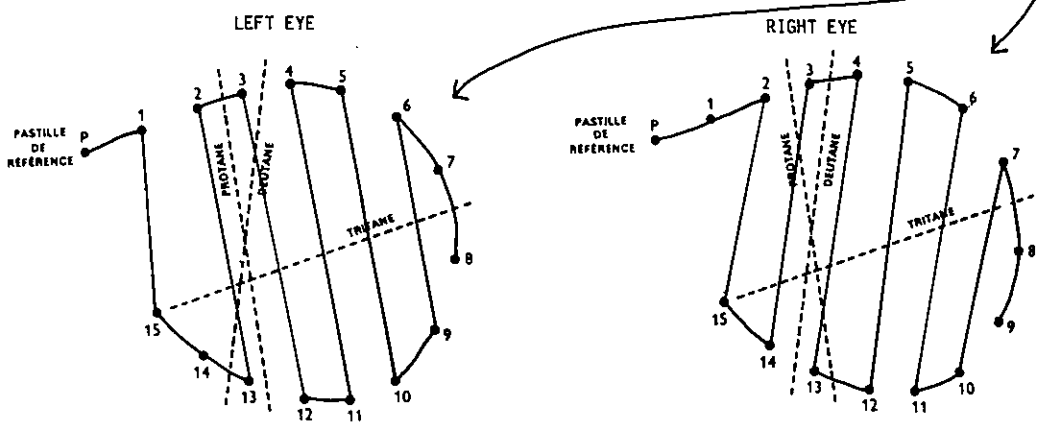

Figure 4: Axes of Confusion seen on the FM Panel D-15 test.

loss and thus must be tested monocularly. Also the differences between adjacent hues are so slight that some patients are frustrated by the fact that they cannot distinguish between them at all. Patients must be able to arrange the caps and thus must not have problems with the concept of ordering the colours into a natural colour sequence.

FARNSWORTH MUNSELL PANEL D-15 (FM D-15)

This test's full name is the Dichotomous Test[5] although it is commonly known as the FM Panel D-15. It was so named because to a colour defective patient the colour circle is virtually cut into two halves along an axis of confusion, (dichotomous means cut into two halves). For deutan patients this axis of confusion is the green to bluish red and for protan subjects it is along the bluish green to red diameter. For tritan patients the axis runs from yellow to blue. These axes are printed on the score sheet (see figure 4).

The FM D-15 test was not designed for screening. This was emphasised by a number of authors[4-6] including Linksz[5] who stated that "..the Farnsworth D-15 Test is not designed to separate colour normals from colour defectives. It is also not a test to separate the colour anomalous from the dichromat. It separates sufficiently affected deutans from sufficiently affected protans. It also separates sufficiently affected deutans and protans from those not seri-

ously affected once the *PRESENCE OF THE DEFECT* has been established by *SOME OTHER MEANS*". For example, a strongly affected deutan will confuse green plus yellow (which is lime green to normal observers) with red plus purple plus yellow (which is orange to normal observers). Both colours appear yellow to the deutan patient who is strongly affected. A mildly affected deutan patient will not confuse these colours on the FM D-15 test as the chroma (or saturation) used is strong enough to enable the patient to distinguish between the lime green and orange caps. Although the mild deutan patient perceives these two colours differently from the colours perceived by the normal observer the patient is able to distinguish between them.

The FM D-15 test consists of 15 loose caps and one fixed cap (the reference cap) in one box (all with Munsell value 5 and chroma 5). The hue of each cap has been chosen so that adjacent caps have approximately equal hue differences. When the caps are arranged in order out of their box they form a hue circle. As a result errors can be made across the hue circle (ie patients can place red caps next to green ones or blue caps next to yellow ones).

When doing the test the patient is instructed to arrange the caps in order in the box starting next to the fixed reference cap. Once completed the box is shut, inverted and reopened. A number is seen on the underside of each cap. The order in which the patient has arranged the caps is recorded on a score sheet (see figure 4). Starting at point 'P' the points on the hue circle on the score sheet are connected according to the order given by the patient.

Patients with a colour vision anomaly sufficient enough to affect their performance will make characteristic errors across the hue circle (see figure 4). For example protans or deutans frequently place the purple cap 15 next to the blue cap 1. They then continue to confuse the bluey greens and purples and so on. Tritans tend to confuse cap 7, a yellow-green with the purple cap 15 placing them side by side (see figure 5a).

Subjects with normal colour vision or mild anomalies that are not sufficient enough to affect their performance may make one or two minor errors such as reversing adjacent caps (see figure 5b and 5c). According to the manual their errors will not usually be across the hue circle or they may have only one error across the circle.[7] The major single error that can occur commonly in subjects with otherwise normal colour vision is placing cap 7 (green) next to cap 15 (purple) then caps 14, 13, 12, 11, 10, 9 and 8 (see figure 5d). This occurs because of the marked colour difference between caps 7 and 8; however, it is not considered to be indicative of colour vision anomaly.[6] Minor errors are reported to be common when testing young children.[8,9] In the author's experience such minor errors in those patients who have had a normal result in the screening tests such as the Ishihara warrant further investigation on the FM 100 hue test to rule out more marked colour vision anomaly.

The FM D-15 is designed to differentiate between those patients with congenital anomalous trichromatic colour vision whose daily tasks or work tasks will be affected by the abnormality from those who will not. According to the manual any patient with congenital anomalous trichromatic colour vision diagnosed by another test such as the Ishihara who has a normal result on the FM D-15 test should have almost no difficulty in performing most tasks in which colour vision is a factor.

The FM D-15 test is very portable, quick and easy to perform. As a result it is tempting to use it as a screening tool. However it must be remembered that it *must* be used in conjunction with another colour vision test as the FM D-15 is designed so that mildly affected colour defective patients will *pass*. It is also an effective test to use to monitor the effect of ageing on colour perception.[10]

CITY UNIVERSITY TEST

The City University test was derived from the FM D-15 for use with patients who have problems with the concept of sequencing or ordering needed to put the caps in the correct order in the FM D-15 box. The original aim of the test was to provide a version of the FM-D 15 which was more simple to perform.[11] Like the FM D-15,

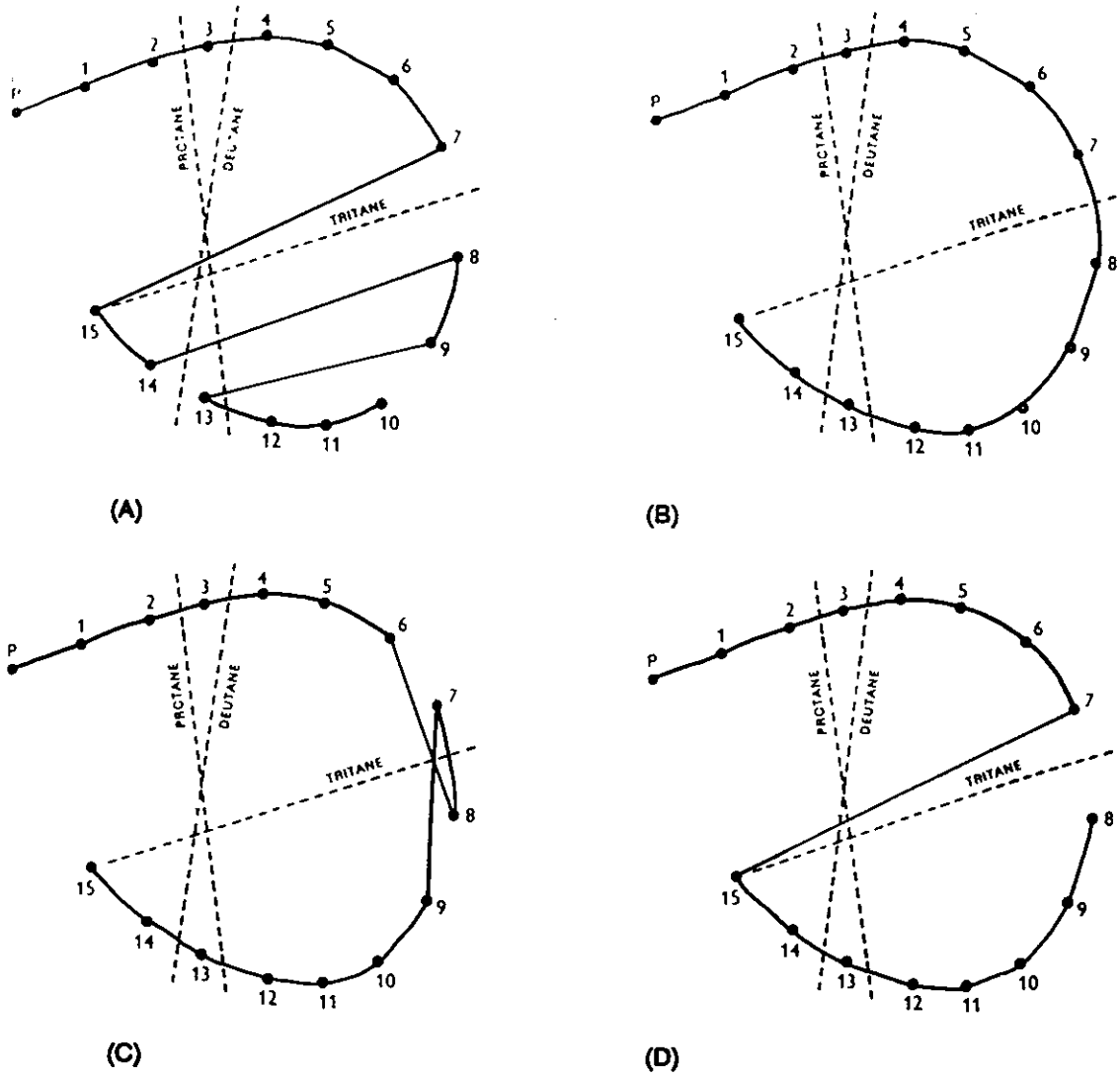

Figure 5: Characteristic error patterns seen on the FM Panel D-15 test; (a) Tritan defect, (b) normal result, (c) normal result with 2 reversals, (d) normal result with 1 major single error.

when used to test patients with congenital anomalous trichromatic colour vision, it distinguishes those who are likely to experience difficulty with their anomaly from those who are not. It was also designed to allow those with only a mild anomaly to 'pass' the test.

The 2nd edition (1980) of the test consists of 11 plates including a control plate. Each plate has one coloured spot in the centre, the central reference spot and 4 surrounding spots known as the comparison spots. Plate A is the control plate.

In each plate the patient is asked to look at the central reference spot and tell the examiner which of the comparison spots is most similar in colour to the central reference spot.

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

Address ..... Patient .....

Examiner ..... Male/Female Date 24 / 11 /1989

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

Illumination (Daylight) Type ..... level .....

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

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

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

Figure 6: City University test score sheet.

The patient's responses (ie the comparison spot to the right R, left L, top T, or bottom B) are recorded on the score sheet provided with the test (see figure 6). If most errors fall in the protan column, for example, then the patient has a protan defect.

According to the test manual, two errors, particularly in plates 7 to 10, suggests that the subject is on the borderline of being handicapped by his/her colour vision anomaly.[12] Errors on 3 or more plates indicates an "unsafe" degree of colour vision deficiency.

In plate A and plates 1 to 6 the coloured spots are 8mm in diameter which subtend 1.5° when viewed at 35cms. Plates A and 1,2,4,5 and 6 contain the same Munsell hues as the FM D-15 with the Munsell value 5 and chroma 5. Plate 3 contains two additional comparison hues with Munsell value 5 and chroma 5.

In plates 7 to 10 the spots are 4mm in diameter and subtend 0.6°. In these plates the Munsell hues are desaturated with Munsell chroma 2. (Munsell value 5 remains the same).

In the second edition of the City University test the control plate (plate A) has a green centre spot which is identical to cap 6 in the FM D-15. The correct comparison spot is the one that is the SAME hue as the reference spot thus is also identical to cap 6 in the FM D-15. It sits directly below the central reference spot. The remaining three comparison spots are all the same purplish hue which is identical to cap 13 in the FM D-15 test. This is designed so that patients with anomalous colour vision can and should respond correctly on this plate. If the patient makes an error or does not respond to this plate there is no point in continuing with the test as the patient is either malingering or their vision is too poor to do the test.

In the remainder of the plates the correct answer is the comparison spot that is the adjacent hue to the central reference spot hue. For example in plate 1, the central reference spot is identical to FM D-15 test cap '14', and the comparison spot below it is identical to FM D-15 test cap '13' which sits adjacent to cap '14' when the FM D-15 caps are placed in the correct order in the box. The normal subject would thus choose the bottom comparison spot (B).

The three remaining comparison spots are chosen from the opposite side of the hue circle and in plate 1 they are the same hues as caps '1', '3' and '8' in the FM D-15. (All these caps are from the other end of the box when the FM D-15 caps are placed in the correct order.)

Thus a patient with a protan defect would choose the comparison spot on the right (R) which is equivalent to the FM D-15 cap 1; a deutan patient would choose the comparison spot on the left (L) which is equivalent to the FM D-15 cap 3 and a tritan patient would choose the comparison spot on top (T) which is equivalent to the FM D-15 cap 8.

As these three remaining spots are comprised of hues from the FM D-15 that represent the most isochromatic confusion for protan, deutan and tritan patients a characteristically incorrect response is given by patients with anomalous colour vision.

When used to evaluate colour vision anomaly

on patients who fail one of the tests designed to test for congenital anomalous trichromatic colour vision, (such as the Ishihara), patients who subsequently pass the City University test have a slight colour vision defect which is not likely to affect their performance in everyday tasks. Those who fail the Ishihara and have between one and 5 errors on the City University test have a moderate colour vision defect and those with more than 5 errors on the City University test can be considered to have a more severe colour vision loss.[12,13]

One criticism of the City University test is that the small targets in the diagnostic plates (plates 7 to 10) make them extremely vulnerable to false positive results thus mixed protan deutan diagnostic responses occur as an artifact of the test format.[11] The targets often give false positives for a tritan defect.

Neither the 1st nor 2nd edition of the City University test, like the FM D-15 test they were derived from, were designed to be used for screening.[11-13] In a study in 1984 Birch[11] tested patients with known colour defects including 64 patients with congenital anomalous trichromatic colour vision and 166 eyes with acquired colour blindness (secondary to diabetes). She reported that neither edition of the test was effective for colour vision *screening*. Her study concluded that the City University test can be used in a test battery to provide information about the severity of the colour defect if a test format other than the FM D-15 is necessary.

A situation where a test other than the FM D-15 was needed to assess colour vision was reported recently in a paper presented by Deveraux,[14] who reported on a population of young adult under achievers learning to use computers. Colour vision assessment with the FM D-15 was not possible in a number of cases as the subjects could not manage the concept of ordering the colours in the natural colour sequence. In every case the subjects were able to perform the City University test.

Just like the FM D-15 test the City University test is quick and easy to perform but it has the added advantage of being able to be used on patients who have problems with ordering the colours into the correct sequence. Once again it is tempting to use the City University test for screening but it should only be used in conjunction with one of the other colour vision tests.

LANTHONY'S DESATURATED D-15 TEST

This test was designed to be used in conjunction with the FM D-15 test; however, it can be used independently as part of the colour vision test 'battery'. The test is performed and is scored in exactly the same way as the FM D-15 test with the caps in each being exactly the same hues as those used in the FM D-15 test. The only difference is that Lanthony's Desaturated test uses lower chroma (less saturated) hue caps than the FM D-15. The chroma is reduced to 2 on the Munsell scale and the value (the proportion of black and white) is increased to 8. As a result there is exactly the same difference between adjacent caps in the desaturated D-15 and the normal FM D-15 but it is more difficult to distinguish between the hues.[15]

If a colour vision abnormality is found when using one or the other colour vision tests the Lanthony's Desaturated test will establish whether or not the defect is likely to affect the patient. As previously stated when a patient is found to have a colour vision defect on one of the other colour vision tests which does not show up on the FM D-15 test it is considered that they are unlikely to be handicapped by their disturbance in colour vision. If a patient with the colour vision abnormality has a normal result on the Lanthony's Desaturated test as well as the FM D-15 test it is extremely unlikely to affect their performance in colour related tasks. Alternately a defect may be apparent on the Lanthony's D-15 test which is not apparent on the FM D-15 test. This adds a further classification to those only mildly affected by their defect.

As the Lanthony's Desaturated test is more sensitive than the FM D-15 it may be used to detect abnormalities in colour vision which are very mild thus not apparent on the FM D-15 test. It may also be used to follow the progression of acquired colour vision loss. One author has reported that the Lanthony's Desaturated test is

more sensitive in detecting deterioration in colour discrimination associated with increasing age.[10]

Once again clinicians should be reminded that this test alone is not a screening test for colour vision abnormalities.

