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Interocular Transfer of the Movement Aftereffect in Central and Peripheral Vision of People With Strabismus

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Purpose. To compare binocularity in central and peripheral vision of people with early-onset strabismus and people with normal binocular vision.

Methods. Ten subjects with early-onset strabismus, and nine subjects with normal binocular vision were tested. To assess binocularity, interocular transfer (IOT) of a rotary movement aftereffect (MAE) was measured. The MAE stimuli were either confined to the central 2.8° of the visual field or were presented 10° into peripheral vision.

Results. In peripheral vision, there was no significant difference in IOT for the two groups of subjects. In central vision, there was a significant decrease of IOT in subjects with early-onset strabismus. Their IOT was, however, significantly greater than zero.

Conclusions. Early-onset strabismus appears to spare binocularity in peripheral vision but reduces it in central vision. It does not abolish binocularity assessed by IOT of MAE, suggesting that some binocular connections survive early-onset strabismus, even in central vision. Invest Ophthalmol Vis Sci. 1994;35:313–317

Interocular transfer (IOT) of aftereffects has been used to assess binocularity in people with strabismus, anisometropia, and unilateral amblyopia.[1] This research is predicated on the suppositions that IOT requires binocularity[2] and that early binocular insult disrupts binocular connections, as evidenced by recordings from single cells in the visual cortex of animals.[3] Initial findings suggested that the amount of IOT was a good index of the amount of binocularity.[1] Those with early-onset strabismus appeared to show no IOT.

The most comprehensive recent investigation, however, suggests that IOT is not a clear-cut index of binocularity.[4] Although Mohn and van Hof-van Duin found that mean IOT from people with early-onset strabismus, amblyopia, and no stereopsis was less than the mean IOT of normals, there were major individual differences, such that some stereoblind observers

showed normal levels of IOT. We confined our study to the movement aftereffect (MAE). We hoped to clarify matters by incorporating two design elements shown even more recently to be crucial in measuring IOT. First, we ensured that no stationary contours were presented to the nonviewing eye during either induction or testing of the MAE. Conjugate eye movements entrained by the eye that views the moving induction stimulus will make stationary contours presented to the other eye move on its retina. Such retinal-image movements are suspected of augmenting, or simulating, IOT via induced movement.[5]

The second aspect of measuring IOT of MAE we thought to be crucial was whether the MAE stimuli were central or peripheral. IOT of threshold elevation has been found to be greater in the peripheral field in strabismic amblyopia than in central vision, consistent with the notion that binocular connections are spared in the periphery.[6] Previous quantitative research on IOT of MAE has used central fields of at least 10° in diameter, so IOT could have arisen from the peripheral field. We masked our stimulus so that it was either visible within a central, circular field of 2.8° in diameter or within an annulus with an internal diameter of 20° and an external diameter of 40° of visual angle. We used an eight-spoked radial grating to equate visibility in central and peripheral vision.[7] Our use of a 2.8° field should confine the stimulus to central vision and give the best chance of determining whether IOT is abolished in people with early-onset strabismus.

METHODS. The research followed the tenets of the Declaration of Helsinki. All subjects volunteered for the experiment and gave written, informed consent. Permission was granted to experiment on humans by the Otago Area Health Board.

Subjects with early-onset strabismus (with or without anisometropia) were selected from the files of the Department of Ophthalmology, Dunedin Hospital, and recruited by letter. All evidenced strabismus before 2 years of age. All but one had had at least one operation to align the eyes. Ages ranged from 12 to 44 years. Initially, there were six female and five male subjects, but one male subject was dropped from the experiment at his own request. Eight subjects showed no stereopsis measured with targets of varying disparity presented in a Synoptophore, the stereo-optical fly test, and the TNO test for stereoscopic vision. Two showed some evidence of stereopsis on the fly test, although we note that this test can be passed with monocular viewing. Details concerning the fully participating experimental subjects are given in Table 1.

Two subjects with normal vision were also recruited in the same way. They were patients who had been examined early for suspected binocular disorders but whose vision proved to be normal. The re-

maining normal subjects were volunteers from among the students and staff of the University of Otago. Ages ranged from 12 to 37 years. There were seven male and two female normal subjects. All had normal or corrected-to-normal visual acuity in each eye and good stereopsis from random-dot stereograms.

