CONTRAST SENSITIVITY ACROSS THE VISUAL FIELD IN CHILDREN WITH AMBLYOPIA

A thesis presented to the graduate faculty of New England College of Optometry in partial fulfillment of the requirements for the degree of Master of Science

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This manuscript has been read and accepted by the Thesis Committee in satisfaction of the thesis requirement for the degree of Master of Science

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Abstract

CONTRAST SENSITIVITY ACROSS THE VISUAL FIELD IN CHILDREN WITH AMBLYOPIA

Mariam Abdel-Kader

New England College of Optometry, 2026

Purpose:

Adults with amblyopia exhibit reduced contrast sensitivity, primarily at high spatial frequencies in central vision. Little is known regarding contrast sensitivity deficits in the peripheral visual field of amblyopic children. In the present study, we assessed monocular contrast sensitivity at three retinal eccentricities in children with anisometropic or strabismic amblyopia as well as typically sighted controls.

Methods:

Contrast sensitivity was measured at 0°, 4°, and 8° of retinal eccentricity in children aged 6-16 with anisometropic amblyopia (n=10), strabismic amblyopia (n=9), and age-matched controls (n=11). Bandpass-filtered Auckland optotypes were monocularly presented at each eccentricity, and spatial frequency was scaled accordingly. Contrast levels were determined using a QUEST adaptive staircase procedure, and fixation was monitored with a Gazepoint eyetracker. Thresholds and fixation compliance were compared across eccentricity, group, and eye using a linear mixed effects model.

Results:

Collapsed across eccentricities, amblyopic eyes in the amblyopic groups had reduced contrast sensitivity compared to fellow eyes ($p<0.001$ for anisometropic, $p=0.045$ for strabismic) with no significance between eyes in controls ($p=0.56$). When comparing across eccentricity both amblyopic groups

showed centralized contrast sensitivity deficits in the amblyopic eye as compared to the fellow eye at 0° and 4°.

Conclusion:

Our findings confirm that both children with anisometropic and strabismic amblyopia demonstrate centralized contrast deficits in their amblyopic eye. In the periphery, these deficits were reduced for both groups, but with a more pronounced effect of eccentricity for anisometropic amblyopes. These findings may inform our understanding of dichoptic therapies for amblyopia that target central or peripheral areas of the visual field.

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Introduction

Amblyopia Overview and Diagnosis

Amblyopia is the leading cause of uncorrectable vision loss in children with a prevalence of 2-4% worldwide (Levi, 2020; Webber, 2018). Unilateral amblyopia is clinically defined as an interocular best-corrected visual acuity difference of two or more lines of Snellen visual acuity (Maurer & McKEE, 2018; Wallace et al., 2018). In addition to reduced visual acuity, individuals with amblyopia experience many other visual deficits, such as a reduction in contrast sensitivity, binocular vision, spatial vision, and stereopsis (Levi, 2020; McKee et al., 2003; Wiecek et al., 2024). These deficits have been primarily evaluated in central vision in adults, while much less is known about peripheral deficits in children with amblyopia (Levi, 2020).

Amblyopia is classified based on etiology and can be characterized as anisometropic, strabismic, or deprivational (Maurer & McKEE, 2018). It is most commonly diagnosed in children born premature or small for gestational age, those with developmental delays, and in children with a first degree relative with amblyopia (Wallace et al., 2018). When amblyopia presents monocularly, there are key risk factors defined that lead to its development. A constant unilateral strabismus occurring at distance and near, anisometropic refractive error of a certain amount (myopia, hyperopia, astigmatism), or a congenital cataract/visual deprivation can all lead to the development of amblyopia (Dorr et al., 2019; Levi, 2020; Maurer & McKEE, 2018). These risk factors must occur within the critical sensitive period (up to the ages of 6-8 years) to result in an amblyopic deficit (Levi, 2020; McKee et al.,

2003). Clinically significant anisometropic amblyopia is generally associated with interocular refractive error differences of approximately ≥1.00 D for hyperopia, ≥1.50 D for astigmatism, and ≥3.00 D for myopia, based on commonly used inclusion criteria in Pediatric Eye Disease Investigator Group (PEDIG) studies (Cotter et al., 2006). However, population-based data from the Vision in Preschoolers (VIP) Study indicate that amblyopia risk may begin at lower levels of anisometropia (approximately ≥0.50 D), increasing in a dose-dependent manner (Pascual et al., 2014). The American Academy of Ophthalmology's preferred practice pattern defines unilateral amblyopia as an interocular best-corrected visual acuity difference of two or more lines accompanied by one of the aforementioned amblyogenic risk factors (Wallace et al., 2018).