ROTH 28 HUE TEST

This test consists of 28 different hue caps. It was designed to overcome the shortcomings of the FM D-15 and the FM 100 hue; namely that the FM D-15 test has a large variation in hue around the circle so it is not very sensitive to overall diminution of differential shade perception[16] and the FM 100 hue test is very time consuming. The FM 100 hue test provides a better quantitative estimation of colour vision deficiencies than the Roth 28 hue test. However the FM 100 hue test is very time consuming, especially when testing monocularly for acquired colour vision defects and, as the closeness of the hue shades used can prove difficult to some normal subjects, the Roth 28 hue test was designed to be used in its place.

The Roth 28 hue test is made up of every third cap from the 100 hue test (1,4,7,10....82) and the numbers underneath the caps have not been altered. Cap number one is used as the reference cap and the other 27 are arranged in order of successive shades in the one box.

The order that the patient places the caps in the box is plotted directly on to a score sheet graph (see figure 7). Each hue is represented on the graph in the same position as it is on the FM 100 hue test graph. Like the FM D-15 test errors may be made across the colour circle as all the caps are presented to the patient from the one box. On average it takes between 2 and 3 minutes to do the test (per eye).

Results may be normal (in which case the plot looks like a circle) or minor errors can occur, for example reversing adjacent caps. Such errors are said to be insignificant unless they are clustered in one particular area. When abnormalities are present the lines joining the cap numbers cross the hue circle. Like the FM D-15 test these lines may be parallel to the deutan, protan or tritan axes depending on the type of anomaly present (see figure 7).

The Roth 28 hue test also has 2 other axes; tetaran and scotopique. Lines are parallel to the tetaran axis when the patient has an extremely rare acquired colour vision anomaly known as tetaranopia. This is a type of blue yellow colour blindness in which blue and yellow are confused and red and green are not. This differs from other forms of blue yellow 'blindness' where red and green are confused and blue and yellow are not.

If colour vision testing is performed in scotopic conditions responses are parallel to the scotopique axis. This may also occur when a patient has total achromatopsia (ie totally colour blind) or alternately the responses from such patients may be totally confused. (The term scotopique suggests scotopic or rod vision only).

The main problem with the Roth 28 hue test is that sometimes the first cap put into the box may be incorrect. The patient will then arrange

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ROTH 28 HUE TEST SCORE SHEET

NAME:_____ HOSP NO:_____

AGE/DOB:_____ SEX:_____

DATE TESTED:_____

RIGHT EYE: 1 82 79 76 40 43 73 46 49 70
67 52 55 61 64 58 37 28 31 4
28 25 7 22 10 19 13 16 - DEUTAN

LEFT EYE 82 1 4 7 10 13 19 16 22 25
28 31 34 37 40 43 46 49 79 52
76 55 73 58 67 61 64 70 - PROTAN

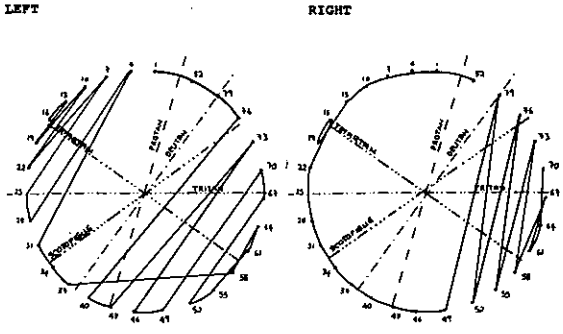

Figure 7: Roth 28 hue test score sheet.

the next couple of caps incorrectly then returns to normal. This will result in a few responses being parallel to an axis of abnormality in a patient with normal colour vision. Its advantages are that it is easier to do than the FM 100 hue test and it is reported to be less confusing then the FM D-15 test.

The literature reviewed suggests that this test should not be used in isolation for screening; however, it can be used in conjunction with other colour vision tests for screening.[17] (In practice the Roth 28 hue test can be used in isolation in cases of suspected congenital anomalous trichromatic colour vision; however, like the FM D-15, very mild cases can 'pass' the Roth 28 hue test.)

In the case of a suspected acquired colour vision abnormality the Roth 28 hue test may be used for screening; however, if a defect is found it should be followed by a FM 100 hue test to quantify the defect so that the defect can be monitored for future progression. The test can also be used to differentially diagnose between colour vision anomalies.

FARNSWORTH F2 TRITANOPIC PLATE

This test consists of a single pseudoisochromatic plate (ie plates such as those used in the Ishihara test). The background spots are light mauve in colour and there are 2 overlapping squares superimposed on the background in the same manner as the numbers on the background in the Ishihara test. To the normal observer one square is yellow-green and the other is blue. The tritan patient sees only the blue square.

This is the only one of the FM tests mentioned that does not test the patient's ability to discriminate between hues. It can be used as a screening test for a tritanopic defect.

CONCLUSIONS

When testing for possible colour vision anomalies clinicians generally want to screen for defects, diagnose the type of defect present and grade (or score) the severity of the defect. Unfortunately, with the exception of the FM 100 hue test, no individual Farnsworth type test can fulfil all three requirements. Although the FM 100 hue test will perform the task of screening it is extremely impractical as it is so time consuming, especially when testing must be performed monocularly when an acquired defect is suspected.

When attempting to screen for colour vision defects possible congenital defects must be separated from possible acquired defects.

(a) Acquired defects

When patients complain of symptoms such as altered colour perception, desaturated appearance of colours, reduced visual acuity (without adequate explanation), central visual field loss, or when the patient is taking certain drugs or toxic substances or if there are any other observations which may be associated with altered colour vision *acquired* colour vision loss would be suspected.

In such a case the FM 100 hue test (performed monocularly) is the most appropriate. The Roth 28 hue test which is a less time consuming alternative will usually reveal an acquired defect but it does not give an exact score. (If the Roth 28 is performed first and the result is normal a FM 100 Hue test should be performed.)

Alternately the FM D-15 test, City University test or the Lanthony Desaturated D-15 may be performed as a starting point but it must be emphasised that if the results on these tests are normal further investigation *must* be carried out.

If a defect is found on any test a FM 100 hue test must be performed *if* the severity of the defect is to be graded. This is most important as acquired defects may be progressive passing through trichromatic, dichromatic to a monochromatic stage. The FM 100 hue test is the only one of the Farnsworth type tests to give a quantitative assessment of the defect and thus can be used to assess the effect of treatment in some instances.

With the exception of the Hardy Rand Rittler test (HRR) *none* of the pseudoisochromatic tests (ie Ishihara, Guys, Matsubara, SPP etc) tests are appropriate to test for acquired colour vision defects. These tests were designed to test for *congenital* red green colour 'blindness' (ie anomalous trichromatic colour vision).

(b) Congenital defect

When screening for *congenital* anomalous trichromatic colour vision which affects up to 8% of males and 0.4% of females, the pseudoisochromatic tests such as the Ishihara or, alternately, the anomaloscope tests are generally the most appropriate as they are quick and easy to use. If an abnormality is found it can be classified into protanomaly or deuteranomaly by the pseudoisochromatic tests. It can be roughly graded into mild, moderate or severe using the FM D-15 test, the City University test or the Lanthony Desaturated D15 test. Exact quantitative grading can be given by the FM 100 hue test but this is probably unnecessary as the condition is non progressive. Testing for suspected congenital anomalous trichromatic colour vision can be done binocularly to save time as the condition is usually bilateral and symmetrical.

Very rarely cases of suspected congenital anomalous trichromatic colour vision are not found on the pseudoisochromatic tests. In such cases the FM 100 Hue or an anomaloscope test should be performed. (Anomaloscopes can be used as a quick, easy and effective screening test for congenital or acquired colour vision loss; however, as they are so expensive they are rarely available in clinics.)

With the exception of anomaloscopes there is no individual quick and easy test that will screen for both congenital and acquired colour vision loss giving a differential diagnosis and a qualitative assessment. As a result a battery of tests may be necessary using a 'less sophisticated' test to find a defect then a more 'sophisticated' (and usually more time consuming) test to evaluate it. However it is imperative that the appropriate battery of tests be used, especially when initially screening.

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THE EFFECT OF SPECTRAL COMPOSITION OF LIGHTING ON VISUAL PERFORMANCE OF PERSONS WITH RETINAL PATHOLOGY

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Abstract

Clinically it has been observed that visually impaired patients have definite preferences for lamp lights of certain wavelength compositions. A pilot study was conducted to assess the visual function of subjects with retinal pathology under three different lighting conditions. It was found that subjects with foveal function had higher levels of visual acuity under green light. Subjects without foveal function did not show this preference. All subjects demonstrated greater levels of contrast sensitivity under blue and green lights.
Key words: *Wave length, colour, visual acuity, contrast sensitivity, retinal pathology, visual function.*

White light is composed of many waves each of a slightly differing length. Groups or bands of wave lengths are seen as different colours. Artificially created light ie lamp light is composed of a broad range of wave lengths or specific groups of wave lengths. Light can be described in terms of the colour (spectral distribution), or in terms of the intensity (brightness).

Several authors have studied the effect of luminance levels on visual acuity. Sheedy, Bailey and Raasch 1984[1] found increased luminance within a specified range improved visual acuity on a letter chart. Conversely Comerford, Thorn and Corwin 1987[2] found contrast sensitivity in myopes did not vary significantly with changes in luminance levels. Brown and Garner 1983[3] and Brown, Zadnik, Bailey and Colenbranders 1984[4] studied the effects of luminance on contrast sensitivity and visual acuity in patients with senile macular degeneration. This work indicated that peak contrast sensitivity function was moved to the lower spatial frequencies at all luminance levels. Visual acuity in these patients showed a greater than expected decrease at lower luminance levels. Hyvarinen, Rovamo, Laurinen and Peltomaa 1981[5] reported the use of contrast sensitivity as an indicator of visual performance for patients with retinitis pigmentosa at low levels of illumination.

Clinically it has been observed that patients with central field loss show a preference for artificial illumination of the cool white, daylight type light as opposed to warm white light. Ninety percent of patients with senile macular degeneration indicated a preference for the cool white, daylight lighting.

The human retina has three cone types each responding maximally to wavelengths of light in the blue, green and red bands of the spectrum Davson 1980.[6] The clinical response of patients indicating preference for lamp lighting of specified wave lengths and the physiological occurrence of different cone types within the human retina suggests a relationship between the viable retina present and the wavelength of light for optimal visual function. From the literature it appears that luminance level can influence visual function of both normal and visually

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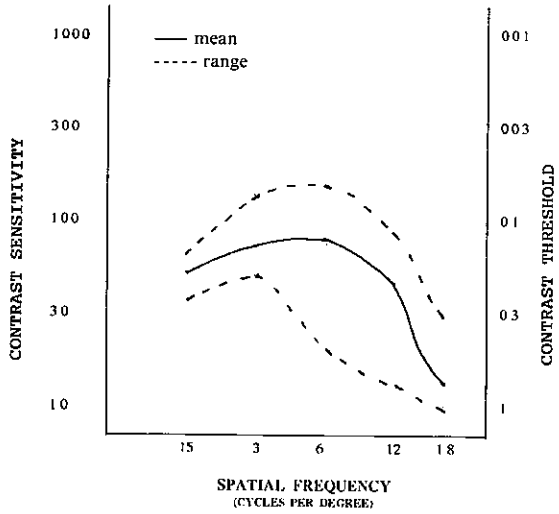

Figure 1: Contrast sensitivity blue light/normals.

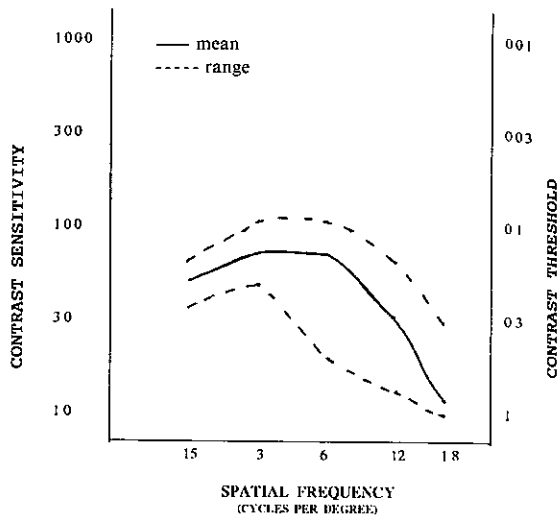

Figure 3: Contrast sensitivity red light/normals.

impaired subjects. This research was intended to study the effect of specified wavelengths of light on the visual function of subjects with peripheral or central field loss.

METHOD

Thirty one subjects were tested. Twenty were visually normal, six had central retinal pathology with absolute central scotoma and five had peripheral retinal pathology with reduced peripheral fields.

Each subject was assessed for visual function using a Log-MAR distance acuity test and the Vistech distance contrast sensitivity system VCTS 6500. Each of these measures of visual function were performed in a light proof room using only Tungstram blue, Tungstram red and Sylvana green 40 watt fluorescent tubes. Luminance for each of these tubes on each of the test instruments was also measured.

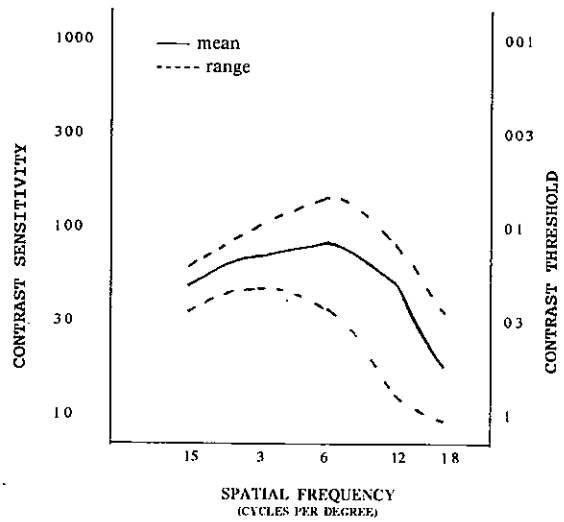

Figure 2: Contrast sensitivity green light/normals.

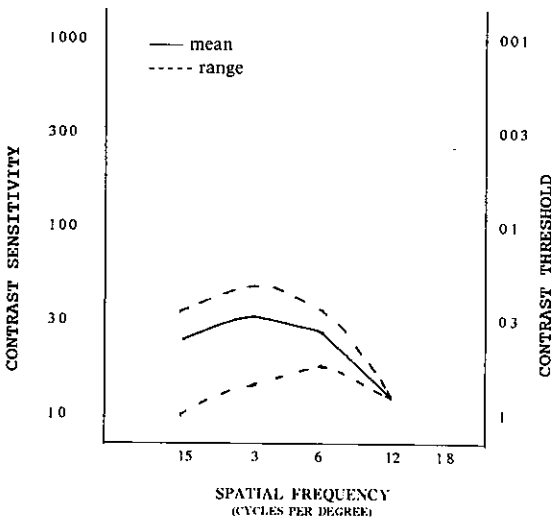

Figure 4: Contrast sensitivity blue light/peripheral field loss.

TABLE 1
Visual Acuity of Normal Subjects Under Specified Light Conditions

Subject	Lighting Condition					
	Red		Green		Blue	
	RE	LE	RE	LE	RE	LE
1	4/4	4/3	4/2.5	4/2.5	4/3	4/3
2	4/4	4/2.5	4/4	4/2	4/4	4/2.5
3	4/2	4/2	4/2	4/2	4/2	4/2
4	4/3	4/3	4/3	4/2.5	4/4	4/4
5	4/3	4/2.5	4/2	4/2	4/3	4/2.5
6	4/3	4/3	4/2	4/2.5	4/3	4/2.5
7	4/3	4/3	4/2.5	4/2.5	4/3	4/2.5
8	4/5	4/4	4/4	4/3	4/5	4/3
9	4/4	4/3	4/3	4/2.5	4/4	4/3
10	4/6	4/6	4/5	4/5	4/6	4/8
11	4/11	4/4	4/2	4/2	4/2.5	4/2.5
12	4/3	4/3	4/3	4/3	4/3	4/3
13	4/2	4/2.5	4/3	4/2.5	4/2	4/2
14	4/3	4/3	4/2.5	4/2.5	4/2.5	4/2
15	4/4	4/3	4/2	4/2	4/2.5	4/2
16	4/3	4/4	4/3	4/3	4/2.5	4/4
17	4/4	4/5	4/2	4/2.5	4/2.5	4/3
18	4/2.5	4/3	4/2	4/2.5	4/2.5	4/2.5
19	4/3	4/3	4/2	4/2	4/2.5	4/3
20	4/2.5	4/3	4/3	4/3	4/4	4/3

RESULTS

The visual acuity of normal subjects (table 1) was enhanced under green lighting and reduced under red lighting. This was significant at the 95% level of confidence using one factor ANOVA repeated measures Fisher PLSD 0.114 and Scheffe F test 14.047. Contrast sensitivity function of the normals Figures 1, 2 and 3 indicated that the finest gratings were seen under the blue and green light sources. The finest gratings with lowest contrast were seen under the green light source. The differences in contrast sensitivity performance were significant at the 95% level of confidence using one factor ANOVA repeated measures: blue versus red Fisher PLSD 2.972 and Scheffe F-test 13.294 and green versus red Fisher PLSD 2.972 and Scheffe F-test 41.341.

