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Optometric correlates of Meares–Irlen Syndrome: a matched group study

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Summary

People who report visual perceptual distortions, typically when reading, that are alleviated by using coloured filters are described as suffering from 'Meares–Irlen Syndrome'. A recent double-masked placebo-controlled trial showed that this condition cannot be solely explained as a placebo effect and that the beneficial filter is idiosyncratic and sometimes needs to be highly specific. Several mechanisms have been suggested for Meares–Irlen Syndrome including ocular motor (binocular and accommodative) anomalies, a sensitivity to patterned stimuli (pattern glare), and a deficit of the transient visual sub-system. We investigated these hypotheses by comparing 16 children, who reported the symptoms described above and who showed a sustained benefit from coloured filters, with 25 control children who came from the same school and were matched for age, reading performance and intelligence. The 'Meares–Irlen Syndrome' group had slightly, but significantly, reduced vergence and accommodative amplitudes and stereo-acuity; they also demonstrated significantly more pattern glare. The two groups did not differ significantly in their visual acuities, refractive error, dissociated or associated heterophoria, AC/A ratio, or ability to perceive 20 Hz flicker. It appears that certain ocular motor factors are correlates of Meares–Irlen Syndrome, rather than the primary underlying cause of the symptoms. The results support the hypothesis that pattern glare may be involved in the mechanism of Meares–Irlen Syndrome.

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Introduction

Meares[1] and Irlen[2] have described a syndrome of visual perceptual distortions and asthenopia, which can be alleviated by coloured filters. This syndrome has been called Scotopic Sensitivity Syndrome or Irlen's Syndrome[3]. It is claimed that the colour of the required filter is idiosyncratic and often needs to be highly specific[4]. A recent double-masked trial has demonstrated a benefit from coloured filters in children with the symptoms described above, a benefit which cannot be attributed solely to a placebo effect[5].

Blaskey and colleagues have argued that the symptoms of Meares–Irlen Syndrome can usually be attributed to ocular motor anomalies[6], and that the most appropriate intervention is vision therapy[7]. In our recent double-masked trial investigating this syndrome[5], we collected extensive optometric data and have recently reported these in detail[8]. These data suggest that an ocular motor mechanism is unlikely to account, in most cases, for the benefit from coloured filters. The present paper explores the optometric correlates of Meares–Irlen Syndrome in more detail by selecting a control group of subjects who were carefully matched to a sub-group of the children with symptoms from the original double-masked trial.

The optometric investigations in this study concentrated on near point functions, where the symptoms of Meares–Irlen Syndrome are most prominent. A previous study had used techniques similar to those in the present study to compare the optometric characteristics of a group of dyslexic

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with age-, gender-, and IQ-matched controls[9–11]. Although it has been claimed that 50% of people with dyslexia have the syndrome (cf. 10% of good readers)[4], the precise relationship between these two conditions remains obscure. Therefore, where appropriate, we have compared the results of the present study of the syndrome with the previous study of dyslexia. The groups in the present study and the groups in the dyslexia study had a similar age (mean 12 years and 10 years, respectively) and IQ (about 90th percentile cf. 75th percentile, respectively).

Methods

Subjects

The minimum selection criterion for the 68 subjects in the double-masked trial[5] was a sustained benefit from coloured overlays. One of the sources of these subjects, a secondary school in Kent, pre-selected the children to be tested with overlays as those reporting symptoms of asthenopia and/or anomalous visual effects whilst reading. Sixteen of the children from this school who had participated in the trial were available for psychometric testing and these comprised the experimental group in this matched-group study. The psychometric tests, which were a group intelligence test (Raven's Progressive Matrices)[12,13] and a group reading test (Suffolk Test)[14], were administered to the experimental group concurrently with the potential control subjects.

For the control group, a further 92 children from the same school were screened (concentrating on those with reading difficulties) with the brief questionnaire that had been used to select the experimental group. Two children were excluded for medical reasons and 12 were excluded because they were not wearing conventional glasses as the prescriber had instructed. Of the remaining subjects, those without symptoms of asthenopia or anomalous visual effects were given the same test with coloured overlays as the experimental group. Forty-seven children reliably reported that none of the colours improved their perception of text and 25 of these were selected from their psychometric test results to form a control group which matched the experimental group in chronological age, intelligence, and reading age. Children with clinically significant refractive errors or ocular motor anomalies were excluded from both groups unless these problems had been corrected for two months[8].