Amblyopia and Child Development

Due to the poor development of the visual cortex and subsequent disruption in binocular vision in children with amblyopia, a feedforward mechanism may be at play with other aspects of child development, thus affecting tasks that rely on vision (Birch & Kelly, 2023). These deficits may be seen in a child's hand-eye coordination, reading and school performance, or in navigating their daily environment (Birch & Kelly, 2023). Fine motor skills and reading are important milestones in a child's life that are dependent on functional binocular vision, and the disruption of their early visual information can hinder their development and academic performance (Birch & Kelly, 2023). Reduced contrast sensitivity, as we primarily see in individuals with amblyopia at high spatial frequencies, may play a contributing role to many of the aforementioned functional deficits, thus revealing the

importance of understanding the extent of contrast sensitivity deficits in children with amblyopia (Bradley & Freeman, 1981; Mohammadi et al., 2019).

The Neural Basis of Amblyopia

Amblyopia typically occurs when abnormal visual experience of a child hinders the development of the visual pathway in one or both eyes, resulting in reduced visual acuity (with no observed ocular pathology) that is not optically correctable (Barrett et al., 2004). Animal models have been used to better understand the neural basis of amblyopia. Hubel and Wiesel conducted pioneering studies on cats and found that the principal abnormality in amblyopia occurs at the primary visual cortex (Hubel & Wiesel, 1962). In these studies, a shift in the ocular dominance columns to the non-amblyopic eye was observed (Barrett et al., 2004; Hubel & Wiesel, 1962; Kiorpes, 2019). Additionally, studies with non-human primate models of severe amblyopia demonstrated that V1 neurons driven by the amblyopic eye responded to lower spatial frequencies than those from the fellow eye (Barrett et al., 2004; Kiorpes et al., 1998). This finding is consistent with psychophysical studies that report a lower sensitivity to high spatial frequencies in amblyopic eyes (Bradley & Freeman, 1981).

Measuring Contrast Sensitivity

Contrast sensitivity for an individual provides information on their threshold between what they can and cannot perceive in their visual environment (Pelli & Bex, 2013). Contrast sensitivity can provide additional information about the visual system as it is a more sensitive measurement of visual function, and may detect deficits in individuals with a normal level of visual acuity (Pelli & Bex, 2013). Psychophysics can be used as a method of estimating an

individual's contrast threshold through the presentation of stimuli with varied levels of contrast per trial (Pelli & Bex, 2013). In these experiments, responses for each trial are scored to be correct or incorrect and these scores are used to create a psychometric function depicting the observer's likelihood of a correct response as a function of stimulus contrast level (Pelli & Bex, 2013). There are several methods that are used to determine the contrast level that will be presented at each trial. The method of constant stimuli utilizes a predetermined set of contrast levels that are tested in random order, requiring extensive trials to test a wider range of contrasts (Pelli & Bex, 2013). However, an adaptive staircase model is more commonly used for these experiments which implements the likely parameters of previous responses to predict the level of contrast that will be presented in the subsequent trial to obtain contrast thresholds in a more efficient manner (Pelli & Bex, 2013; Watson & Pelli, 1983). QUEST is an example of an alternative adaptive staircase model which utilizes the Bayesian estimate of threshold to predict subsequent stimuli (Watson & Pelli, 1983). An alternative forced choice task (AFC) can be used as a response method in which observers are provided with stimuli choices to choose from when asked to identify the test stimulus that was presented in each trial (Pelli & Bex, 2013). In order to decrease probability of guessing the test stimulus correctly, it is beneficial to increase the number of choices in an AFC task (Pelli & Bex, 2013).

Once contrast threshold is obtained for several spatial frequencies in an observer, a contrast sensitivity function can be obtained. The contrast sensitivity function provides a graphical representation of contrast thresholds plotted as a function of spatial frequency, typically consisting of contrast threshold at 5 or more spatial frequencies within the range of

1-16 cycles per degree plotted on a logarithmic scale (Pelli & Bex, 2013). While the full contrast sensitivity function can provide valuable information regarding an observer's visual function, obtaining this data requires extremely lengthy testing due to the high number of required trials, and can be particularly difficult to obtain in vulnerable populations such as children (Pelli & Bex, 2013; Rogers et al., 1987).