TABLE 2
Visual Acuity of Centre Field Loss Subjects Under Specified Lighting Conditions

Subject	Lighting Condition					
	Red		Green		Blue	
	RE	LE	RE	LE	RE	LE
1	4/32	—	4/32	—	4/40	—
2	4/40	4/40	2/20	2/20	4/40	4/40
3	2/40	1/40	1/24	1/32	2/40	1/40
4	1/32	—	1/32	—	1/32	—
5	4/20	1/32	4/16	1/32	4/20	1/32
6	1/24	1/40	2/32	2/32	2/40	2/32

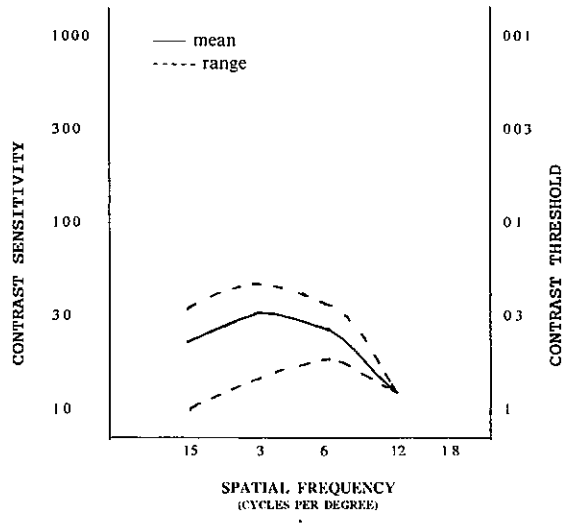

Figure 5: Contrast sensitivity green light/peripheral field loss.

The central field loss subjects performed slightly better under green light in terms of visual acuity (Table 2). The differences between blue and red and green and red being significant at the 95% level Fisher PLSD 5.644, no significant difference was found between blue and green

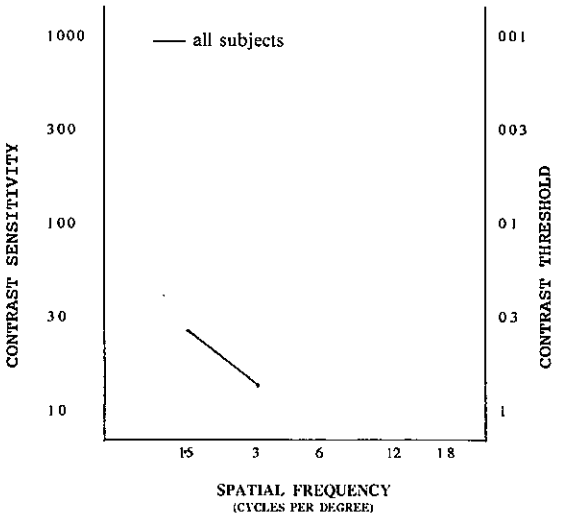

Figure 6: Contrast sensitivity red light/peripheral field loss.

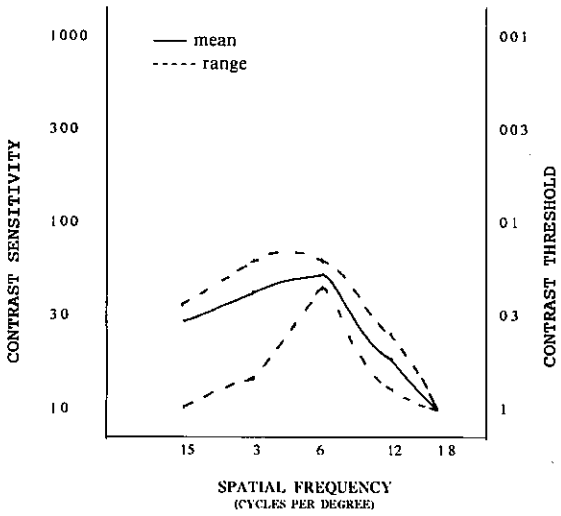

Figure 7: Contrast sensitivity blue light/centre field loss.

conditions. The finest contrast sensitivity gratings were seen under blue light with the lowest levels of contrast being seen under both blue and green light. Contrast sensitivity was reduced under red light. (Figures 4, 5, 6). The differences between blue and red, and green and red were significant at the 95% level one factor ANOVA Fisher PLSD 5.644.

The visual acuity performance of peripheral field loss subjects was better under green light

TABLE 3
Visual Acuity of Peripheral Field Loss Subjects Under Specified Lighting Conditions

Subject	Lighting Condition					
	Red		Green		Blue	
	RE	LE	RE	LE	RE	LE
1	4/24	4/32	4/16	4/32	4/20	4/24
2	4/32	4/40	4/10	4/10	4/12	4/12
3	4/16	4/12	4/6	4/6	4/8	4/8
4	4/40	<2/40	4/6	2/40	4/10	2/40
5	2/40	4/24	2/8	4/8	2/12	4/10

and the most reduced under red light (Table 3). This was significant at the 95% level one factor ANOVA blue versus red Fisher PLSD .085 and Scheffe F-test 7.646 and green versus red Fisher PLSD .085 and Scheffe F-test 16.03. The finest gratings and lowest contrast being seen under both blue and green lights (Figures 7, 8, 9). This was significant at the 95% level one factor ANOVA blue versus red Fisher PLSD 5.649 Scheffe F-test 32.41 and green versus red Fisher PLSD 5.649 Scheffe F-test 36.397.

Comparison of the subjects with pathology indicated a significant difference in visual acuity results between central and peripheral field loss groups under both green and blue light; green one factor ANOVA Fisher PLSD 0.146 Scheffe F-test 21.407; blue Fisher PLSD 0.115, Scheffe

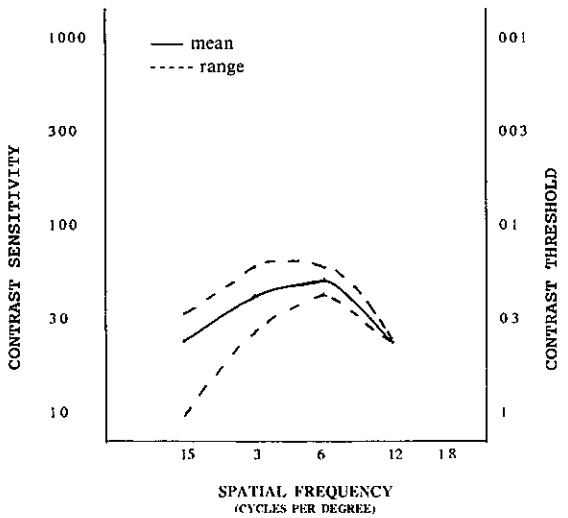

Figure 8: Contrast sensitivity green light/centre field loss.

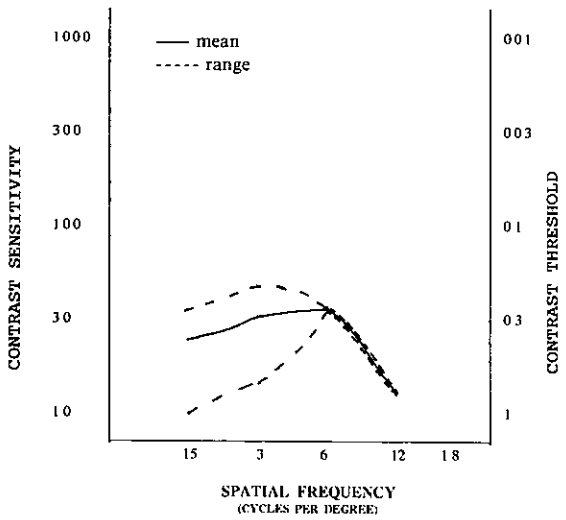

Figure 9: Contrast sensitivity red light/centre field loss.

TABLE 4
Mean Light Intensity

Light Type	Light Intensity (lux)
Red	3.83
Green	57.08
Blue	16.50

F-test 20.627 at the 95% level. The only significant difference for contrast sensitivity was under green light, one factor ANOVA Fisher PLSD 12.243 Scheffe F-test 6.764 at the 95% level.

DISCUSSION

Normal subjects and subjects with peripheral field loss achieved their best levels of visual function under green light. Subjects with central field loss did not perform significantly better under green or blue light. All subjects recorded the most reduced visual function under red light. Subjects with central field of vision do appear to perform better under green light whereas subjects lacking central field are less influenced by colour. The intensity of the light (Table 4) may also influence this result as the red was the least intense light source, green the highest with blue between but toward the low intensity of red. This data tends to support Sheedy, Bailey and Raasch 1984 that visual acuity will increase with increased luminance. The central field loss group showing no preference between the blue and green lighting reflects the findings of Brown, Zadnik, Bailey and Colenbranders 1984 that persons with senile macular degeneration are less likely to show improved visual acuity with increased luminance.

The extent to which intensity as compared to colour composition influenced visual function cannot be deduced from this experiment. Visual function did improve with increased luminance when taking the visual acuity parameter in isolation. However when contrast sensitivity is considered all three groups of subjects demonstrated improved visual function under both blue and green lights, indicating intensity was not the sole factor.

This study appears to support the hypothesis that the wavelengths present in light can influence visual performance. There is some support for the clinical observation that the wavelengths of light which will enhance visual function will vary in the presence of retinal pathology and subsequent field loss. The extension of this study to a wider range of light sources and at controlled levels of illumination would be of benefit to the field of visual rehabilitation and to the commercial lighting industry.

ACKNOWLEDGEMENT

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THE ORTHOPTIST AND DRIVING SKILLS

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Abstract

The focus of this paper is on a new role for the orthoptist using orthoptic skills in assisting a client with physical limitation to gain a driver's licence. The paper also highlights a way in which orthoptists can liaise with occupational therapists to help the client.

Key words: *Driver rehabilitation, interdisciplinary role, Klippel Feil Syndrome, vision and driving.*

INTRODUCTION

Visual function is an essential component of the driving skill. In New South Wales, the Road Traffic Authority[1] requires drivers holding an automobile licence to have a visual acuity level of 6/12 in either eye or two eyes together and a field of vision that extends 130° monocularly or binocularly. The additional ability to move the eyes fully into all positions of gaze while maintaining a single and clear image is not a legal requirement but is beneficial to safe driving. The best possible visual function for a person learning to drive will help the attainment of driving skills.

A case study of a client with Klippel Feil Syndrome who wanted to gain a driver's licence is used to illustrate this extension of the orthoptist's role. The paper will describe the physical features of the client and link them to the difficulties which were encountered in gaining driving skills. The adaptations that were made to the car to help the client will be described and the interaction between the orthoptist and the occupational therapist reported.

CLIENT FEATURES

1. General Features

Carolyn, aged 18 years, contacted the Driving Rehabilitation Centre at Cumberland College with the intention of learning to drive. She was diagnosed as having Klippel Feil Syndrome, features of which are listed in Table 1. Specifically, Carolyn has a short stature and severe kyphoscoliosis (humped back and lateral curvature of the spine). She has severely restricted neck movement and very limited trunk movement in all directions. She has limited lung capacity and requires a respirator at night in order to maintain adequate breathing. Due to her physical status, her physical function was limited to gross body abilities i.e. she is unable to run effectively or carry out strenuous gross motor activities. She fatigues easily and is prone to shortness of breath.

Carolyn's cognitive capacity appeared normal, being educated to year 12 and gaining admission to university, she has led a relatively quiet life, with regular admissions to hospital. Being unable

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TABLE 1
Klippel Feil Syndrome (Duke Elder)[2]

Features:
- congenital condition
- fusion of a number of cervical vertebrae
- associated with elevation of the scapula
- spina bifida in the cervical region
- dysplasia of the cervical cord

Appearance:
- head appears to rise directly from the thorax
- some degree of torticollis
- head movements severely restricted
- bimanual synkinesia of the hands
- ocular signs:
  - Duane's Retraction syndrome
  - total external ophthalmoplegia
  - congenital paralysis of conjugate lateral gaze

to carry out normal social and leisure activities, her level of maturity was quite young for her age.

2. Ocular Features

An orthoptic assessment revealed that Carolyn has:

(a) a right esotropia with hypotropia, with a face turn to the left (Figure 1). Carolyn could voluntarily change fixation to the right eye by adopting a face turn to the right or shutting her right eye. When fixing with the right eye she has a left esotropia with left hypertropia. Carolyn cross fixates.

(b) slight myopia. Her visual acuity with glasses is 6/9 RE, 6/6 LE and binocularly 6/6 corrected.

(c) ocular movements revealed a bilateral Duane's retraction syndrome with no abduction of either eye beyond the midline (Figure 2). Each eye retracted on attempted adduction. On attempted abduction the adducting eye elevated with the left eye, elevating further than the right eye. There was a torsional movement of the right eye on attempted dextro elevation.

The eyes could directly elevate and depress but the above limitations continued as the eyes moved into dextro and leavo elevation and dextro and leavo depression.

(d) The visual fields were full using the Goldmann perimeter. As each eye had limited mobility, the extent to which the eyes could follow into the peripheral field was plotted. The arc perimeter was used and the procedure modified so that Carolyn, with her chin in the middle of the two chin rests, followed a print target into the periphery. She was asked to state when the print went out of focus, denoting loss of foveal fixation. In Figure 3a, the solid lines show the extent to which the target could be foveated. The dotted line indicates the normal peripheral field tested with both eyes open. Comparison of Carolyn's responses with those of subjects with normal eye movements (Figure 3b) shows that, for Carolyn, the foveated field is considerably smaller. This confirmed the effects of her limited eye movements.

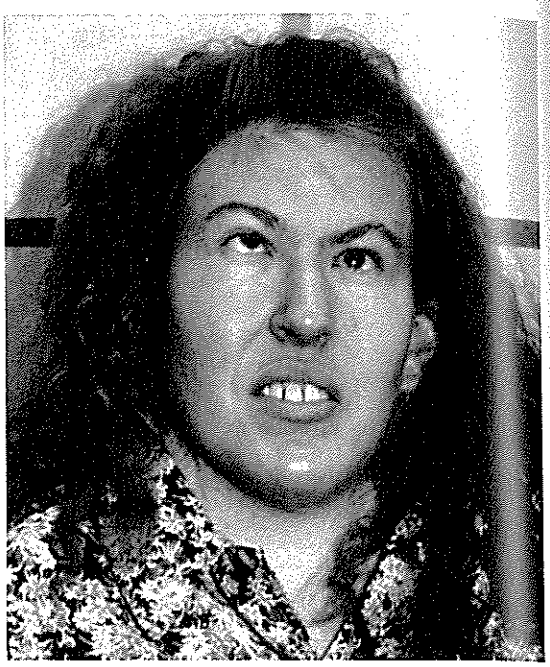

Figure 1.

CLIENT ABILITY AND DRIVING

Carolyn has problems in the following areas:

- Visual

With the aid of head movements she is able to cross fixate so that she uses the right eye to see the left field and the left eye to see the right field. By cross fixation, Carolyn is able to use the appropriate side mirrors. She is unable to use her eyes to see to the extreme

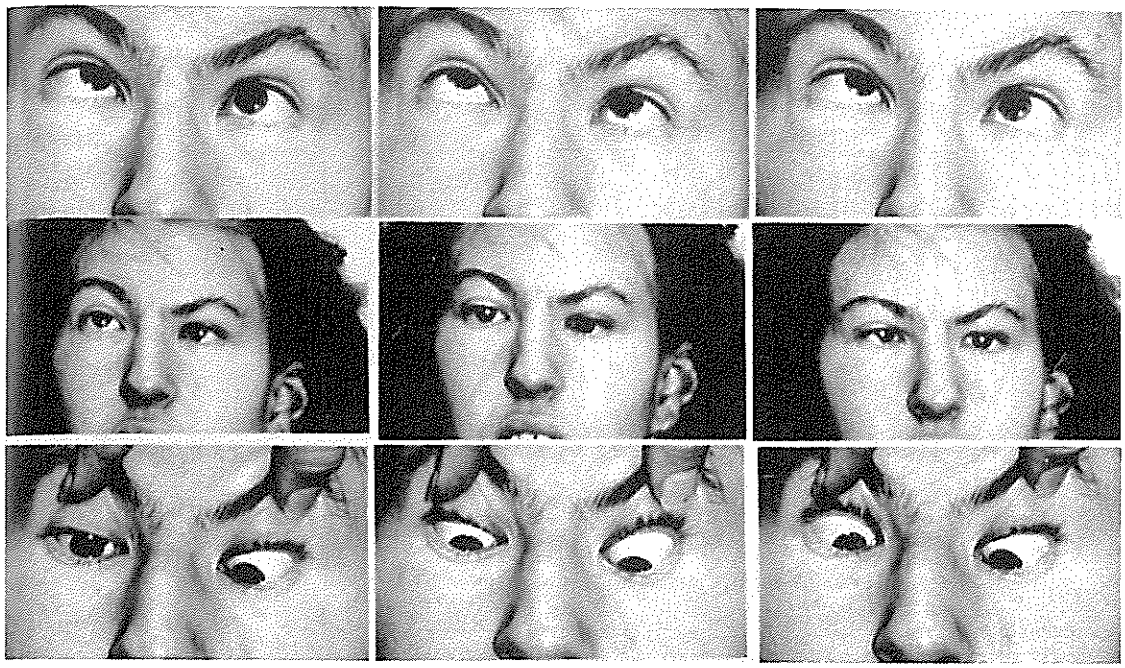

Figure 2.

right or left. Elevation movements to use the rear view mirror are adequate.