Optometric tests

Monocular and binocular near presenting visual acuities (VAs) were assessed[8] and the refractive errors were assessed by distance retinoscopy[15] in negative cylinder notation. The optometrist's (author BE) retinoscopy result previously had been found to correlate well with the result of a subjective refraction ($n = 82$, $r = 0.96$). The mean spherical equivalent refraction (SER) and mean cylindrical correction for each subject were calculated[8]. Binocular ocular motility, near cover test, near dissociation test, gradient AC/A ratio, Mallett Unit near associated heterophoria, near point of convergence, near vergence reserves, stereo-acuity, and amplitude of accommodation were assessed as described elsewhere[8].

It has been claimed that up to two-thirds of dyslexic children have a deficit of their transient visual system[16]. The 'transient deficit hypothesis', although controversial[17], has been suggested as a mechanism for a benefit from coloured lenses in some cases of dyslexia[18–20]. One method of measuring transient channel activity that has detected deficits in dyslexic populations is to assess the modulation threshold to a virtually homogeneous field that is flickering at 10 Hz or more[10,21]. We investigated flicker perception with apparatus where a stabilised d.c. light source illuminated a diffusing sphere having two apertures, each covered by a polariser, the axes of polarisation being orthogonal. A third polariser rotated in front of the stationary polarisers so as to reduce the light from one aperture while increasing the light from the other. The light from the apertures entered each arm of a bifurcated optic fibre that combined and diffused the light at the focal point of an eye piece. One of the light paths was interrupted by a rotating wheel with sectors of equal angular width and spacing (rotating so as to produce 20 Hz flicker), the other by a neutral filter with equivalent time-averaged transmission. The Maxwellian view system provided a diffuse monocular field in the right eye of 8° that was of constant time-averaged luminance (about 150 cd m^{-2}) and was tolerant of head position. The left eye was occluded and a method of limits was used, with five ascending and five descending readings, in alternation.

'Pattern glare' is an adverse response to mid-spatial frequency high contrast repetitive patterns[22], including lines of text[23], and it has been proposed that this might be a cause of Meares–Irlen Syndrome[3,24]. We tested for pattern glare using the method described elsewhere[8]. Symptoms were investigated using a detailed questionnaire summarised in Evans *et al.*[8]

Results

Psychometric and general descriptive data

The mean reading age of both groups was over 2 years behind their mean chronological ages (*Table 1*). Since the mean intelligence test results of both groups were close to the 50th percentile, this suggests that many of the subjects had a specific reading difficulty (dyslexia). 48% of the control group and 25% of the experimental group were female. One experimental subject wore glasses constantly, another just for reading, and one control subject wore glasses only for reading. These subjects wore their glasses for the near binocular vision, orthoptic, accommodative, and psychophysical tests.

Table 1. Summary of psychometric and general descriptive data

Variable	*Group*	*Mean*	*SD*	*Median*	*Minimum*	*Maximum*
Age	Experimental	12 years 7 months	0.7	12.3	11.6	14.2
	Control	12 years 2 months	0.4	12.1	11.7	12.8
Intelligence	Experimental	44.4	27.6	46.5	8	95
	Control	51.5	25.9	50	1	96
Reading	Experimental	42.1 (9 years 9 months)	9.5	44.5 (10 years 2 months)	27 (7 years 10 months)	57 (>13 years)
	Control	42.7 (9 years 10 months)	14.6	44 (10 years 1 month)	3 (<6 years)	67 (>13 years)

Age is chronological age, intelligence is the percentile score from Raven's Progressive Matrices[12], and reading is the raw score (reading age in parentheses) from the Suffolk reading test[14]. SD is the standard deviation.

Optometric tests

There was no significant difference between the groups' presenting VAs (binocular: control −0.028, experimental +0.005), mean SER (control +0.53 D, experimental +0.77 D), or astigmatism (control −0.38 D, experimental −0.34 D). Ocular motility testing did not reveal any incomitancies, but did detect saccadation of smooth pursuit eye movements in two of the experimental and three of the control subjects. Cover testing at near did not reveal any heterotropia or vertical heterophoria and showed the horizontal heterophoria to be not significantly different in the two groups (Mann–Whitney *U*-test, $P > 0.05$).

The near dissociation test detected a few vertical heterophorias which were not significantly different in the two groups (Mann–Whitney *U*-test, $P > 0.05$). The horizontal results (mean of five readings) were not significantly different either when the type and size of heterophoria (esophoria treated as positive and exophoria as negative values) were analysed nor when the absolute magnitude of heterophoria, regardless of type, was investigated. The range of the five readings was also similar in both groups, as was the mean AC/A ratio.