Clinically, contrast sensitivity is not routinely measured due to limited timing, however there are several non-psychophysical methods that are utilized. Low contrast acuity and Pelli-Robson charts are both efficient methods that can provide limited information regarding patient's ability to distinguish varied contrast levels (Bailey, 1982; Pelli et al., 1988). In pediatric populations, studies have assessed the feasibility and repeatability of using paper and tablet-based measures of contrast sensitivity (Anderson et al., 2023; Crossland et al., 2024). Tablet-based gaming methods of measuring contrast sensitivity in children include PopCSF and Manifold, which use an adaptive staircase to determine contrast threshold (Crossland et al., 2024). Spotchecks are a paper-based contrast sensitivity test in which patients are asked to identify spots of varying contrast within a box on the page to obtain an estimate of their CSF peak (Crossland et al., 2024).

Contrast Sensitivity in Amblyopia

A reduction in best corrected optotype acuity is the primary sign of amblyopia upon diagnosis, particularly when there is a deficit of two lines or more on visual acuity charts between eyes (Maurer & McKEE, 2018). However, studies have also shown amblyopic deficits in other visual functions such as contrast sensitivity and binocular vision (McKee et

al., 2003). Contrast sensitivity has been found to be reduced in anisometropic amblyopia particularly at high spatial frequencies (Bradley & Freeman, 1981; Mohammadi et al., 2019). This has been reported in central vision/in the foveal region in the amblyopic eye, where experimenters have used psychophysical measurements to compare contrast sensitivity at different spatial frequencies between the amblyopic and fellow eyes (Bradley & Freeman, 1981). An early study in individuals with anisometropic amblyopia utilized sinusoidal gratings within a central six degree circular patch to estimate a contrast sensitivity function for each subject (Bradley & Freeman, 1981). Amblyopic subjects demonstrated reduced sensitivity in the amblyopic eye at high spatial frequencies, with variable contrast sensitivity levels compared to the fellow eye at lower spatial frequencies (Bradley & Freeman, 1981). Rogers et al. conducted a study comparing contrast sensitivity in amblyopic and fellow eyes in children with anisometropic and strabismic amblyopia (Rogers et al., 1987). For both the strabismic and anisometropic amblyopic groups, this study found a linear relationship between visual acuity and contrast sensitivity: as acuity worsened, contrast sensitivity also decreased (Rogers et al., 1987). Peak contrast sensitivity was shifted to lower spatial frequencies in participants with more severe amblyopia (Rogers et al., 1987). This study also found that in treated amblyopes that had achieved 20/20 Snellen acuity in their amblyopic eye still had lingering contrast sensitivity deficits, providing an explanation as to why patients with treated amblyopia may still report weaker vision in their amblyopic eye following treatment (Rogers et al., 1987).

Few studies have evaluated contrast sensitivity deficits in amblyopia in the peripheral visual field, none of which were conducted in a pediatric population. A study by Thomas

evaluated contrast deficits in three strabismic amblyopic adults at fovea, 2°, 5°, and 10° of retinal eccentricity (Thomas, 1978). They reported that in strabismic amblyopia, contrast sensitivity deficits were primarily localized to central vision; and individuals with more severe visual acuity loss showed broader contrast deficits across the visual field (Thomas, 1978). Another study by Katz et al. measured contrast sensitivity in amblyopia at 0°, 10°, and 20° of retinal eccentricity in three individuals with amblyopia: one anisometropic, one strabismic, and one mixed strabismic and anisometropic amblyope (Katz et al., 1984). This study found contrast sensitivity deficits occurred primarily in central vision of the amblyopic eye, with decreasing deficits in the periphery (Katz et al., 1984). These studies evaluating contrast sensitivity in the peripheral visual field are limited in their sample size and were restricted to adults with amblyopia.

Amblyopia Treatments

Currently, the standard treatment for amblyopia is the optical correction of the amblyopic eye with spectacles or contact lenses, accompanied by patching the non-amblyopic eye, to encourage neural pathway development in the weaker eye (Falcone et al., 2021). While this treatment is effective, there is a high level of noncompliance with patching–particularly in young children, those with developmental delays, and those with severe amblyopia (Falcone et al., 2021).