- Body Rotation
  Carolyn has limited head and body rotation. Physical support to gain visual information from the periphery is not possible.

An assessment by the occupational therapist revealed that:

- Sitting Posture
  Carolyn's short stature necessitates low seating with good back support to enhance comfort and reduce back pain. Any prolonged sitting in one position induces discomfort. Therefore, when driving, Carolyn needs to be well supported and be able to have regular postural changes.
- Driving Position
  Most car seats encourage hip flexion of less than 90°. This is most uncomfortable for Carolyn, so a seat adaptation is necessary to raise her pelvis to be in line with her knees. An extended back rest was also utilized, enabling good back and neck support and to prevent whiplash or back injuries.
- Physical Abilities
  Carolyn has limited upper limb strength, with reduced range of motion of both shoulders. Extensive physical effort induces back pain and fatigue. Lower limbs function is adequate for driving, with good strength and mobility of limbs.
- Endurances
  Carolyn tires easily, both physically and from the point of view of her lung capacity. Endurance is therefore limited and it was suggested she drive an automatic vehicle with power steering.
- Tolerance
  Initially, Carolyn developed back pain and shortness of breath after only 10 minutes of driving. Following an extensive programme and the reduction of back and neck strain with the use of mirrors, Carolyn can drive pain free for one full hour.

ACTIONS TO ASSIST DRIVING

Following the initial assessment by the orthoptist and the occupational therapist, Carolyn was

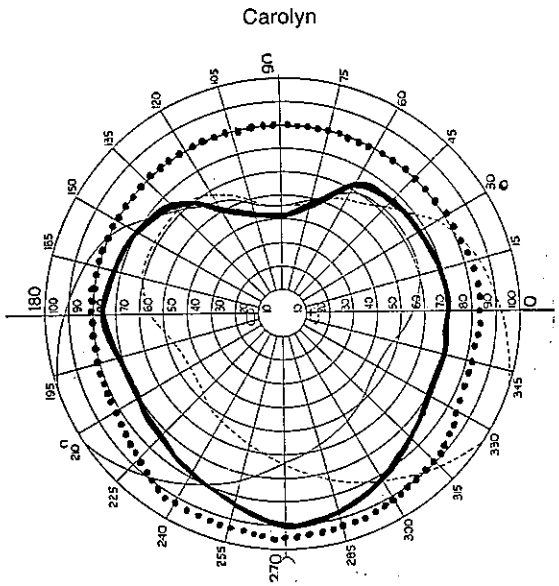

Figure 3a.

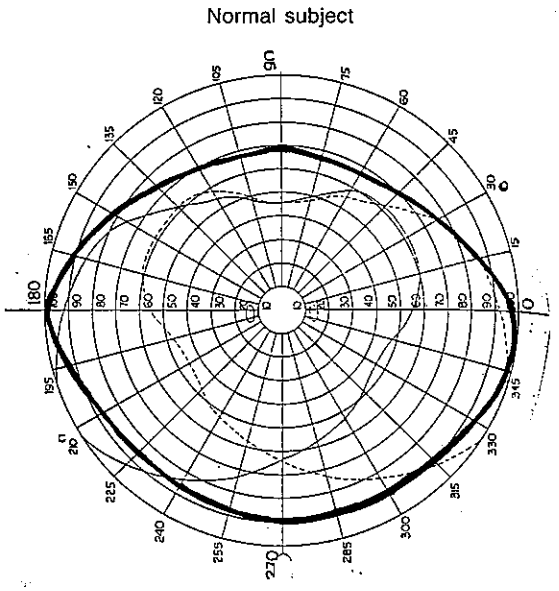

Figure 3b.

taken for an "on-road assessment" with a qualified driving instructor in a car with dual controls. The occupational therapist was present to observe, monitor and disclose the reasons for problems that occurred while Carolyn was driving. Following the assessment, modifications to her position were made. A driving programme was also designed to try and overcome Carolyn's difficulties.

At the initial on-road assessment, the orthoptist was also present to observe Carolyn's eye and head movements and to determine if her ocular limitations were hindering her ability to learn to drive. The orthoptist was positioned immediately behind the driver and observed the eye movements through the rear vision mirror. The head movements were directly observed.

It was noted that Carolyn has difficulty at intersections when she has to look to the extreme right and left to see if the road is clear. She was observed to sit forward using the steering wheel for support and rotate her trunk to bring the visual area into view. While that is satisfactory when the car is stationary, for instance at cross roads, such an action when trying to change lanes means the steering wheel is rotated in the direction of the trunk movement and the car moves off into that direction.

To help overcome the ocular problem, two additional mirrors were positioned at the top of the windscreen directly in front of the driver (Figure 4). The mirrors were placed with the reflecting surfaces towards each other with the adjacent tips towards the windscreen. Carolyn was instructed to move her head and change fixation, to use the mirrors to gain a view of the extreme right and left. In order to see to the right, she had to fix with her right eye and look into the left mirror. To see to the left she fixed with the left eye and used the right mirror. Figure 5 demonstrates Carolyn's normal field of vision plus the effect of the special mirrors.

Trunk rotation also caused some discomfort and it was felt advisable to try and avoid the action. The occupational therapist advised an adjustment in the height and position of the

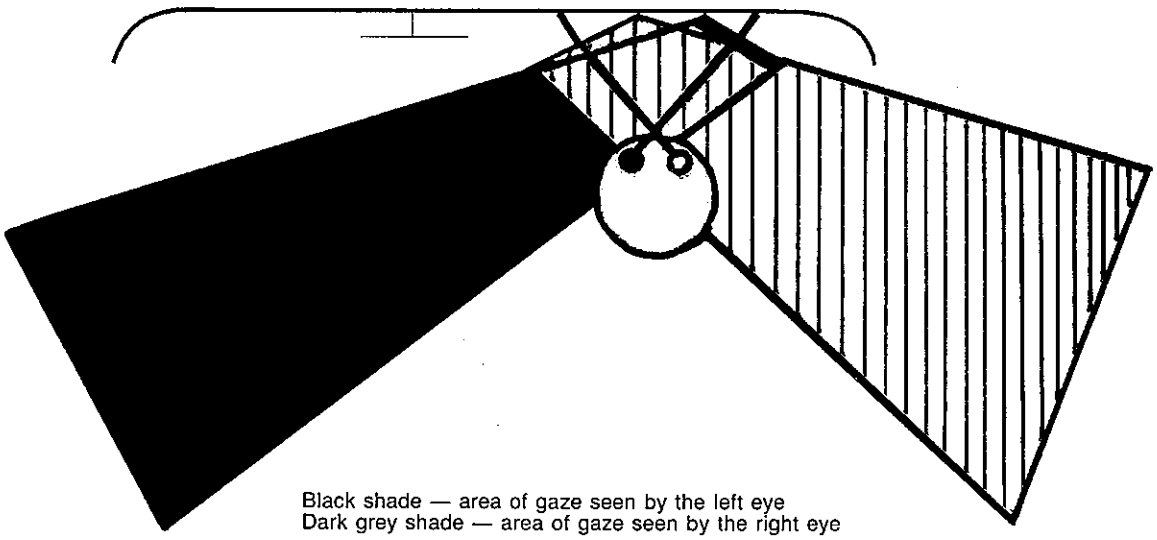

Figure 4: Mirrors positioned to allow appreciation of extremes of gaze without head movements.

driver's seat to enable Carolyn to work with the controls and increase her comfort.

The driving instructor followed up all the guidelines given by the orthoptist and the occupational therapist, ensuring that they were carried out during the instruction period.

To encourage Carolyn's eye movements she was set the task of practising fast fixation change e.g sitting with her back to the television and looking into a mirror to see the picture, then changing fixation to some printed material placed in front of her.

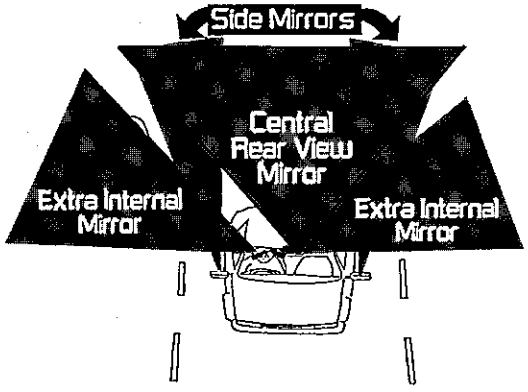

Figure 5: Representative of visual information gained by rear view, side mirrors and additional internal mirrors.

As part of the routine with the Driving Rehabilitation Centre, follow-up assessments were carried out with the occupational therapist present as well as the driving instructor. On one other occasion, the orthoptist was present to evaluate Carolyn's progress.

With the aid of the team approach Carolyn gained her driving licence.

DISCUSSION

The fact that Carolyn successfully gained her driver's licence serves as a good example of team work involving the liaison between the orthoptist, the occupational therapist and the driving instructor.

This is a new area for orthoptists. Conventional skills were used to demonstrate the capabilities and limitations of a client who has many problems to overcome in achieving a personal goal: to drive a car. The orthoptist's knowledge applied to the client's need lead to a variation in assessment approaches, assessment in an environment outside the conventional clinical setting and the development of approaches which adapted the conventional driving set up to enable the client to drive safely and comfortably within the requirements of the Roads and Traffic Authority.

In addition to the benefits for the client, the orthoptist developed an additional role, which is to educate the occupational therapist on how to detect the presence of eye conditions which may limit an individual's driving skills. This has lead to a heightened understanding of the need for, and value of, orthoptic guidance on ocular function in driving.

The orthoptist's involvement with the occupational therapist has highlighted the complexity of the driving process and the need to consider the many components involved in becoming a licensed driver. The orthoptist has gained knowledge about the physical requirements for the driver and the need to integrate the ocular skills with these requirements. Liaison with the occupational therapist has lead to the development of mutual respect for each other's profession and an increased knowledge of the requirements for driving skills.

The Driving Rehabilitation team at Cumberland College includes an orthoptist when there is indication that the ocular function of a client is outside normal limits. In these situations, an orthoptic assessment is carried out and guidance provided about the effects of any existing ocular condition on driving skills. Should any orthoptic treatment be required, the client is required to seek ophthalmological assessment.

The involvement of a team whose members have expertise in recognising the components that are necessary for driving, namely the occupational therapist for physical and cognitive skills, the orthoptist for visual skills and the driving instructor for teaching the driving process. The team provides a good model for members of the public who have special needs and who wish to drive.

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BLOWOUT FRACTURE

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Abstract

This paper reviews 29 cases of blowout fracture seen over a three year period. It outlines the theories on the mechanism of blowout fracture and concludes that it is most likely caused by a combination of bone and global force transmission. The ocular motility restriction is no longer thought to be due to muscle entrapment, but rather traumatic disruption to the connective tissue septa and the orbital tension, either by entrapment or fibrosis. Ten patients were noted to have visual field damage and emphasis is placed on routine visual field testing at the initial examination. The poor results achieved in young patients with blowout fracture were consistent with other surveys.

Key words: *Blowout fracture, mechanism, connective tissue, optic nerve damage, childhood blowout.*

INTRODUCTION

This paper presents a clinical overview of twenty nine patients with blowout fractures seen over a three year period. It details the clinical findings and discusses current thoughts on the mechanism of the fracture and its management.

Blunt trauma to the periorbital region may result in fractures of the orbital bones and or soft tissue damage. 'Blowout' fracture is a term first introduced by Smith and Regan in 1957[1] after a cadaver experiment. It was initially used to describe a traumatic fracture of the orbital floor with the rim intact. The term is now used in a less specific sense and encompasses similar fractures of other orbital bones. It is important to differentiate the two types of blowout fracture:

1. Pure blowout — rim intact
2. Impure blowout — rim involved.

ANATOMY

It is relevant to consider the anatomy of the bony orbit and some of its important relations, as understanding of this sheds some light on the nature of blowout fracture and the clinical consequences.

Seven bones take part in the formation of the orbit (Wolff):[2]

ROOF — consists of two bones, the major portion being formed by the orbital plate of the frontal bone. It is extremely thin, translucent and fragile, except where it is formed by the lesser wing of the sphenoid bone.[2] Traumatic fracture of the roof is uncommon and complex. It occurs as a result of high velocity trauma and the thin orbital plate of the frontal bone tends to be displaced downwards and is termed a 'blow in' fracture. It is not included in this paper.

MEDIAL WALL — consists of four bones, the largest of which is the orbital plate of the ethmoid bone. This is by far the thinnest orbital wall. Isolated blowout fracture of the medial wall is uncommon. Supposedly the numerous bony

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septa of the ethmoid air cells give the lamina some support. The continuity of the orbital plate of the ethmoid with the thin unsupported portion of the orbital floor facilities blowout in this area.[3] The optic foramen is at the posterior limit of the orbital plate of the ethmoid and therefore severe fractures may result in optic nerve damage. The naso-lacrimal duct and its bony canal are vulnerable in medial wall fractures.

FLOOR — consists of three bones, the largest of which is the orbital plate of the maxilla. The floor is separated from the medial wall by a fine suture and this area is vulnerable to blowout fracture. The floor lies over the maxillary sinus and is therefore unsupported. The infra orbital canal runs in the floor of the orbit and opens at the infra orbital foramen. It transmits the infra orbital nerve and anaesthesia of this is a frequent sign of blowout.

LATERAL WALL — formed by two bones. This is the thickest of the orbital walls and is especially strong at the orbital margin. The most posterior portion is the thinnest, and therefore more vulnerable to blowout fracture.

ORBITAL MARGIN — formed by the frontal bone superiorly, and by the zygomatic bone and the maxilla inferiorly. The strong superior orbital rim protects the thin orbital roof. A fractured zygoma is frequently encountered in impure blowout.

MECHANISM OF INJURY

The mechanism of blowout is a controversial issue. There are two main theories, the hydraulic theory and the buckling theory, both proposed as a result of cadaver experiments.

1. HYDRAULIC THEORY: Proposed by Smith and Regan in 1957.[1] Basically the theory proposes that blunt trauma transmits a force to the globe. The resulting increase in the intra orbital pressure results in fractures of the weaker plates of the maxilla and ethmoid. The orbital contents are forced into the fracture site.
2. BUCKLING THEORY: Proposed by Fujino in 1974.[4]

   This theory proposes that the blunt trauma transmits a force to the bony orbital rim, resulting in a transient deformation, with transmission of the force to the weaker portions of the orbit. The entrapment of soft tissue is explained by the different rate of recovery of bone and soft tissue after impact,[5] the recovery of soft tissue being slower than bony structures.

In a recent summary of current theories of the mechanism, Kersten[6] proposed that an isolated hydraulic effect, which is probably the most widely accepted theory at the moment, is highly unlikely. He feels it is inconsistent with the clinical findings in three major areas.

1. The very nature of the trauma would impact globe and rim. Very few injuries isolate the globe on impact force.
2. The mechanism of hydraulic force would imply a considerable force on the globe itself. Fujino,[5,7] proposes hydraulic pressure requires a force up to ten times greater than the force required to produce buckling of the orbital floor.[8] Thus one would expect a high incidence of ocular injuries with hydraulic transmission, but this is not the case in most surveys.
3. The theory of hydraulic transmission would imply that the thinnest and weakest bones would be affected. The most common site for blowout is the posterior medial aspect of the floor.[9] There is a low incidence of isolated medial wall blowout, although this is by far the thinnest bone.

Blowout fracture can occur from a variety of mechanisms and a combination of the hydraulic and buckling theory would appear to be the most compatible with the clinical findings in most surveys.

CLINICAL TESTS

Careful clinical assessment and accurate recording of data are of great importance in the diagnosis and management of blowout fracture. The aetiology of blowout points to the potential for legal compensation claims and this aspect also necessitates a comprehensive initial examination.

Ten of the twenty nine patients in this series became compensation claims. Following orthoptic/ophthalmic assessment of corrected near and distance visual acuity, media and fundus examination, the following clinical diagnostic tests should be done routinely —

DIAGNOSTIC TESTS

1. Observation — infra orbital nerve anaesthesia
   — retraction
   — enophthalmos
2. Cover test — near and distance measurements.
3. Binocular single vision assessment (if possible) — as it may shed light on pre existing problems.
4. Diplopia — A. Hess
   — B. Field of binocular single vision.
5. Ocular motility.
6. Visual field assessment.

X-RAYS, COMPUTER TOMOGRAPHY, MRI

These tests are all methods of producing visualisation of anatomical changes in blowout fracture.