The apparatus consisted of a light-proof box with an eyepiece and shutter at one end through which an eight-spoked, square-wave, radial grating was visible at the other end. The shutter allowed the stimulus to be viewed by the left or the right eye from a viewing distance of 57 cm. One of two field stops were used: for central vision, a 2.8°-diameter circular area was displayed; for peripheral vision, an annulus with an internal diameter of 20° and an external diameter of 40° were displayed. Both stimuli had a white, central fixation spot of 0.5° in diameter. Luminance of the light parts of the grating and the fixation spot was 44.30 cd/m^2, luminance of the dark areas was 3.06 cd/m^2, and Michaelson contrast was 0.87.

The grating was rotated at 25 revolutions per minute by a Lafayette Color Mixer and Series 200 control motor (model 13012) (Lafayette Manufacturing Co., Lafayette, IN). Subjects tracked the MAE by turning a plain knob at the same speed and in the same direction as any illusory rotation. This knob turned a pointer over a protractor, visible only to the experimenter. We used this measure rather than the traditional measure of the duration of the MAE for two reasons. First, our previous studies of the MAE[5] showed durations of the MAE were highly variable, even within the same subject, being affected by changes in response criteria. If the strength of the MAE decays exponentially, then small changes in the strength at which a subject decides the MAE has ceased will lead to large differences in the time recorded. Small adjustments of the position of the knob, however, lead to small variations in the overall rotation recorded, thereby reducing variability. Second, depending on how durations are recorded, such a measure can lead to spurious recording of an MAE when there is none. A subject takes some time to decide that a stationary stimulus really is stationary. If a subject has to press a button, or announce, when the MAE has ceased, this delay means that a zero duration measure is not possible. With the knob, however, if the subject sees no rotation, he or she does not turn the knob, which leads to a meaningful zero score.

Standard instructions were read to all subjects. Subjects responded to calibration trials to give them practice at turning the knob to track real movement and to show them the sorts of stimuli they would view in the experimental trials. On each calibration trial, the stimulus rotated at 25 revolutions per minute for 5 seconds. The subject was instructed to fixate the central spot and to turn the knob at the same speed and in

TABLE 1. Experimental Subjects' Biographic and Optometric Data

Subject	Age (yrs)	Sex	Acuity	Refraction	Str Aniso	Onset (yrs)	1st Surg (yrs)	Stereo (sec)
CF	22	F	R 6/6 L 6/6	0 0	LCS N	≤2.0	12.0	>800
DR	14	F	R 6/5 L 6/9	+1.25/−.50 × 165 +1.00	LDS L	0.2	1.8	Nil
EA	30	F	R 6/9 L 6/5	0 0	RDS R	≤1.0	4.0	Nil
IH	15	F	R 6/5 L 6/9	+4.75/+1.75 × 60 +5.00/+1.75 × 105	LCS L	1.5	4.0	>400
NR	22	M	R 6/60 L 6/5	+5.0/−1.50 × 125 0	RCS R	≤2.0	NA	Nil
PR	16	M	R 4/4 L 4/5	+4.00/+0.25 × 60 +5.50/+1.0 × 115	LCS L	1.3	3.1	Nil
PS	18	M	R 6/5 L 6/5	0 0	RDS N	≤2.0	16.0	Nil
RC	12	F	R 4/3 L 4/3	0 0	RDS N	2.0	4.0	Nil
RK	44	M	R 4/4 L 4/5	+4.25 +5.00	LDS N	≤2.0	4.0	Nil
SB	16	F	R 4/8 L 4/3	+7.00/+0.50 × 30 +6.25/+0.50 × 170	RCS R	1.9	5.0	Nil

Acuity is Snellen acuity in m; R = right eye; L = left eye. Strabismus (Str) and ansiometropia (Aniso): L = left eye, R = right eye, C = convergent, D = divergent, A = alternating, S = strabismus, n = Normal.

the same direction as the stimulus. There were eight trials consisting of the full crossing of direction of rotation (clockwise/counterclockwise), viewing eye (left/right), and retinal location (central versus peripheral).

Next, subjects had the central, direct MAE demonstrated. Subjects were instructed to look with their preferred eye into the apparatus and to fixate on the white spot. The experimenter then set the stimulus into rotation for 1 minute. At the end of this time, the stimulus was made stationary and the subject was asked to say what he or she saw. All but three subjects spontaneously reported the MAE. These three reported the MAE on a second trial.