To improve adherence with amblyopia treatment, dichoptic therapies have been developed as an alternative and to encourage binocularity by presenting balanced visual stimuli between the eyes to reduce suppression in the visual cortex (Falcone et al., 2021).

Dichoptic therapies use three main techniques to balance visual stimuli interocularly: anti-suppression therapy which reduces contrast in the image presented to the non-amblyopic eye, balanced binocular viewing which blurs images presented to the non-amblyopic eye, and interactive binocular treatment which presents different parts of a visual scene to each eye (Falcone et al., 2021). With the emergence of dichoptic therapies as a promising treatment for amblyopia, there has been increasing interest in assessment of interocular suppression across the visual field (Babu et al., 2017; Wiecek et al., 2024). Many of these emerging amblyopia treatment methods employ contrast balancing to dichoptically measure binocular combination, but little is known about how monocular contrast sensitivity deficits vary across the visual field in children before or during treatment, or about how non-uniform contrast sensitivity deficits may impact both binocular assessment and treatment. This has become increasingly important as different modalities of dichoptic therapies target various regions across the visual field (Wygnanski-Jaffe et al., 2023; Xiao et al., 2022). Luminopia is an FDA-approved head-mounted virtual reality dichoptic therapy which utilizes contrast modulation during content presentation (Xiao et al., 2022). Notably, Luminopia presents its dichoptic viewing by modulating contrast across different regions throughout the entire visual field (Xiao et al., 2022). Comparatively, CureSight is a dichoptic therapy which utilizes blur to modulate its dichoptic presentation, and this system utilizes a central blur scotoma to focus on treating central vision in the amblyopic eye (Wygnanski-Jaffe et al., 2023).

A more complete understanding of contrast deficits across the visual field in children with amblyopia can inform the development of emerging dichoptic therapies and may

improve efficacy by targeting treatment to most impacted regions of the visual field, while avoiding potential adverse effects of treating unaffected regions. Our current knowledge on contrast perception in amblyopia remains limited in children, largely due to small sample sizes and a focus on adults (Katz et al., 1984). This study aims to better characterize contrast sensitivity deficits in children and define the pattern of these deficits across the visual field.

Methods

Participant Characteristics

Participants aged 6-16 years were recruited at the Boston Children's Hospital Ophthalmology clinic. We recruited individuals with a diagnosis of anisometropic or strabismic amblyopia (n=10 and n=9 respectively), with a best-corrected interocular visual acuity difference of at least two lines, and a Snellen visual acuity of 20/25 or better in the fellow eye. This acuity difference was confirmed on the day of testing by an ophthalmologist, optometrist, ophthalmic technician, or certified orthoptist. Participants with strabismic amblyopia included all amblyopes that met the interocular acuity criteria with a constant deviation at distance and near with a magnitude of at least 5 prism diopters. Individuals with and without history of amblyopia treatment/strabismus surgery were included in this study. Participants with amblyopia were excluded if they presented with other ocular abnormalities, developmental delays, or other serious comorbidities. All control participants (n=11) had a best corrected Snellen visual acuity of 20/20 or better in each eye with no other ocular abnormalities. Summary demographic information is included in Table 1 and more detailed clinical information is listed in Supplementary Table 1. Written informed consent and assent were obtained from all participants and a legal guardian, and all experimental protocols and procedures were approved by the Institutional Review Board of Boston Children's Hospital and complied with the declaration of Helsinki.

Table 1: Subject Clinical Characteristics

Group	Total	Female	Mean Age	Mean Interocular LogMAR Acuity Between Eyes	Mean SER AE/NDE	Mean SER FE/DE
Anisometropic Amblyopia	10	5 (50%)	9.5	0.32	+2.00 D	+0.28 D
Strabismic Amblyopia	9	4 (44%)	10.3	0.32	+2.36 D	+1.83 D
Control	11	2 (18%)	11.5	0.00	-0.80 D	-0.75 D

Experimental Methodology

We used a similar psychophysical protocol to Wiecek et al., which evaluated binocular imbalance in amblyopia at central and peripheral retinal locations (Wiecek et al., 2024). Customized MatLab (R2018a) software was implemented using the Psychtoolbox (Version 3.0.14) interface to present stimuli on a gamma-corrected 42LM6200-UE LG monitor (57 x 32 deg). Participants were positioned 105 cm from the display, wearing corrective lenses as required for best corrected visual acuity. Ocular dominance in control participants was measured using the hand in hole test, as described in Lopes-Fereirra et al (Lopes-Ferreira et al., 2013). A Gazepoint eye tracker was used to monitor fixation, and the test eye was calibrated using a 9-point calibration system monocularly using Gazepoint software prior to the start of the experiment. Each participant's head position was aligned in a chinrest to maintain head position prior to eye tracker calibration and for the duration of the experiment. The untested eye was covered with an adhesive eye patch, and the first eye tested was randomized across all participants.