1. X-rays
   These will provide information on facial and rim fractures and are often ordered initially. However they are not reliable in pure blowout. Occasionally antral or ethmoidal opacification gives a clue to blowout.

2. CT scans
   Most studies point to CT scans as providing the most informative diagnostic pictures.[10] A good coronal CT scan can show fracture site, extent of the fracture and soft tissue information. Although some clinicians feel it is unnecessary, there is an argument for routine CT scanning of all suspected blowouts as it removes the speculation (Figure 1).

3. MRI
   This is generally thought to be complimentary to CT scans rather than a superior test. It gives soft tissue information while CT scans give higher bone resolution.

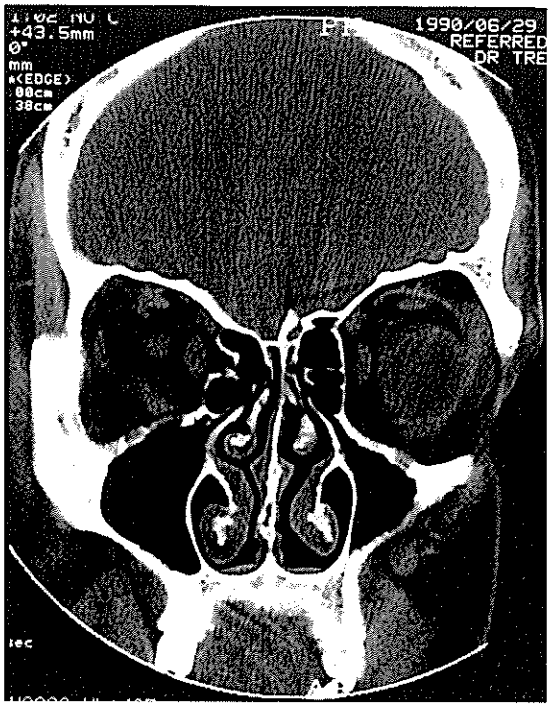

Figure 1. CAT scan of patient with left medial wall blowout. Fracture site and extra ocular muscles are clearly visible.

CLINICAL RESULTS

Twenty nine patients were examined the age range being seven years to fifty three years, with a mean age of twenty seven years. The results are analysed in the following Tables 1, 2 and 3.

TABLE 1
Distribution of Blowout Fracture

Unilateral	Bilateral	Right Eye	Left Eye	Male	Female
28	1	19	11	25	4

TABLE 2
Aetiology and Corresponding Type of Blowout Fracture

Aetiology		Pure Blowout	Impure Blowout
Motor vehicle accident	8	3	6
Assault	8	5	3
Football	5	3	2
Fall	5	4	1
Other	3	2	1

TABLE 3
Fracture Site and Incidence of Fractured Zygoma

Fracture Site		# Zygoma
Orbital floor	20	7
Floor and medial wall	6	4
Floor and lateral wall	3	2
Medial wall	1	

CLINICAL FINDINGS AND DISCUSSION

Classically the patient presents with a history of blunt trauma and may have local soft tissue signs such as periorbital haematoma, oedema, or subconjunctival haemorrhage. A subconjunctival haemorrhage with no posterior limit implies a fracture may be present as the blood may have tracked forward from the posterior fracture site.[11]

Patients with medial wall blowout fracture may present with subcutaneous emphysema or more seriously cerebro spinal fluid rhinorrhea. Trauma disrupts the meninges and cerebro spinal fluid leaks into the nose.

OPHTHALMIC SIGNS

Funduscopy revealed retinal damage in five of the twenty nine patients.

Retinal haemorrhage	1
Macula haemorrhage	1
Disc haemorrhage and chorioretinal tear	1
Retinal oedema	1
Macula oedema	1

Moderate to full traumatic dilatation of the pupil may occur with absent or diminished reactions to direct and consensual light reflexes. Five patients in this series showed a dilated pupil on initial examination.

VISUAL ACUITY

Traumatic impact on the globe may cause decreased visual acuity due to such complications as hyphema, lens dislocation, retinal tears and retinal or macula oedema.

In this series nine patients showed decreased acuity in the affected eye on initial examination although in five of these the acuity was reduced only to 6/9. The visual acuity loss was transient in seven of the cases. One patient had permanent visual acuity loss and one patient was lost to follow up.

DIPLOPIA

Diplopia was present on initial examination in all twenty nine patients. Diplopia is initially influenced by soft tissue swelling, and in some cases may be constant rather than confined to certain positions of gaze. It is typically vertical and may reverse when moving from elevation to depression. A horizontal component need not necessarily imply a medial or lateral wall blowout, as horizontal recti have connective tissue septa extending to the orbital floor (Koorneef).[12]

In this series the diplopia pattern was:

Purely vertical diplopia	22
Purely horizontal diplopia	1
Vertical and horizontal diplopia	6

It is important to remember diplopia may occur due to decompensation of a pre existing phoria or tropia, or traumatic damage to an ocular muscle or nerve (Table 4).

OCULAR MOTILITY

The concept of true muscle entrapment following blowout has been questioned. It is not consistent with diplopia patterns, with findings on high resolution CT scans or with surgical observations.

Koorneef,[12] in anatomical studies details a highly organised connective tissue septa around each eye muscle and between the eyeball and the

TABLE 4
Pre Existing and Acquired Palsies Associated with Blowout Fracture

Pre Existing Problems	No. of Cases	Acquired Palsies	No. of Cases
Congenital fourth nerve palsy	1	Seventh nerve palsy	2
Microtropia	2	Fourth nerve palsy	3
Intermittent divergent squint	1	Inferior rectus palsy	1

orbital walls. He feels it is disruption to this tissue septa and to the orbital tension that produces ocular motility imbalance. Some authors[13] state the connective tissue septa is so highly organised that it is possible to predict the fracture sight from the motility imbalance. In summary:

Reasons for Ocular Motility Imbalance:
1. Soft tissue oedema and haemorrhage.
2. Incarcerated tissue.
3. Herniation of orbital fat/connective tissue into the maxillary antrum. In rare cases it may be muscle.
4. Post traumatic oedema and scarring of tissue and fat causing disruption to orbital septa tension.
5. Traumatic muscle/nerve palsy.

In this series the ocular motility pattern was:

Restricted elevation	4
Restricted depression	4
Restricted adduction and abduction	1
Restricted elevation and depression	14
Restricted elevation, depression and horizontal movement	6

INFRA ORBITAL NERVE ANAESTHESIA

The infra orbital nerve is a branch of the Maxillary Division of the 5th cranial nerve. It runs forward from the inferior orbital fissure and travels in the floor of the orbit, first in a groove, then a canal and emerges on the face through the infra orbital foramen.[2] It is commonly affected in blowout directly from trauma to the floor, zygoma or maxillary region or from the surgery. Clinically it is important to test for anaesthesia in the areas it supplies.

The nerve supplies sensation to the skin and conjunctiva of the lower lid, the lateral aspect of the nose, the anterior cheek and upper lip. The anterior superior alveolar nerve descends from the infra orbital nerve into or along the maxillary bone and supplies sensation to the gums and anterior three teeth on the same side.[14] These may also be numb and indicate a fracture of the anterior plate of the maxillary bone or of the maxillary wall.

In this survey twenty patients had infra orbital nerve anaesthesia to some degree — one of these being a surgically induced trauma. Nine patients still had persistent anaesthesia on their final check.

ENOPHTHALMOS

Enophthalmos has been defined as a difference of greater than 2 mm in the position of the globes along the antero-posterior axis. Fells reported that a difference of greater than 3mm constitutes a cosmetic problem.[15] The cause is a discrepancy between the volume of the bony cavity and the volume of its contents.[16]

Reasons for Enophthalmos:
1. Enlargement of the bony cavity i.e. increased bony cavity volume.
2. Displacement of soft tissue out of the orbit — usually herniation of contents into the maxillary sinus i.e. decreased content volume.
3. Tethering of the globe posteriorly by trapped orbital fat or tissue.
4. Fat necrosis and decreased soft tissue volume.

Bite[16] performed three dimensional CT scans to measure cavity and soft tissue volumes in patients with established enophthalmos. Their results suggest that in the majority of cases post traumatic enophthalmos is caused by increased bony cavity volume rather than soft tissue loss. In the cases with no volume discrepancies, tethering and contracture of retrobulbar tissue were thought to be responsible. Only two patients in this series developed marked enophthalmos.

RETRACTION

Retraction of the globe can occur when the eye moves in a direction opposite to the trapped tissue or fibrosis. Absence of retraction provides a better prognosis for spontaneous recovery of ocular movements in pure blowout.[11] Blowout fracture of the medial wall may have retraction on abduction or more rarely adduction. This is sometimes termed 'Acquired Retraction Syndrome'. This occurred in one patient in this series (Figure 2).

VISUAL FIELD LOSS

In this series twenty seven patients were tested

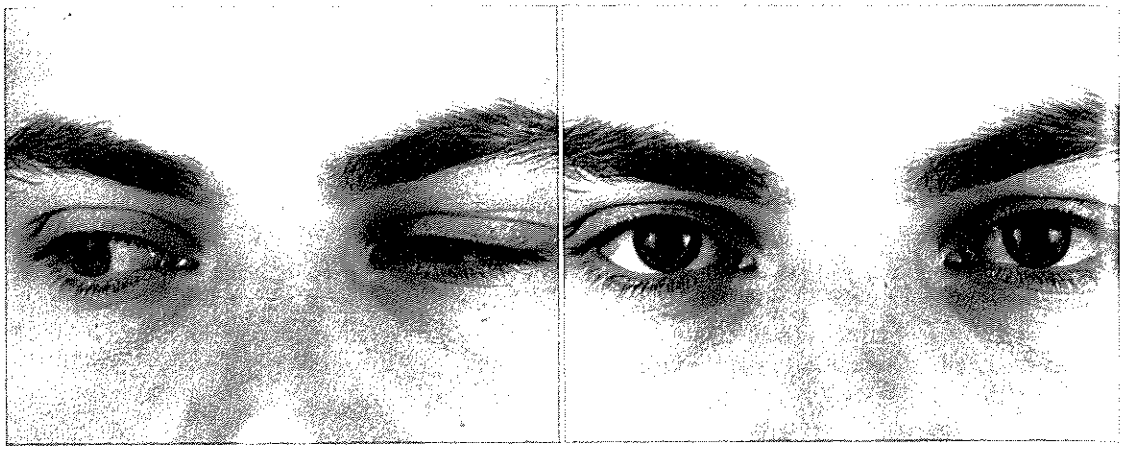

Figure 2. Patient with Left Acquired Retraction Syndrome in primary position and dextro gaze.

on the Goldmann perimeter. Ten patients showed varying degrees of visual field disturbance that related to optic nerve trauma. It was permanent in nine of them.

Optic nerve damage may occur due to:

1. Direct damage to the nerve from traumatic force.
2. Shearing injuries.
3. Compression of the nerve at the apex.
   A. Haemorrhage
   B. Bone fragments
   C. Increased orbital content volume e.g. post bone grafting.

Of the ten patients with field disturbance six were tested pre-operatively and the remaining four post operatively. This occurred as two were late referrals and two were tested early in the series, before the importance of routine testing became obvious.

It is not possible to pinpoint the exact aetiology in each case. However, four patients with known orbital apex trauma all sustained marked defects extending from the disc to the periphery. One patient with a normal field pre-operatively sustained optic nerve trauma in the post operative phase.

MANAGEMENT

In discussing management of blowout fractures, both pure and impure must be discussed as separate clinical entities. The management approach to each is quite different, although good ocular motility without enophthalmos is a common goal.

IMPURE BLOWOUT

Impure Blowout is essentially a surgical problem and is managed by ocular plastic surgeons. The rim is repaired, any accompanying depressed zygoma fracture is elevated and repaired and the orbital floor is explored and repaired at the same time.

Some aspects of the surgical management:

1. Lower Lid Incision — this is the common approach.
   (a) Lash margin
   (b) Orbital rim margin
   (c) Lid fold margin (between a & b)
2. Caldwell — Luc Approach — via the mouth above the canine and second molar teeth, to the maxillary antrum. This is a difficult approach and may cause damage to the orbital contents, alveolar nerve or in children the undescended molars.
3. Medial Canthal Incision — is commonly used to explore a medial wall. Sometimes a bicoronal flap is used to provide wide exposure of a medial wall fracture.

IMPLANTS USED

1. Silastic sheeting for smaller fractures.
2. Autogenous bone grafting for larger fractures.

BONE GRAFTING SITES

1. Calvarial bone grafts are usually from the parietal bone. The advantage of these grafts is that they are vascularised and do not resorb. The disadvantage is that they do not bend and are not easily contoured.
2. Split rib grafts are selected because the site is easily accessible, and has the ability to regenerate. Another advantage is that they are easily contoured and thus ideal for reconstructing curved surfaces such as the orbital floor.
3. Iliac crest or hip grafts are again easily accessible. Some consider they resorb more readily than calvarial grafts.

The twelve impure blowouts in this series all had surgery by plastic surgeons. Nine had implants and three rim repair only.

PURE BLOWOUTS

This was once considered an urgent surgical problem. However, recent studies on the anatomical structures involved and better understanding of the problem have led to a much more conservative approach. Reports by Putterman[17] and others suggest many pure blowouts recover satisfactorily without surgical intervention. The most sensible criteria for intervention advocated by two British surveys are:[18,19]

1. Diplopia not resolving within 14 days.
2. Fractures with large herniation.
3. Incarceration with global retraction.
4. Enophthalmos. 3 mm or more.

TIMING

The optimal time for surgery is within the first fourteen days and if left longer than two months the prognosis is poor.

In this series there were seventeen pure blowouts and eleven of these required surgery. Eight patients had surgery within the first week, one patient within two weeks and two patients within nine weeks.

While these findings indicate that a high percentage of patients required surgery, after studying the data, four of those operated on within the first week had serious blowout fracture which required further surgery.

MANAGEMENT RESULTS

IMPURE BLOWOUT	12 cases
Complete Recovery	3 cases
Good Recovery	3 cases (minimal residual problems)
Symptoms	3 cases
Failed to attend	3 cases

PURE BLOWOUT	17 cases
Complete Recovery	4 cases
Good Recovery	5 cases
Symptoms	6 cases
Failed to Attend	2 cases

BLOWOUT FRACTURE IN CHILDHOOD

Blowout fractures in childhood, as with facial fractures are relatively uncommon.[20] One reason for this may be that the facial bones in children are softer and more elastic than in adults. A second reason often given is that blowout fracture cannot occur until the sinuses have developed. Wolff[2] states that the sinuses are very

TABLE 5
Results of Children with Blowout Fracture

Age	Injury	Fracture Site	Treatment	Results
F 8	MVA	Medial floor	Silastic implant ten days later	Post op poor depression then lost to follow-up
M 9	Elbow and fall	Floor — apex	Two explorations. One (with implant) after one day and one six weeks later	1. Gross U/A elevation 2. U/A depression 3. Inferior field loss
M 9	Fall	Medial floor	Exploration after one day and tissue freed	1. Gross U/A elevation 2. Retraction

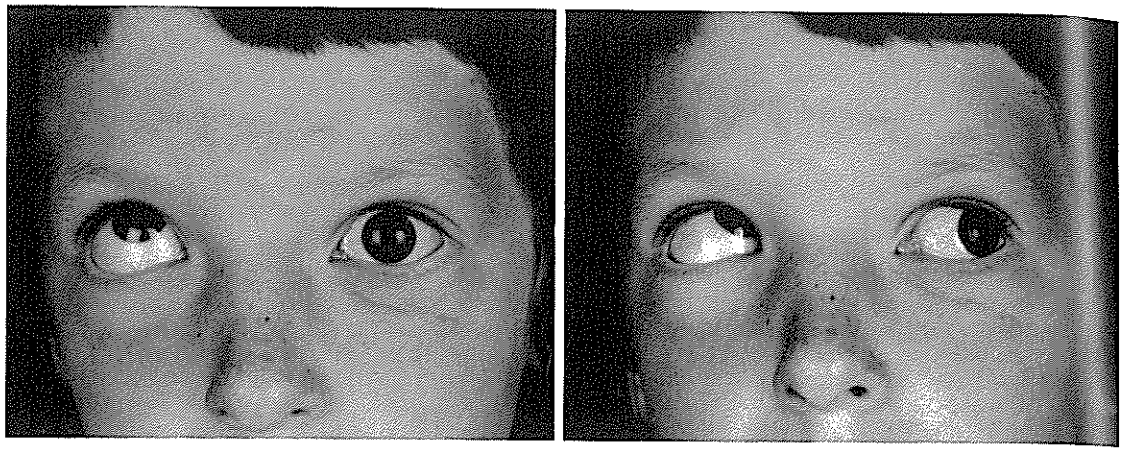

Figure 3. Restriction of left elevation in child following blowout fracture repair.

small in the young and start to increase in size around seven to eight years of age. However, children as young as three and certainly around seven to eight years can show clearly defined maxillary sinuses on CT scans.