When the Mallett near associated heterophoria results were investigated in a manner analogous to the near dissociation test, the two groups were again not significantly different. The prevalence of an unstable fixation disparity and of suppression of one of the Nonius strips did not differ significantly in the two groups. The median near point of convergence was at the test ceiling in the control group (4.5 cm) and was a little more remote in the experimental group (5.25 cm). This difference approached, but did not reach, significance (Mann–Whitney *U*-test, $P = 0.085$). The experimental group had significantly poorer (Mann-Whitney U, $P = 0.0022$) stereo-acuity (median 35″) than the control group (median 20″).

The vergence amplitude is the total range over which a person can change their vergence and this was significantly reduced in the experimental relative to the control group (*Table 2*). Evans *et al.*[9] found that the most significant ocular motor correlates of dyslexia were reduced convergent and divergent amplitudes, resulting in a very significantly reduced vergence amplitude. Interestingly, the divergent reserves of the Meares–Irlen Syndrome group in the present study (*Table 2*) were slightly worse than the dyslexic group in the previous study (mean break point 13.6 ± 5.4 Δ). The divergent reserves of the control poor readers in the present study, however, were similar to those of the control *good* readers in the previous study (mean break point 16.1 ± 6.2 Δ). Similarly, the difference between the convergent reserves of the Meares–Irlen Syndrome group and the poor readers in the present study was similar to that between the dyslexic group (mean break point 15.4 ± 6.7 Δ) and the control good readers (mean break point 19.0 ± 7.8 Δ) in the previous study. These data may suggest that

Table 2. Descriptive and comparative statistics for vergence reserves (in Δ)

Variable	*Group*	*n*	*Mean*	*SD*	*P*
Divergent Blur	Control	20	13.7	4.6	0.004
	Experimental	14	9.2	3.35	
Divergent Break	Control	25	15.9	3.5	0.006
	Experimental	15	12.4	4.1	
Divergent Break–recovery	Control	25	4.1	1.9	0.003
	Experimental	15	2.3	1.1	
Convergent Blur	Control	14	13.4	5.4	0.185
	Experimental	10	9.7	7.9	
Convergent Break	Control	25	16.8	7.5	0.180
	Experimental	13	12.9	9.6	
Convergent Break–recovery	Control	25	4.4	2.8	0.053
	Experimental	13	2.4	3.3	
Amplitude Blur	Control	11	27.8	9.4	0.024
	Experimental	9	17.8	8.6	
Amplitude Break	Control	25	32.7	8.9	0.027
	Experimental	13	25.2	10.6	

n is total number, SD is the standard deviation. The *P* values in the right-hand column were calculated using the unpaired *t*-test.

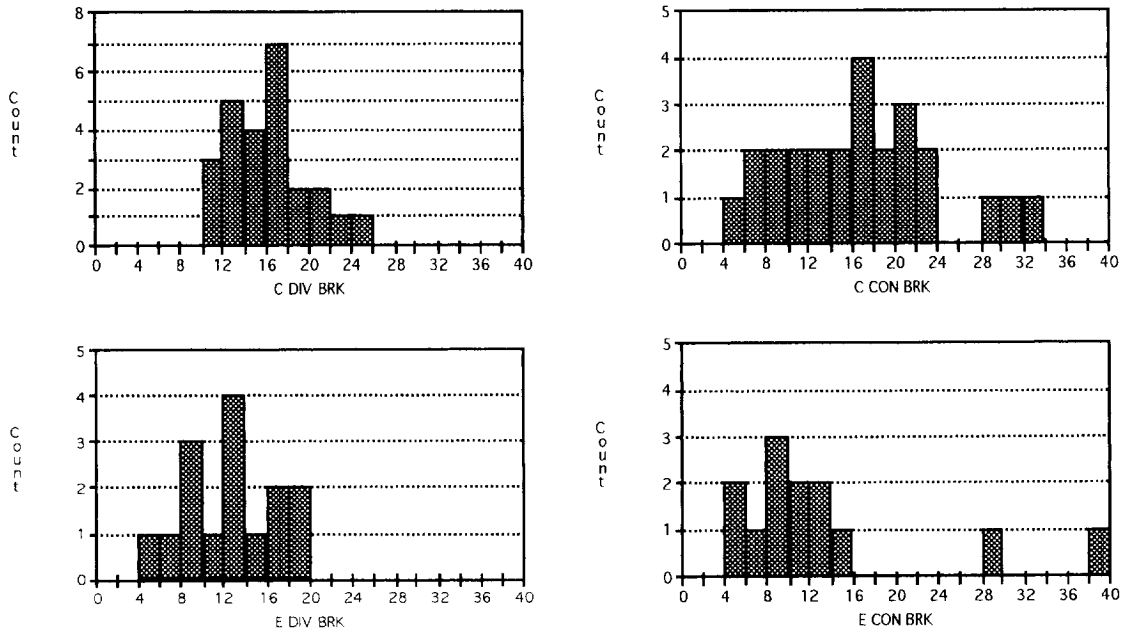

Figure 1. Frequency distributions of the vergence reserves (in prism D) to break (diplopia) point (BRK) for divergence (DIV) and convergence (CON) of the control (C) and experimental (E) groups.

dyslexic children with Meares–Irlen Syndrome tend to have poor vergence reserves, and those without the syndrome do not. In other words, binocular instability may be a correlate of the syndrome, which only occurs in some people with dyslexia.