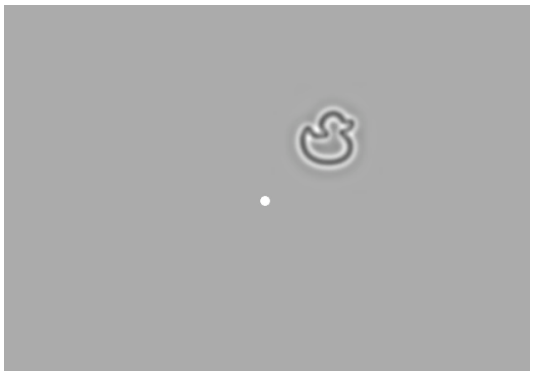
Monocular contrast sensitivity was assessed with a set of ten Auckland optotypes (Hamm et al., 2018). The optotypes were bandpass-filtered with a log cosine filter with a peak spatial frequency at five cycles per optotype (Solomon & Pelli, 1994) and a 1 octave bandwidth (see Figure 1C). Stimuli were presented monocularly with varying contrast levels against a mean luminance background (65.6 cd/m^2) at either central fixation, 4°, or 8° eccentricity. Stimuli at 4° and 8° eccentricity were presented at one of four angular locations (45°, 135°, 225°, and 315°) in a clockwise fashion. These angular locations were selected to minimize the impact of known meridional asymmetries in contrast sensitivity (Himmelberg et al., 2020). Optotype size and spatial frequency were logarithmically scaled to increase in size with eccentricity to account for cortical magnification and eccentricity-dependent contrast sensitivity loss in the periphery (4 cycles per degree (cpd) centrally, 2 cpd at 4°, and 1 cpd at 8° eccentricity) (Virsu et al., 1987; Virsu & Rovamo, 1979).

For each trial, participants were instructed to maintain central fixation while a randomly selected optotype was presented. The contrast of the optotype was gradually ramped on and off using a Gaussian temporal window (σ_t = 100 ms). This was followed by a 10AFC response screen with all ten Auckland optotypes (see Figure 1B). Participants were instructed to identify the optotype aloud while the experimenter subsequently clicked on the chosen optotype to record the response of the participant. An audio cue indicated whether participants selected the correct response. Stimuli were presented a total of 60 times centrally and 15 times at each location at each eccentricity (60 total trials per eccentricity). Contrast thresholds were estimated using the QUEST adaptive staircase (Watson & Pelli, 1983), with one independent staircase per location. QUEST parameters were set to 50% performance

criterion, slope $\beta = 3.5$, lapse rate $\delta = 0.01$, and guess rate $\gamma = 0.1$. Each staircase was initialized with a prior threshold estimate of 0.1 with prior standard deviation of 0.04. The algorithm selects the most informative contrast level to maximize information gained for each trial to estimate the contrast threshold at each location. The chosen contrast level on each trial, as well as the final threshold estimate, was calculated from the mean of the QUEST posterior probability density function. Figure 1 shows an example of the visual stimulus at 4 degrees eccentricity (A), response screen (B), and the 10 band-pass filtered Auckland optotypes that were used (C).

Throughout the experiment, subjects were instructed to maintain fixation on a central white dot (0.5°) and were reminded to remain doing so throughout testing. If a participant lost focus, they were given a short break, and the experimenter redirected them to the task after confirming they were ready to proceed. The experiment took approximately 25 minutes to test both eyes.

A



B

C

Figure 1: Visualization of screen during experimental trials. Panel A shows an example stimulus at 4 degrees eccentricity that the subject must identify while maintaining central fixation. Panel B depicts the 10 AFC option response screen for the subject to choose from to identify the stimulus following presentation. Panel C demonstrates the 10 band-pass filtered Auckland optotypes used in this experiment.