Finally, subjects responded to 16 experimental trials. Throughout each trial, subjects were required to fixate the central spot. Subjects viewed the rotating stimulus for 1 minute; that was followed by a 1-second dark period and then by the stationary test stimulus. Subjects were asked to respond to any MAE they saw by turning the knob in the same direction and at the same speed as the grating appeared to be moving. They were asked to remove their hands from the knob when they were sure the aftereffect had stopped. After the subject finished responding, there was a rest period of at least 45 seconds before the next trial. Order of trials was random.

For each subject, the experimental design consisted of four factors:

Direct/IOT. In *Direct*, the adapting eye and the test eye were the same; in *IOT*, the adapting eye and the test eye were different.

Test eye. The subject viewed the test stimulus with either the left eye or the right eye.

Retinal location. The subject viewed either a central stimulus subtending 2.8° of visual angle on the retina or a peripheral stimulus 10° from the center of fixation and subtended 10° of visual angle in peripheral vision.

Direction of rotation of the stimulus during adaptation was varied so as to be clockwise on half the trials and counterclockwise on the other half. This gave a 2 × 2 × 2 × 2 design, resulting in 16 trials per subject.

RESULTS. A datum for a subject from each trial was the amount in degrees he or she rotated the knob; this gave the size of the MAE for that trial. We noticed that subjects who reported large MAEs also had large variability in their data. For example, the correlation between mean MAE and standard deviation was $r(18)$ = .935, $P < .001$. To enable statistical analyses of these data by analysis of variance (ANOVA), we normalized the data by adding one to each datum (so that no data were zero) and then took the natural logarithm. We subjected these data to an ANOVA with four within-subjects factors (direct/IOT, central/peripheral, test left eye/right eye, and clockwise/counterclockwise) and one between-subjects factor (normal versus strabismic). We found that direct MAEs were greater than IOT, $F(1,17) = 84.70$ and $P < .001$, and that central MAEs were greater than peripheral MAEs, $F(1,17) = 66.28$, $P < .001$. Critically, however, there was a three-
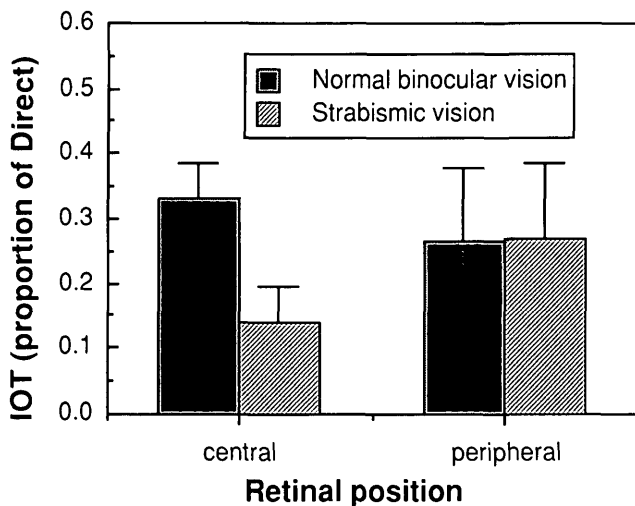
FIGURE 1. Mean IOT, expressed as a proportion of each subject's direct MAE, for those with normal and those with strabismic vision, plotted as a function of retinal location (central versus peripheral). Vertical bars show standard errors.

way interaction between these two factors and subjects' binocular vision, $F(1,17) = 14.33$, $P < .01$. Direct MAEs were similar for the two groups of subjects in both central and peripheral vision, but there was less IOT MAE in the central vision of subjects with strabismus. There were no other meaningful significant main effects or interactions.

To assess statistically the three-way interaction, we expressed each subject's raw, nonnormalized, mean, central, and peripheral IOT MAEs as proportions of the mean direct MAE from the same conditions. The results are presented in Figure 1.

Figure 1 shows that IOT in the two groups of subjects was essentially equal for peripheral MAEs, $t(15) = .02$, but that those with disordered binocular vision had less IOT than those with normal vision for central MAEs, $t(17) = 2.49$, $P < .05$. Nevertheless, the amount of central IOT shown by subjects with disordered binocular vision was significantly greater than zero, $t(7) = 2.67$, $P < .05$. The results from the two subjects with strabismus who had some evidence of stereopsis were not notably different from those of the others. Their data can be deleted without any significant change in the pattern of results.