We were surprised that the difference between the convergent break points of the two groups in the present study did not reach significance. This may be explained by *Figure 1*, which shows that the convergent reserves of the Meares–Irlen Syndrome group were considerably reduced compared with the controls, with the exception of two 'outlying' subjects. This observation was confirmed by a Kolmogorov–Smirnov test, which suggests that the distribution of the two populations was rather different (two-tailed, $P = 0.078$).

Although the associated heterophoria data suggested that the heterophorias were equally well compensated in both groups, we investigated compensation further by calculating the difference between the heterophoria and the opposing vergence reserve ("Sheard's value"). The experimental group had a little less "vergence in reserve", although the difference between the two groups did not reach significance ($P = 0.056$).

The mean accommodative amplitude was reduced in the experimental group (*Table 3*). The order in which the measurements were taken was right, left, then both eyes. In the control group, the amplitude increased in consecutive conditions; this practice effect was not apparent in the experimental group. This may be why the right eye result was not significantly different in the two groups or, alternatively, there could have been a greater fatigue effect in the experimental group.

Table 3. Descriptive and comparative statistics for accommodative amplitudes (D)

Eye	*Group*	*n*	*Mean*	*SD*	*P*
Right	Control	24	15.4	3.7	0.240
	Experimental	14	13.9	3.7	
Left	Control	24	16.2	3.4	0.017
	Experimental	15	13.1	4.1	
Both	Control	24	17.1	3.6	0.014
	Experimental	15	13.9	4.2	

Key to abbreviations as in *Table 2*.

Psychophysical data

Flicker data were collected from 21 of the control subjects and 11 of the experimental subjects. The flicker threshold did not differ significantly between the two groups (Mann–Whitney *U*-test, $P = 0.42$). It has been suggested that there may be two sub-groups of dyslexic people with hyper- and hypo-sensitive transient visual systems[25,26]. The distributions in *Figure 2* do not confirm this hypothesis, although the sample size is small. The experimental group reported more anomalous visual effects (pattern glare) than the control group in viewing the experimental grating (Mann–Whitney *U*-test, $p = 0.025$), but both groups experienced a similar amount of anomalous visual effects on viewing the control grating (Mann–Whitney *U*-test, $P = 0.90$). The hemifield results were not significantly different in the two groups, although both groups saw more illusions in the left hemifield condition than the right.

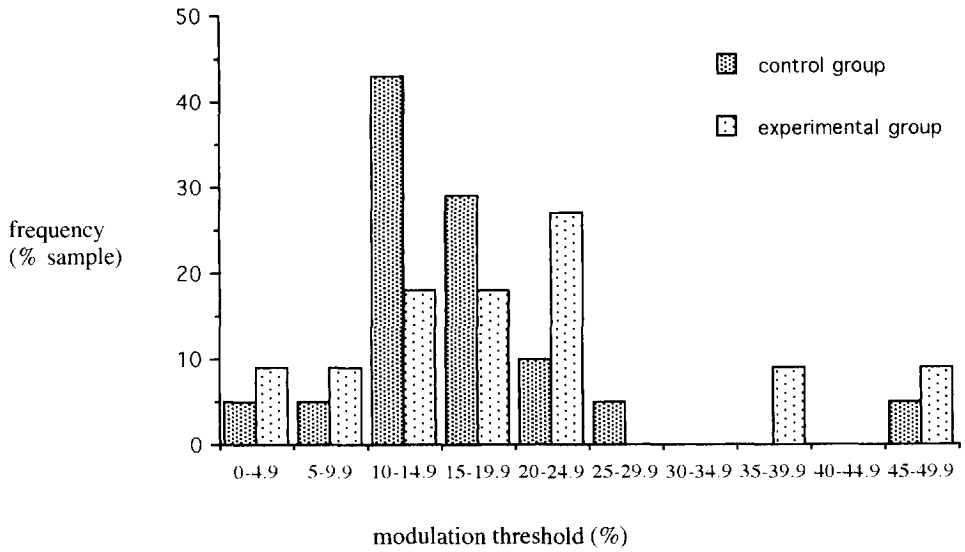

Figure 2. Frequency distributions of flicker threshold [% modulation; $(L_{max} - L_{min})/(L_{max} + L_{min}) \times 100$] in the control and experimental groups.