When blowout does occur in the young child, the prognosis is often very poor. Studies by McCarry[21] and others have concluded that younger patients have a poorer prognosis particularly where there is limitation of ocular movement and retraction. Waddel[19] and others have proposed that scar tissue may develop more quickly in the young child.

In this series three children had blowout fracture. Of the two children who had a long term follow up the results were disappointing, showing marked ocular motility disturbances (Table 5) (Figure 3).

CONCLUSIONS

Experiments have shown that blowout fracture may be caused by a force to the globe, to the rim, or both. The data in this paper agrees with Kerstens[6] conclusion that isolated hydraulic pressure alone is unlikely to be the cause of most pure blowouts.

There was a high incidence of visual field damage especially with apex fractures. It can occur as a result of surgery, and it is important to test visual fields on initial examination if possible.

Children in this survey responded poorly to the treatment of their blowout fractures. This occurred in spite of early surgical intervention and the reasons for this are not as yet fully understood.

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TRANSIENT SUPERIOR OBLIQUE SYNDROME IN SCLERODERMA A CASE STUDY

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Abstract

A 27-year-old Eurasian woman with a 13 year history of scleroderma (progressive systemic sclerosis) developed typical features of Superior oblique syndrome (Brown's syndrome), which spontaneously resolved after 12 months, leaving a palpable nodule in the left superior oblique tendon. The aetiology and associations of this disorder are briefly reviewed.

Key words: *superior oblique, Brown's syndrome, scleroderma.*

CASE REPORT

The patient, a 27-year-old Eurasian woman, had presented at age 14 with Raynaud's phenomenon and tight skin of the fingers and face. A clinical diagnosis of scleroderma (diffuse, Type III)[1] was supported by skin biopsy.

Scleroderma is a connective tissue disorder of unknown aetiology characterised by vascular insufficiency, particularly in the form of Raynaud's phenomenon, bilateral skin stiffness, various systemic disturbances associated with over-production of collagen, and features of disturbed immunity. It has been subdivided into 'types' according to the extent of the skin involvement. Although uncommon it is not excessively rare and has an estimated prevalence in Australia of approximately 10 cases per 100,000 of population.

Subsequently dysphagia, respiratory restriction and limitation of large joints developed. Her general management included oral Prednisolone, intermittent D-Penicillamine, oesophageal dilatation and Cimetidine for gastro-oesophageal reflux.

In April, 1989, at age 26, she noticed increasingly frequent vertical doubling of images on attempted upgaze, which was intermittently relieved in association with a palpable 'click' in the upper medial corner of her left orbit, and some discomfort in that region. Examination in April, 1990 revealed restriction of elevation of the left eye in adduction, with local tenderness in the region of the left trochlea. Ocular movements and Hess charts were entirely consistent with Brown's syndrome, in relation to acquired isolated inferior oblique paralysis. (Figs. 1, 2).

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Figure 1: Facial features and restriction of left eye on dextroelevation.

No click was detected on examination. Rose Bengal staining and Schirmer test demonstrated a mild lacrimal deficiency. Contraction and stiffening of her eyelids and lips were also consistent with the primary diagnosis of scleroderma.[2]

Computerised topography of the orbits and brain was performed, and showed no abnormality.

Twelve months after the onset of her ophthalmic symptoms, the patient was woken at

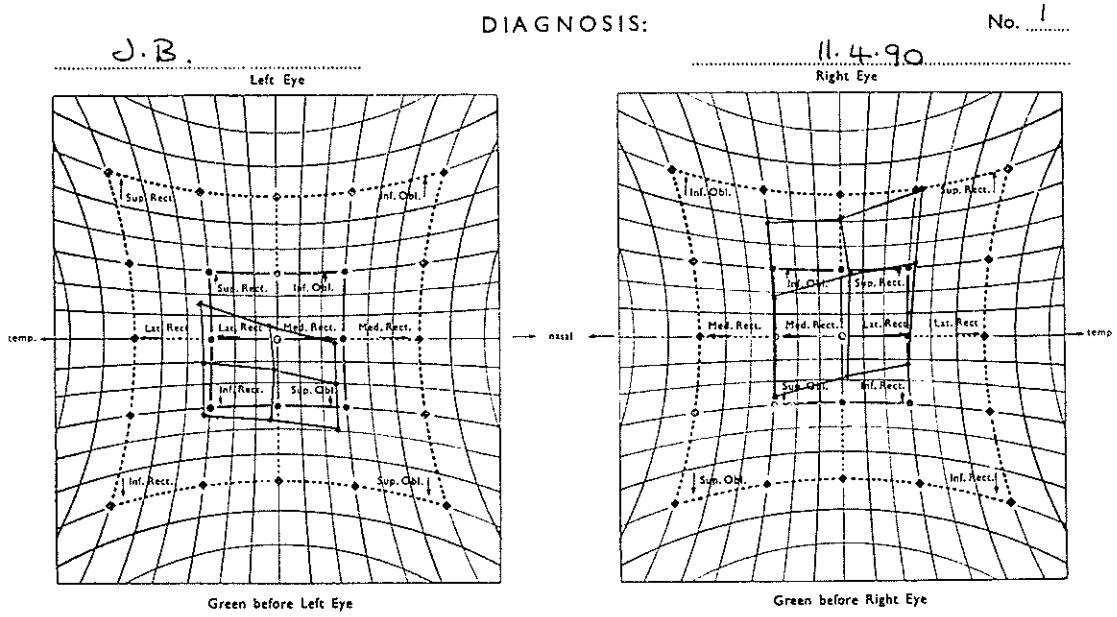

Figure 2: Hess chart 10 months after onset.

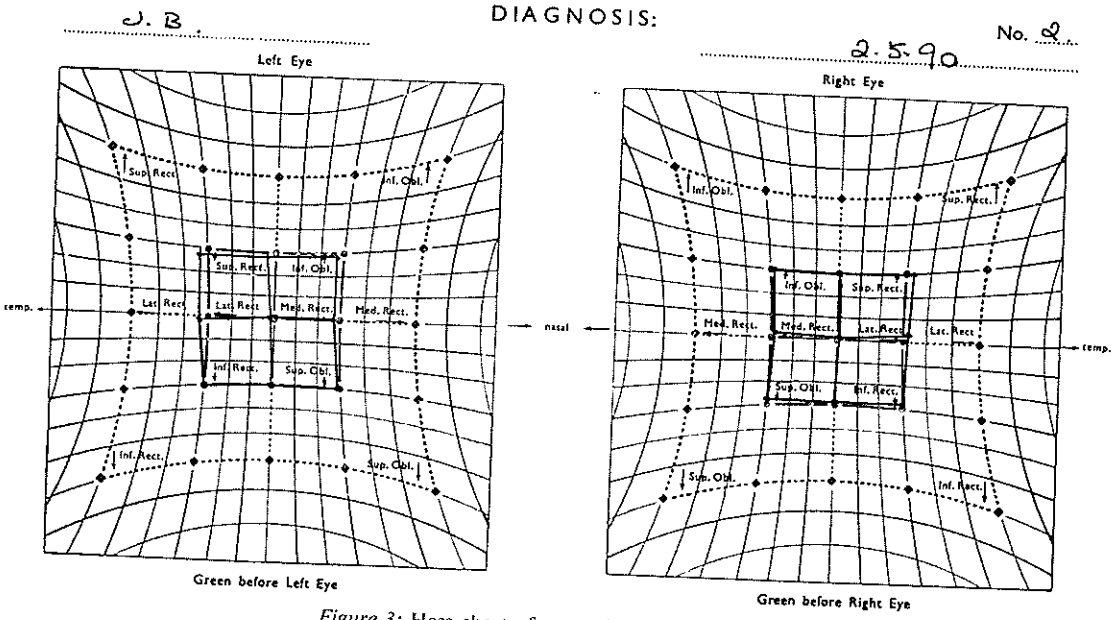

Figure 3: Hess chart after resolution of diplopia.

night by a pain above her left eye, and found that the diplopia had resolved, which has not re-occurred. Subsequent examination showed normal ocular muscle balance and motility (Fig. 3). A mobile nodule could be palpated in the antero-medial part of the superior oblique tendon.

DISCUSSION

In an early description of the syndrome bearing his name, H.W. Brown[3] proposed shortening of the anterior tendon sheath of the superior oblique muscle as the cause of this musculo-fascial disorder seen most often in children, and often resolving spontaneously. However, the most effective surgical treatment was found not to be excision of the tendon sheath, but tenotomy or tenectomy of the superior oblique tendon,[4] a finding consistent with other theories of pathogenesis such as reduced elasticity or shortening of the tendon[5], or a nodular thickening of the tendon obstructing its free movement through the trochlea.

The most common systemic association of this condition is with rheumatoid arthritis,[7] implying an increased frictional resistance to tendon movement. Local treatment by cortico-steroid injections has been helpful to a varying degree in such cases.[8,9,10] Local inflammatory[11] and neoplastic disease,[12] and surgical trauma[13] have been implicated rarely. A bilateral case developing in pregnancy has been reported.[14] Current understanding of the causes and mechanism has been comprehensively reviewed by Wilson et al.[15]

The late onset of this patient's disorder is unusual,[6] and supports the assumption that her systemic illness was a causative factor. It is suggested by Weilby that, in analogy with trigger finger,[17] the nodular thickening of the tendon may be secondary to frictional resistance to movement through the trochlea, with over-riding and bunching of concentric layers of collagen within the tendon. Spontaneous resolution could thus occur through the restoration of the normal arrangement of the tendon fibres, perhaps accompanied by some reduction of swelling within the tendon. This suggestion is consistent with the common experience of release with a 'click',[18] and in the case described here, by the residual palpable thickening of the tendon.

The specific relationship of Brown's syndrome with scleroderma or other rheumatic disorders

is not clear. The superior oblique tendon does not have a true synovial sheath, so that the syndrome is not a teno-synovitis. Localised infiltration and/or oedema is seen in other tendons and ligaments in rheumatoid arthritis, more rarely in scleroderma; involvement of the superior oblique tendon may be rare simply because of its smallness in proportion to the bulk of other tendinous structures. If frictional damage is a factor in nodule formation, the immobilisation of the tendon when totally trapped within the trochlea will tend to encourage spontaneous recovery, which is frequently observed.

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ACQUIRED INTERMITTENT SUPERIOR OBLIQUE TENDON SHEATH SYNDROME (SOTSS) — 3 case reports

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Abstract

Three cases of acquired, intermittent, unilateral Superior Oblique Tendon Sheath Syndrome (SOTSS) or Brown's Syndrome are presented. They presented with differing symptoms and on clinical assessment of ocular movements demonstrated a marked restriction, and at times, a complete absence of elevation in adduction of the affected eye – depicting a marked inferior oblique underaction. This abnormality of movement possibly as a result of prevention of muscle movement through the trochlea was found to be due to a localized inflammation, possibly of the orbit in one case, a systemic disease in another, and of unknown cause in the third. The intermittent nature and 'click' phenomenon will be described and discussed.

Key words: *Acquired SOTSS, 'click' phenomenon.*

INTRODUCTION

Superior Oblique Tendon Sheath Syndrome (SOTSS) or Brown's Syndrome is a condition characterised by a restriction or absence of elevation in adduction, in an affected eye[1] It is most commonly seen as a congenital condition but as demonstrated by the following cases can be acquired. When acquired the condition can be associated with an intermittent nature and the 'click' phenomenon. It can be acquired at any age[1,2] but usually seen in childhood[3,4] and in young adults[3,6]. The onset of this condition may be sudden or gradual and may eventually disappear completely with or without any treatment[1,6].

Inflammation and trauma appear to be the most common aetiological factors responsible for many of the acquired intermittent SOTSS. Some of the inflammatory conditions include — generalised systemic inflammation such as juvenile or adult rheumatoid arthritis[2,5,7,8,9,10] and Systemic Lupus Erythematosis (SLE)[12]; localised inflammation of the superior oblique muscle or tendon as a result of a stenosing tenosynovitis (ST)[2,5,6,8,10,11,12,14]; sinusitis[7,9]; pregnancy[16]; orbital tumors[8]; and inflammation of unknown cause[11,13]. Trauma to the area of the trochlea[9,11]; damage to the orbital walls[8]; surgery and/or trauma to the superior oblique muscle, as well as anomalies of the tendon sheath of the superior oblique[3,7,9], are other common causes reported for the acquired types of SOTSS.

This interference to normal superior oblique muscle movement through the trochlea is believed to be as a result of some intermittent mechanical restriction or defect of the superior oblique muscles' tendon or its sheath, most commonly involving the area just behind the trochlea, which prevents the free passage of the muscle and therefore no elevation in adduction.

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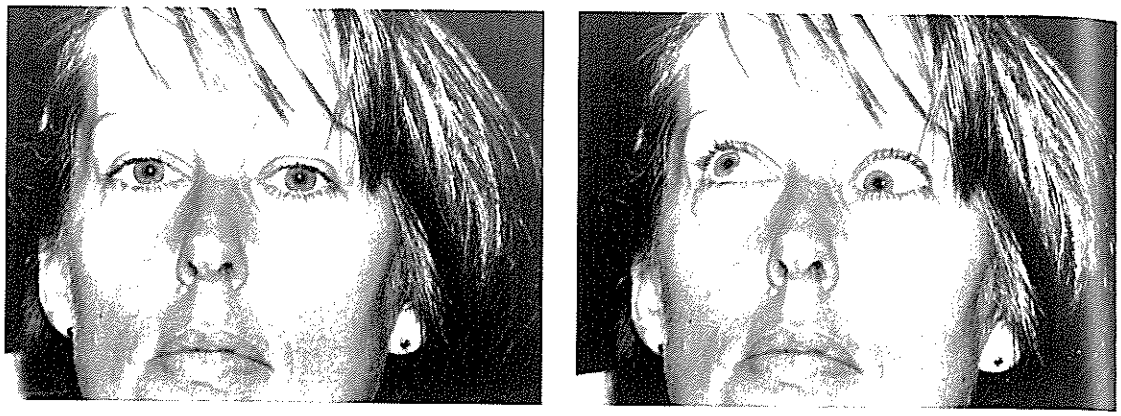

Figure 1: Case 1 on presentation in primary position and dextroelevation note marked underaction of left eye in dextroelevation

This mechanical restriction may be in the form of thickening, swelling, nodules or adhesions of the superior oblique muscle, tendon sheath, or some anomaly of the trochlea or its surrounding region[8,9,18].

Stenosing Tenosynovitis (ST) of the hand, a condition usually seen in children or young adults, has been found to show similar mechanisms to that of intermittent acquired SOTSS[11]. The anomaly appears to lie in the tendon and sheath of the hand and superior oblique muscle, respectively.

Case 1

A thirty-one year old lady presented with an inability to move her left eye up and inwards for two days. She stated that this occurred intermittently over the previous two years beginning with difficulties focussing then an inability to move the eye, this usually lasting for approximately half a day. She also stated that over the years she felt a 'click' just before full ocular muscle movements of her eye was possible. Pain and diplopia had never been experienced. Her general health was good, she wore glasses for a small hypermetropic astigmatic refractive error, and there was no other relevant ocular family history except that both her children had convergent squints which required surgery.

Ocular examination showed the following:-

Visual Acuity — corrected right and left 6/6 N5

Cover Test — near and distance a small right hypertropia 5▲ increasing on right gaze

Abnormal head posture — slight tilt to left

Ocular Movements — marked left SOTSS, left superior rectus underaction, slight tenderness

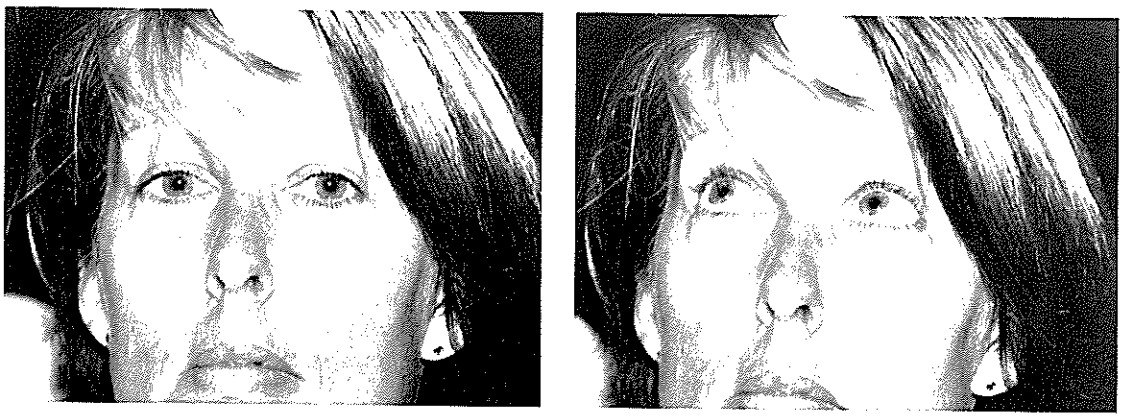

Figure 2: Case 1 post steroid treatment, six days later. Full muscle movement of left eye in dextroelevation

over the left trochlea and injected upper nasal fornix. See figure 1.