DISCUSSION. The general level of IOT of MAE, expressed as a proportion of direct MAE and shown in Figure 1, lies between about .25 and .35. This is smaller than the level arrived at in previous research,[8] although it is within the normal range.[4] There are at least two explanations for this low level of proportionate IOT: We used a different measure of the total extent of rotation than was used in previous research. Previous studies that have used measures similar to ours[4] have found less IOT than studies measuring, say,

duration of MAE.[9] Second, we used atypical MAE stimuli—small central stimuli and stimuli visible at least 10° into peripheral vision. Whatever the explanation for the general level of IOT, the important comparison for our purposes concerns the results of those with compromised and those with normal binocularity.

Comparison suggests that IOT levels of MAE equal to those found in normal subjects can be recorded from the periphery of subjects with early-onset strabismus. In central vision, however, the IOT level is less than it is in normal subjects. This supports the idea that only central vision is compromised by early binocular insult.[6] One implication for future studies would be the necessity of measuring peripheral stereopsis.

Nevertheless, even with an MAE confined to central vision, we found significant amounts of IOT in those with disordered binocularity, suggesting appreciable binocular connections in central vision. This is consistent with physiological studies showing that with binocular testing, cortical binocular connections survive early binocular insult.[10] The question, however, persists: Why did our subjects evidence such connections with dichopic testing?

To put the question another way: Why should people with early-onset strabismus show binocularity via IOT yet so little evidence of binocularity via stereopsis? At least two possible explanations can be imagined: First, stereopsis and aftereffects may depend on different channels within the visual system that are differentially sensitive to disruption by strabismus. Second, the two phenomena might be mediated by the same channel, with the binocular disruption effected by inhibition. In this account, inhibition would be maximal when each eye is tested simultaneously, as with stereopsis, but reduced when the eyes are tested sequentially, as in IOT. It remains to be learned which of these possibilities is more likely.

Key Words

Acknowledgments

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Confirmation of the Validity of the Psychophysical Light Scattering Factor

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Purpose. To reevaluate the validity of the light scatter factor (LSF) formula of Paulsson and Sjöstrand, $LSF = L/E$ $(M_2/M_1 - 1)$, where L is the target luminance, E is the illuminance of the glare source at the eye, and M_2 and M_1 are modulation contrast thresholds measured with and without the glare source, respectively. This equation has recently been deemed invalid by Yager, Yuan, and Mathews.

Method. Ratios of contrast thresholds with and without glare were measured for three glare illuminance levels for each of three stimulus luminances. This results in five different ratios of L/E, spanning a range of 1.60 log units.

Results. The data show an excellent fit to the Paulsson and Sjöstrand equation, and the LSF scores conform well to previously published normative values.

Conclusion. The light scatter factor equation of Paulsson and Sjöstrand is confirmed as valid without resorting to the need for correction factors based on variables such as pupil size. Invest Ophthalmol Vis Sci. 1994;35:317–321

As a result of the imperfections of the ocular media, light from any peripheral glare source will be scattered within the eye, with some falling on the fovea. This scattered light causes a reduction in visual performance, commonly termed disability glare. A measure of the quality of the ocular media may be obtained by determining the proportion of illuminance arriving at the eye from a peripheral glare source (E), which is subsequently scattered to the fovea. This has the same effect as an equivalent veiling luminance, Leq, superimposed upon the stimulus itself.

The light scattering factor (LSF) of the eye can be defined as

$$LSF = Leq/E \qquad [1]$$

The veiling luminance reduces stimulus contrast by a factor $L/(L + Leq)$, where L represents mean stimulus luminance. Therefore, the ratio of contrast thresholds measured with and without the presence of a glare source (M_2 and M_1, respectively) is given by

$$M_2/M_1 = (L + Leq)/L = 1 + Leq/L$$

thus,

$$Leq = [(M_2/M_1) - 1] L$$

substituting into [1],

$$LSF = (L/E) \cdot (M_2/M_1 - 1) \qquad [2]$$

As Paulsson and Sjöstrand[1] point out, this equation allows an intrinsic light scattering factor to be determined for any given glare angle. In addition, the LSF calculated in this way should remain independent of the precise stimulus conditions used for its determination. The reason for this is that variations in L and E should be counteracted by corresponding variations in contrast thresholds.