Symptomatology

The symptom questionnaires were completed by 14 members of the experimental group (88%) and 15 (60%) of the control group. The two groups did not differ significantly in the proportion of respondents who reported a previous eye examination, a history of developmental abnormalities, or allergies, or seizures. The data on seizures may have suffered from a 'floor effect', since two out of 13 experimental group respondents reported a history of seizures compared with none of the 14 control respondents. Five (36%) of the experimental respondents and none of the controls had a history of ocular (mainly orthoptic) anomalies (Fisher's exact probability test, $P = 0.01$). All the respondents stated that their general health was good and only two subjects (one in each group) gave details of any medication: both for hayfever and asthma.

The questionnaire responses relating to visual symptoms should be interpreted with caution since similar, though less specific, questions were used as selection criteria. Although the experimental subjects were significantly more likely to report certain symptoms (distance transient blur, near blur, near transient blur, words sometimes jumping around and changing size, confusing letters or words, reversing letters or words, skipping or re-reading or omitting words or lines, reading slowly, tiring easily/having a short attention span, and light sensitivity), they did not report significantly more of other symptoms (words fading/disappearing, words having faint colours, diplopia, tendencies to close or cover one eye, to hold print at unusual distances, to blink excessively, tilt their head on one side, move their head when reading, to use their finger as a marker, or to describe poor general co-ordination). This militates against the argument that the experimental group's increased symptoms resulted from greater suggesti-bility. The similar prevalence in the two groups of diplopia and of a tendency to close or cover one eye when reading suggest that undetected binocular anomalies do not account for the benefit from filters in the Meares–Irlen Syndrome group.

The significantly greater number of headaches reported by the experimental group may simply reflect the use of headache in the selection criteria. The factors that may, when associated with a headache, suggest a diagnosis of migraine were not reported more often by the experimental group. The questionnaire did not reveal any significant differences between the two groups in the prevalence of a family history of specific learning difficulties, orthoptic anomalies, migraine, colour vision defects, or epilepsy.

Discussion

The similarity of the near visual acuities and refractive errors of the two groups means that a refractive mechanism for Meares–Irlen Syndrome is unlikely. The type and degree of heterophoria was also similar in the experimental and control groups. Although the low vergence reserves might suggest a reduced ability to overcome heterophoria in the syndrome group, the vergence reserves were, in clinical terms, only slightly lower in the experimental than the control group. Further, Sheard's value (a measure of the ability of the vergence reserves to overcome the heterophoria) was not significantly different in the two groups and data from the double-masked trial shows that Sheard's value was not significantly related to the benefit from coloured filters nor to symptoms in Meares–Irlen Syndrome[8]. In

the present study, the Mallett Unit associated heterophoria, which is the best predictor of whether a heterophoria is symptomatic or not[27], was not significantly different in the two groups. We believe that these results, taken together with other recent research[8], suggest that the low vergence reserves are primarily a correlate rather than a cause of the syndrome.

It is interesting that a previous study found low vergence reserves to be a correlate of dyslexia[9]. The present data suggest that the dyslexic children who have low vergence reserves may also be those who have Meares–Irlen Syndrome. A slightly, but significantly, reduced amplitude of accommodation is another ocular motor correlate of dyslexia which, in the present study, was also a correlate of the syndrome.

The ocular motor correlates of dyslexia have been linked with certain sensory visual correlates, most notably impaired flicker perception[11]. The present study failed to detect any difference between the experimental and control groups in their ability to detect 20 Hz flicker. This could be because of the experimental technique that was adopted, although Evans *et al.*[8] also found that the spatial contrast sensitivity function deficit in dyslexia[10] was not present in a group of subjects with Meares–Irlen Syndrome.

It is conceivable that children with Meares–Irlen Syndrome share the ocular motor correlates of dyslexia, but lack the sensory visual correlates of dyslexia. However, we feel that more rigorous psychometric techniques should be used to evaluate sensory visual function in Irlen's Syndrome before this conclusion can be stated with confidence. We would stress that the ocular motor deficits that we have detected in the syndrome are very subtle and are unlikely to account for our subjects' symptoms, nor for their benefit from coloured filters. Future work should address the possibility of a common underlying cause for Irlen's Syndrome and for the low vergence and accommodative amplitudes. The claim[28,29] that the benefit from coloured filters results from a 'transient processing deficit' is hard to substantiate from the evidence available at present.

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