A CT scan and full blood examination were ordered and were normal. Oral steroids (prednisolone 5mg) were prescribed to relieve the discomfort. At review in six days, she was much happier and ocular movements were full. See figure 2. Medication was ceased and when reviewed four months later she was symptom-free and her ocular examination was normal.

Case 2

A forty year old lady presented complaining of intermittent vertical diplopia and an inability to look up and to the right. She stated that at times she would forcefully move the eye into this position whereby it was accompanied by a 'click' followed by a discomforting 'tight' sensation of the left eye. She had noticed this intermittently for approximately three months. She was on medication for hypertension and suffered from sinus problems. She had presented to the clinic six months earlier after suffering food poisoning with the complaint of difficulty when focussing. At this time, all tests including ocular movements were unremarkable.

Ocular examination on this visit showed the following:-

Visual Acuity — right and left 6/6, N5 uncorrected

Cover Test — near and distance — orthophoric in primary position.

On right gaze — right hypertropia

Convergence — to 6 cms

Stereopsis (TNO) — 60 sec'

Ocular Movements — marked left SOTSS with no other over or under actions and some discomfort around the left eye.

A CT scan revealed no abnormality of the orbit or sinuses. She was given a course of oral steroids (prednisolone). One month later she still had a marked left SOTSS which was evidently worse in the morning and again could be overcome by forceably moving the eye into the affected gaze whereby she felt a 'click' and was able to demonstrate a full range of movement. This could be demonstrated several times during the consultation. At this stage the diagnosis of left superior oblique tendonitis was made and she was given an injection of Decadron to the trochlea.

One month later she stated that the injection had helped for several days, but the condition had since reoccurred with the muscle abnormality now being constant, vertical diplopia worse, and the eye was no longer 'clicking'. She complained of muscle aches and joint pain and was sent for a full blood examination. Rheumatoid factor was negative. Antinuclear antibodies were present in a titre of 1:40 which is indicative of an underlying systemic disorder, particularly that of Systemic Lupus Erythematosis (SLE).

At the next visit, ocular examination showed a constant marked left SOTSS which could eventually be forced into full movement by extreme effort and slight pressure to the area of the left trochlea. See figure 3 & 4.

This patient is still being investigated by her GP and on last examination was given an injection of Depomedrol (longer lasting steroid) to the trochlea.

Case 3

A five year old boy presented because his parents were concerned that their son's eyes "didn't seem to be co-ordinated" and that his vision seemed to have deteriorated since hitting his head during a fall two weeks previously. There was no bruising or direct injury to either eye. He was a healthy boy with no relevant ocular family history and whose kindergarten eye test had been normal the year before.

Ocular examination revealed the following:-

Visual Acuity — right and left 6/6 (Sheridan Gardner)

Cover Test — near and distance — orthophoric in primary position.

On left gaze — left hypertropia

Convergence — to 6 cms

Stereopsis (TNO) — 60 sec'

Ocular Movements — marked right SOTSS with no other over or under actions. On attempting to move the eyes into laevoelevation the boy complained of pain and was very reluctant to move the eye into this position. Five minutes later, on repeated testing of ocular movements, a full range of movement was elicited.

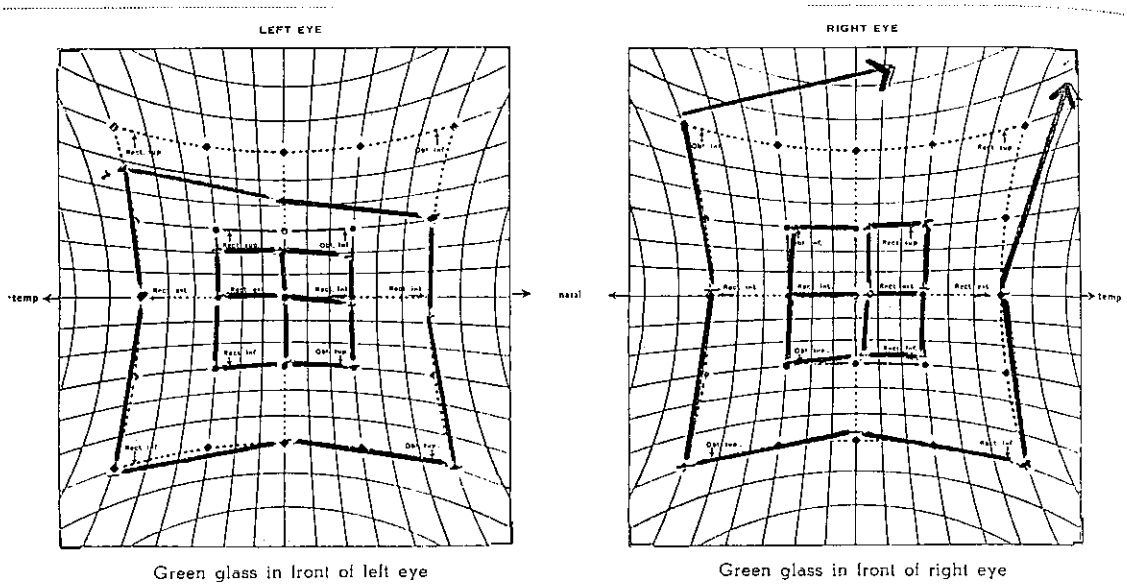

Figure 3: Case 2 Hess chart showing muscle under and overactions before 'click'

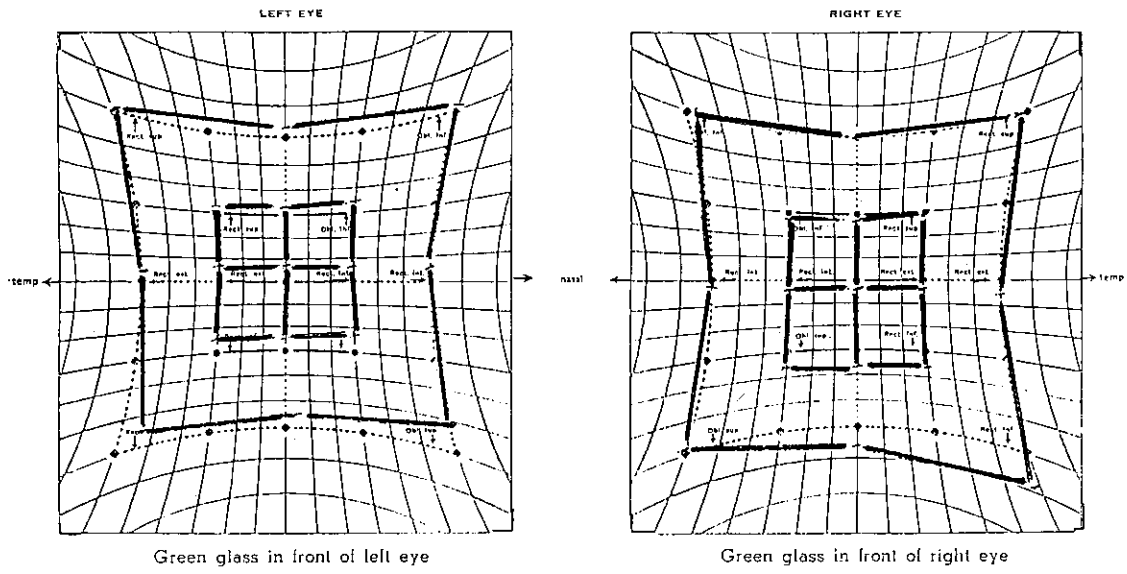

Figure 4: Case 2 Hess chart showing full muscle movements after 'click'

There was no sign of proptosis or obvious pain over the right trochlea and pressure to the area of the trochlea did not produce a 'click' or full ocular movements. However, some twenty minutes later a marked right SOTSS was again observed which recovered to full movements within moments.

Six weeks later, the ocular examination showed a constant right SOTSS. Repeated attempts to move the eye into laevoelevation or applying pressure to area of right trochlea did not achieve full movement. Since this last visit the parents have noticed the restriction of movement only intermittently. On review three months later similar intermittent ocular movements were seen.

DISCUSSION

On ocular examination, all three cases showed a marked intermittent SOTSS. The hypothesis that this condition mainly affects young adults and children is consistent with the three cases presented in this paper.

All three cases experienced different presenting symptoms. This is important because it shows that a variable range of symptoms exist for this condition. Cases 1 and 2, experienced difficulties with focussing prior to the onset of the muscle movement abnormalities. It was not possible to accurately determine whether focussing was a problem on questioning the young boy, however, the fact that the parents felt he was not seeing well could indicate a focussing problem. Blurring or 'haziness' of vision have been noted by others[9,17].

The onset of the presenting symptoms could not be attributed to any previous injury or illness in cases 1 and 2. However, a fall experienced by the young boy, two weeks prior to presentation may have been a predisposing factor in producing the right SOTSS.

Pain and discomfort was experienced by all three cases, this being in the region of the upper, nasal canthal area and experienced when attempting to move the affected eye into the affected gaze. This was overcome by avoiding the affected gaze in all cases. It was overcome completely in case 1, by a short course of oral steroids, and temporarily overcome by injections of decadron and depomedrol into the trochlea in case 2.

The muscle restriction observed in each case had an intermittent nature, in that, the restriction changed from moment to moment and visit to visit. With the change in muscle restriction, from marked restriction to full muscle movement it was possible to observe a sudden release of the muscle along with an audible 'click' which was also felt by the patients. The muscle restriction in each case could be overcome either by extreme effort, repeated attempts, or by applying pressure to the area of the trochlea with an index finger.

Interestingly, cases 1 and 2 complained that the muscle restrictions and 'clicking' were worse in the morning and becoming more frequent. This could indicate that the restriction is as a result of some unknown causal entity occurring when the eye is not being used, but improves as the day progresses and with repeated eye movement.

It appears that the 'click' is always experienced and felt in the area of the trochlea. The 'click' most likely indicates a sudden release of the muscle as it passes through the trochlea overriding any obstruction and therefore resulting in full muscle movement. The 'click' phenomenon is regarded to be a stage in the resolution of the condition[9]. The 'wearing down' of swelling or enlargement of trochlea with growth is believed to be a cause of this intermittent condition[3,9].

CONCLUSION

The muscle abnormalities, intermittent nature, 'click' phenomenon and symptoms presented in these three cases show some of the common signs associated with acquired, intermittent SOTSS as reported by others.

As seen the symptoms and possible causes are numerous, therefore it is most important to attempt to determine the cause of the condition before any form of treatment is considered. A thorough systemic examination should be carried out.

Ocular movements should be tested routinely on all patients, particularly adults, as this condi-

tion may be a common phenomenon which is being missed and may be an important sign leading to the diagnosis of some underlying systemic disease.

ACKNOWLEDGEMENT

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BOTULINUM TOXIN FOR THE TREATMENT OF BLEPHAROSPASM AND HEMIFACIAL SPASM

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Abstract

Forty eight patients, thirty two with blepharospasm and sixteen with hemifacial spasm were treated with botulinum toxin A. Patients considered the treatment to be successful despite the temporary effect and transient side effects that occurred with 72% of treatments given to hemifacial spasm patients and with 55% of treatments for blepharospasm patients. The duration of effect was longer for hemifacial spasm patients than blepharospasm patients (13.4 weeks compared with 7.6 weeks $p<0.01$). The patients did not become tolerant to the drug after repeated treatments.

Key words: *Botulinum toxin A, blepharospasm, hemifacial spasm.*

INTRODUCTION

Botulinum toxin A is derived from one of the several antigenically distinct neurotoxins produced by Clostridium botulinum. Clostridium is a spore forming, motile bacteria that is found widespread in soil and may cause disease in man and other animals.[1] It is often the cause of food poisoning (botulism) when canned meat or vegetables are contaminated with the spores and not sufficiently heat-treated prior to sealing.

Botulinum toxin A was first crystallised in 1946 in an army laboratory[2,3] where the purpose of the experiments was to describe the chemical nature of botulinum toxin. This was notable for two main reasons; firstly, this was the first crystallisation of a bacterial exotoxin and secondly, this enabled the toxin to be used for clinical investigations. Subsequently, various people investigated the effects of botulinum on selective blockage of synaptic transmission in animal subjects.[4,5]

Type A botulinum is used for therapeutic treatment because of its extreme potency and because it can easily be transformed into a crystallised form.[6] It is thought to inhibit the release of acetylcholine at the neuromuscular junction[7-11](see fig. 1).

For therapeutic use, botulinum toxin A is used in extremely small quantities and the dosage is determined in units. One unit is the amount of toxin found to kill 50% of 18-20 Swiss Webster mice and this is termed the 'lethal dose' (LD50). This is approximately 0.4 ng. The LD50 for a human is approximately 2000 ng. Since the average dose per injection for a blepharospasm patient is 8 ng (20 units), this is approximately 1/250th of the LD50 for humans.[12]

The clinical disease of botulism is characterised by blurred vision, diplopia or photophobia followed by dysphagia, dysphonia, nausea and vomiting. Clinical signs include respiratory impairment, specific muscle weakness as well as

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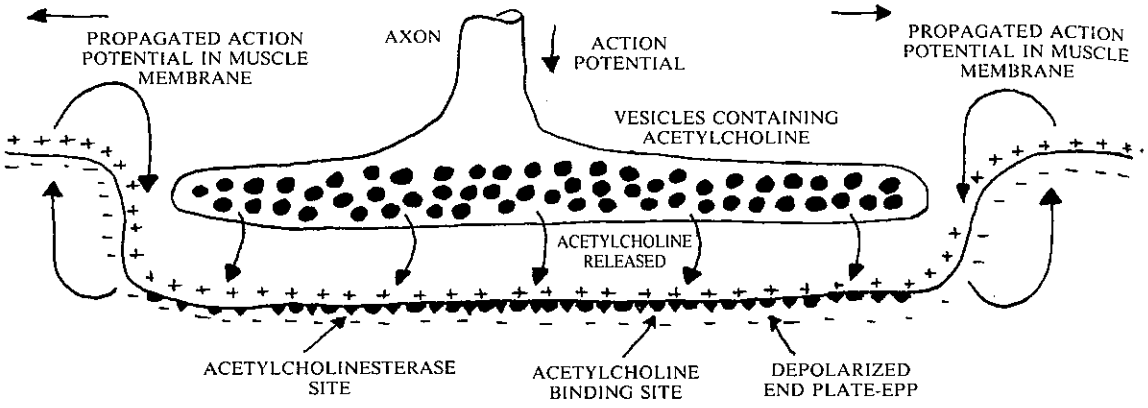

Figure 1: Neuromuscular junction showing the events that occur which lead to an action potential in the muscle.

a dry throat, mouth or tongue and sometimes dilated fixed pupils.[6]

The first investigation of botulinum for the treatment of ocular complaints was by Scott in 1973. He found there were significant alterations in ocular alignment after injection of botulinum into the horizontal recti of rhesus monkeys, the results varying with the concentration. From this, he proposed that botulinum could be used as an alternative to strabismus surgery or for other disorders such as blepharospasm and lid retraction in endocrine exophthalmos.[13]

For the treatment of strabismus, particularly in paralytic types, botulinum can be injected into the overacting muscles in order to change the position of the globe within the orbit. It is reasonable to use botulinum for up to two years following paralysis to prevent or reverse contracture.[14]

BLEPHAROSPASM

Blepharospasm is characterised by involuntary, spasmodic contractions of the orbicularis oculi. In most cases there is no known cause but it may occur with dysfunction of the basal ganglia or rostral midbrain. It may also occur in extrapyramidal disorders such as Parkinson's disease, Huntington's disease, progressive supranuclear palsy and can be drug induced in conditions such as tardive dyskinesis.[15,16]

Blepharospasm has been treated with bilateral selective facial nerve avulsion which often produces only temporary benefit. Orbicularis stripping procedures have been described and these are said to result in fewer complications.[15]

Centrally acting drugs have also been used in an attempt to ameliorate the severity of the spasm.[16-18] These have been reported to provide relief in less than 30% of cases[19] and mechanical aids such as ptosis props have also been used with little success.[15]

HEMIFACIAL SPASM

Hemifacial spasm is characterised by unilateral contractions of the muscles innervated by the facial nerve. There is usually no known cause. Rare causes include aneurysms or tumours of the cerebellar-pontine angle.[17] There is also a theory that compression of the facial nerve due to a vascular anomaly can be a cause of hemifacial spasm[17,20,21] and neurosurgical exploration often results in separation of the nerve from an artery. This is a major surgical procedure and is not always successful and hearing loss can be a complication.[22] Other treatments have included selective facial nerve neurectomy. However, aberrant regeneration and recurrence of the spasm often results.[23] Centrally acting drugs have also been used with limited success.[17]

It was not until 1984 that botulinum toxin A was used in the treatment of facial spasm when Frueh et al[24] found that a group of patients noted significant relief in the eyelid spasm in the 2 or 3 days following treatment.

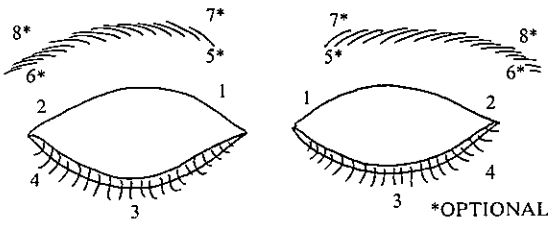

Figure 2: Standard sites of injection for blepharospasm patients.

Following local injection of botulinum toxin for strabismus or facial spasm there is thought to be noticeable improvement within 48 hours[26] and maximum effect within 10-12 days.[26]

There have been a number of local side effects of botulinum reported to date. These are transient and usually well tolerated. These include ptosis, epiphora, dry or irritated eyes, exposure keratitis, ectropion, diplopia, local bruising, lower facial weakness and others.

PATIENTS, MATERIALS AND METHOD

The effect of botulinum toxin A was assessed on a series of forty eight outpatients with blepharospasm or hemifacial spasm and a total of 122 treatments were given.

Initial consultation involved establishing or reaffirming a diagnosis of blepharospasm or hemifacial spasm and discussing the suitability of treatment with botulinum toxin.

Prior to treatment, the patients were made aware of the possibility of side effects and appropriate forms were completed according to protocol supplied by Scott.

The patients were asked to rate on a scale of 0–4 how debilitating their condition was (see below).

0–No inconvenience
1–Minor inconvenience
2–Some interference with lifestyle eg. reading, driving, social activities
3–Significant interference with lifestyle eg. significantly affects reading, driving
4–Severely affects lifestyle eg. unable to drive, unable to read.

Prior to injection, the botulinum toxin was diluted with unpreserved saline to the following concentrations of 100 units/mL, 50 units/mL and 25 units/mL. The different concentrations were used depending on the total dose required. If a patient required a large dose, usually a stronger concentration was used to minimise the volume of solution injected. Injections were given using a 1mL syringe and a 30 gauge needle. For blepharospasm patients, a minimum dose was given on the first visit usually in standard sites around both eyes (see Figure. 2). In most cases this was 10 units per eye, with 2.5 units being injected at each site.

For hemifacial spasm patients, injections were given in standard sites around the eye (see Figure 3) and sometimes additional sites in the region of the upper cheek were given to help reduce lower facial spasm.

One week following the initial treatment, most patients were seen for review. Following subsequent treatments, patients were instructed to phone one week following each treatment. Due to the distance some patients had to travel, they were contacted by phone at one week following all treatments.

At one week following treatment, patients were asked if there was any subjective improvement in the eyelid spasm and were asked to rate this as a percentage improvement. Furthermore, they were asked to rate the debility from the scale (0-4). Patients were then contacted on average every 4 weeks or when they presented for further treatment to review their progress.

RESULTS

Forty eight patients were treated with botulinum toxin A. A summary of the patients can be seen in Table 1.

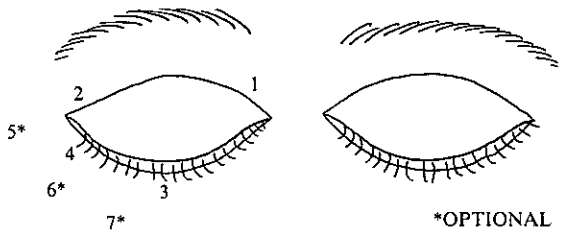

Figure 3: Standard sites of injection for hemifacial spasm patients.

TABLE 1
Number of patients for each group in the trial

Blepharospasm	32	3 Meige's syndrome
Hemifacial spasm	16	2 Parkinson's disease

A total of 122 treatments were given. Of these, 90 were given to blepharospasm patients and 32 were given to hemifacial spasm patients.

There was no significant difference in the age of onset for blepharospasm patients and hemifacial spasm patients using the two sample t-test. (57.6 years compared with 59 years. $T=0.4$ $p>0.05$).

Nine patients had undergone surgery prior to entering into the trial. One patient with hemifacial spasm had two operations (see Table 2).

Fourteen patients were also taking medications related to their ocular condition at the time of entering the study. These drugs included antiparkinson agents, antipsychotic agents, muscle relaxants, antianxiety agents, dopamine depletors, anticonvulsants, sedatives and tricyclic antidepressants. Treatment with these drugs had limited success.

Of the patients who had only one injection, 94% of patients reported a noticeable improvement in the eyelid spasm (>40% subjective improvement in the eyelid spasm). Of those patients that had more than one injection, 4% of patients failed to respond on two or more consecutive treatments (less than 40% improvement). In 122 treatments, 12.5% of injections produced an improvement of less than 40%.

Two patients with blepharospasm dropped out of the study, both of whom had 1 treatment only given to 1 eye. Both chose to discontinue with treatment because of lack of efficacy and one suffered with diarrhoea for 12 hours following treatment and developed a rash over her hands.

TABLE 2
Summary of the surgical procedures undergone by 9 of the patients

	Blepharospasm	Hemifacial Spasm
Blepharoplasty	3	2
Orbicularis stripping	—	1
Facial nerve decompression	—	2
Facial neurectomy	—	2

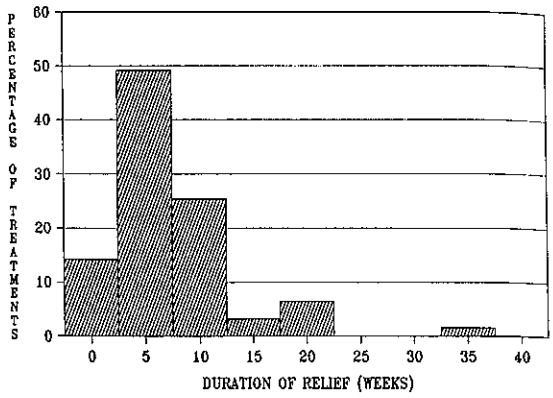

Figure 4: Mean duration of relief for treatments given to blepharospasm patients.

However, we were unable to determine whether these were associated with the treatment.

In this study blepharospasm patients had a mean duration of relief of 7.6 (SD 6.2) weeks compared with 13.4 (SD 7.3) weeks for the hemifacial spasm patients. This was found to be statistically significant using the two sample t-test ($T=3.2$ $p<0.01$) (See Figures 4 & 5).

The mean percentage improvement rated by the patient was higher for the hemifacial spasm patients than for the blepharospasm patients (82.3% compared with 65.2%). This was found to be a significant difference using the two sample t-test ($T=3.7$ $p<0.001$).

Patients rated how debilitated they were by the scale (0-4) above prior to treatment and at one

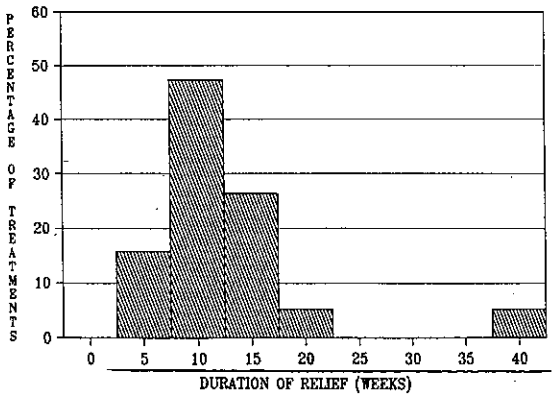

Figure 5: Mean duration of relief for treatments given to hemifacial spasm patients.

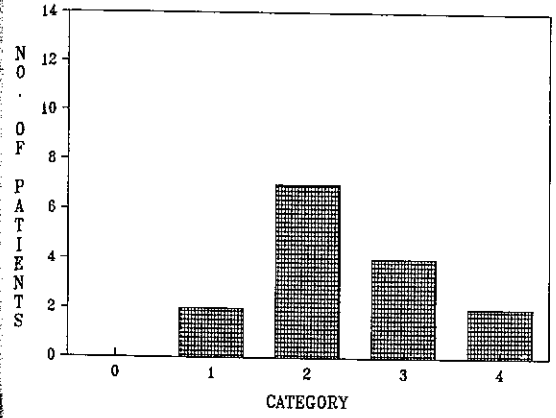

Figure 6a: Pre treatment debility rating for hemifacial spasm patients.

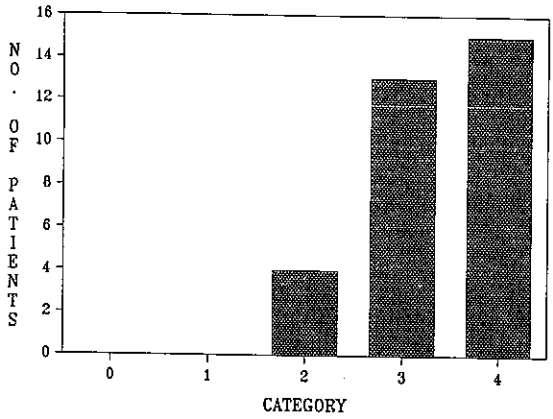

Figure 7a: Pre treatment debility rating for blepharospasm patients.

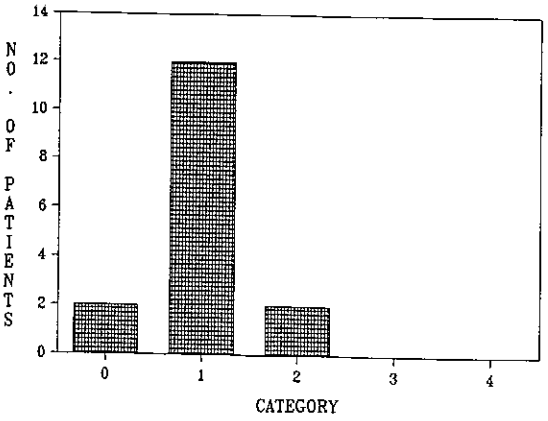

Figure 6b: Post treatment debility rating for hemifacial spasm patients.

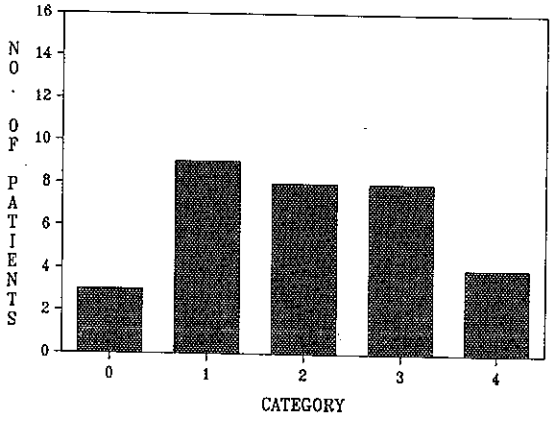

Figure 7b: Post treatment debility rating for blepharospasm patients.

week following each treatment. Overall, patients found that they were less debilitated following the first treatment (See Figures 6 & 7).

When the duration and improvement of the first treatment were compared for those patients with pre-treatment debility rating of 4, 3 or 2, there was no significant difference in the improvement for any of the categories using a one-way anova ($F=1.29$ $p>0.05$). However, there was a significant difference in the duration using a one-way anova ($F=5.95$ $p<0.01$).

For blepharospasm patients that had repeated injections on the same dose, there was no significant change in the duration of the improvement using a one-way anova ($F=0.65$ $p>0.05$) and $F=2.42$ $p>0.05$) respectively.

There was no significant correlation between the dosage and the duration of effect. Using the two sample t-test there was no significant difference for the blepharospasm patients in the duration for those who were given 20 units compared with those who were given 40 units ($T=1.05$ $p>0.05$). However, individual cases did prove to respond to an increase in dose.

The mean duration of effect for the Meige's syndrome patients and the Parkinson's disease patients combined was 4.9 (SD 4.0) weeks. This was found to be significantly less than the general

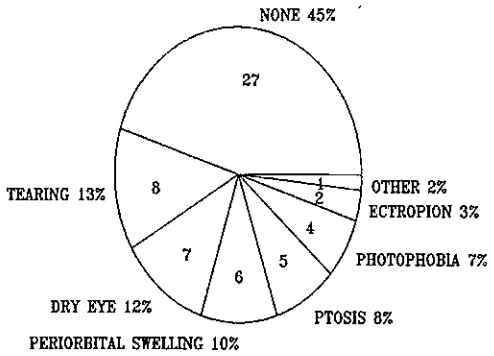

Figure 8a: Percentage of side effects for blepharospasm patients given ≤20 units. (No. of treatments = 60).

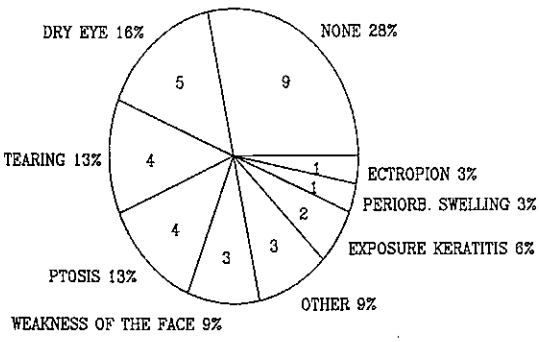

Figure 8c: Percentage of side effects for hemifacial spasm patients (No. of treatments = 32).

blepharospasm group using the two sample t-test ($T = 2.3$ $p < 0.05$). However, there was no significant difference in their improvement following treatment using the two sample t-test ($T = 1.0$ $p > 0.05$).

Six of the patients who had undergone surgery prior to treatment had hemifacial spasm. Using the two sample t-test there was no significant difference in the duration of effect between the 2 groups ($T = 0.09$ $p > 0.05$).

The types of side effects experienced for both blepharospasm and hemifacial spasm patients were ptosis, epiphora, dry or irritated eyes, ectropion, exposure keratitis and puffy lids (see Figures 8a 8b 8c). Other side effects included blurring of vision, bloodshot eyes, dull ache in the eye, local bruising. One patient who had undergone a facial neurectomy for hemifacial spasm and had aberrant regeneration, developed a drooping of the upper lip resulting in difficulty in chewing and speaking, as well as biting the inner cheek when eating. This weakness spontaneously improved.

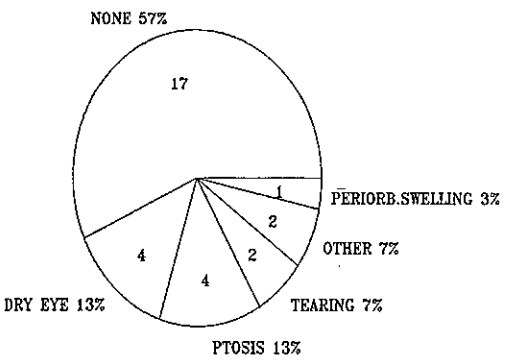

Figure 8b: Percentage of side effects for blepharospasm patients given >20 units (No. of treatments = 30).

For blepharospasm patients that were given a total of less than or equal to 20 units, 45% of treatments produced no side effects. As the dose was increased to greater than 20 units 57% of treatments produced no side effects. However, the rate of ptosis increased from 8% to 13% but the number of patients in this group was small. Of the injections given to hemifacial spasm patients, 26% produced no side effects.

DISCUSSION

The duration of effect of botulinum toxin A for hemifacial spasm patients was longer than for blepharospasm patients (13.4 weeks compared with 7.6 weeks) over all treatments. This is comparable with other studies[28,29] and the improvement for the hemifacial group was higher than the blepharospasm patients (82.3% compared with 65.2%).

The blepharospasm patients who received the same dose with each injection were found to have no significant difference in the duration or improvement. This would suggest that the botulinum does not have a prolonged effect after several treatments possibly due to the atrophy of the orbicularis as has been postulated previously[24] nor did the botulinum appear to lose its effect after several treatments.

It has been speculated that after several treatments a patient could develop antibodies.[24] However, this was found not to be the case in a later study which found no detectable antibodies in the blood subsequent to several injections of botulinum toxin.[30]

Overall, there was no significant correlation between the dose of botulinum and the duration of effect. That is, if the dose is increased the duration will also increase. Furthermore, the improvement did not increase with higher doses. However, there were individual cases that definitely responded to an increase in dose. This may suggest that there is a threshold which may be related to the distribution of the drug in the lids or the distribution and number of receptors. This may be an area of further research.

Side effects were more common in the hemifacial spasm group, occurring with 72% of treatments. The hemifacial spasm patients experienced lower facial weakness unlike the blepharospasm patients and this was most likely due to the fact that most hemifacial spasm patients had injections in the region of the upper cheek to help reduce lower facial spasm. Others have reported the risk of ptosis from one treatment to be approximately 8%[31,32] and this is comparable with our results.

The side effects for blepharospasm patients receiving less than or equal to 20 units were not significantly different to those receiving greater than 20 units. 45% of treatments produced no side effects with patients receiving less than or equal to 20 units. The rate of ptosis increased with the larger doses from 8% to 13% and this has been reported previously.[31]

CONCLUSION

In summary, botulinum toxin provides a viable alternative for the treatment of blepharospasm and hemifacial spasm and in more recent times has become the treatment of choice. Side effects are common but transient and tolerable in most cases. The hemifacial spasm patients who are more likely to suffer from side effects have a longer duration of effect and better improvement in the eyelid spasm.

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