Statistical Analysis

Participant responses for each polar angle were collapsed within each eccentricity using MatLab software to obtain one contrast sensitivity threshold for 0°, 4° and 8° eccentricities. Contrast sensitivity threshold ranged between 0-1 (0 – 100% contrast), with higher thresholds corresponding to lower sensitivity. All statistical analysis was completed with RStudio Version 2024.04.2+764 using the following R packages: R.matlab, ggplot2, ggpubr, pracma, dplyr, plyr, tidyr, lme4, lmerTest, and ggsci. A linear mixed-effects model was used to evaluate the effects of group, eye, and eccentricity as fixed effects on contrast threshold, with interactions between group, eye, and eccentricity and a random effect for subjects. A Type III ANOVA was also performed using Satterthwaite's method for denominator degrees of freedom. We performed pairwise comparisons by computing estimated marginal means using the emmeans package in R and p-values were adjusted using the Tukey method to account for multiple comparisons.

For eye tracking analysis, post-hoc fixation stability was assessed by calculating the Bivariate Contour Ellipse Area (BCEA) of the gaze position samples (x- and y-coordinates that were converted to degrees of visual angle) recorded over the duration of the stimulus (Castet & Crossland, 2012). Prior to analysis, any invalid samples (i.e., off-screen values) were removed, and a linear mixed-effects model with a type 3 ANOVA was used to assess the

impact of group and eccentricity on fixation stability with interaction terms for eccentricity, group, and eye, with a random effect for subjects.

Results

Linear Mixed Effects Model

Statistical analysis was completed using a linear mixed effects model (Equation 1) and an ANOVA comparing group, eye, and eccentricity, with interaction terms for eccentricity, group, and eye, and a random effect for subject. Pairwise comparisons were performed using estimated marginal means using the Tukey HSD correction for multiple comparisons.

$$[1]\ Threshold \sim Group * Eye * Eccentricity + (1 \mid Subject))$$

This model revealed a significant main effect of group on contrast thresholds, with $F(2, 45.5)=3.17$, $p=0.05$, eye with $F(1,141)=20.66$, $p<0.001$, and eccentricity with $F(1,141)=8.10$, $p=0.005$. Interaction terms between eye and eccentricity were significant ($F(1,141)=6.61$, $p=0.01$) revealing that differences between amblyopic and fellow eye across all amblyopes changes with eccentricity. Interactions between group and eye were also significant ($F(2,141)=9.21$, $p<0.001$) indicating that differences between amblyopic and fellow eyes varied across groups. The interaction between group and eccentricity and the 3-way interaction between group, eye, and eccentricity were both not statistically significant ($F(2, 141) = 0.78$, $p = 0.46$ and $F(2, 141) = 1.98$, $p = 0.14$ respectively).

Pairwise Comparisons

Figure 2 compares contrast sensitivity thresholds for each group across eyes. When collapsed across all eccentricities, fellow eyes in the amblyopic groups had lower thresholds

compared to amblyopic eyes, with $p<0.001$ for the anisometropic group, $p=0.045$ for the strabismic group, and no significance found between eyes in the control group ($p=0.56$).

Within the anisometropic amblyopia group, deficits in the amblyopic eye were most pronounced in central vision. Direct comparison of the amblyopic and fellow eyes in this group revealed significantly lower contrast sensitivity in the amblyopic eye at 0° ($b=0.055$; $p<0.001$) and 4° ($b=0.034$; $p<0.001$), with no significant difference found at 8° of retinal eccentricity ($b=0.012$; $p=0.22$).

Similarly, in participants with strabismic amblyopia, the amblyopic eye contrast sensitivity deficits were more centrally localized. Interocular comparisons showed the amblyopic eye had significantly poorer contrast sensitivity at 0°($b=0.024$; $p=0.02$) and 4° ($b=0.013$; $p=0.045$) with no significant difference at 8°($b=0.002$; $p=0.8461$).

Among control participants, contrast sensitivity did not differ significantly between dominant and non-dominant eyes across all three eccentricities ($b=-0.003$; $p=0.78$, $b=-0.003$; $p=0.56$, and $b=-0.004$; $p=0.64$ for 0°, 4°, and 8° respectively). Additionally, no statistical significance was found across all control eyes when comparing between all eccentricities tested (all $t(141) = 0.68$, Tukey-adjusted $p = 0.78$). Because eccentricity was modeled as a continuous predictor, these contrasts reflect the same underlying linear effect. This consistency confirmed stimulus size and spatial frequency were scaled appropriately with eccentricity.

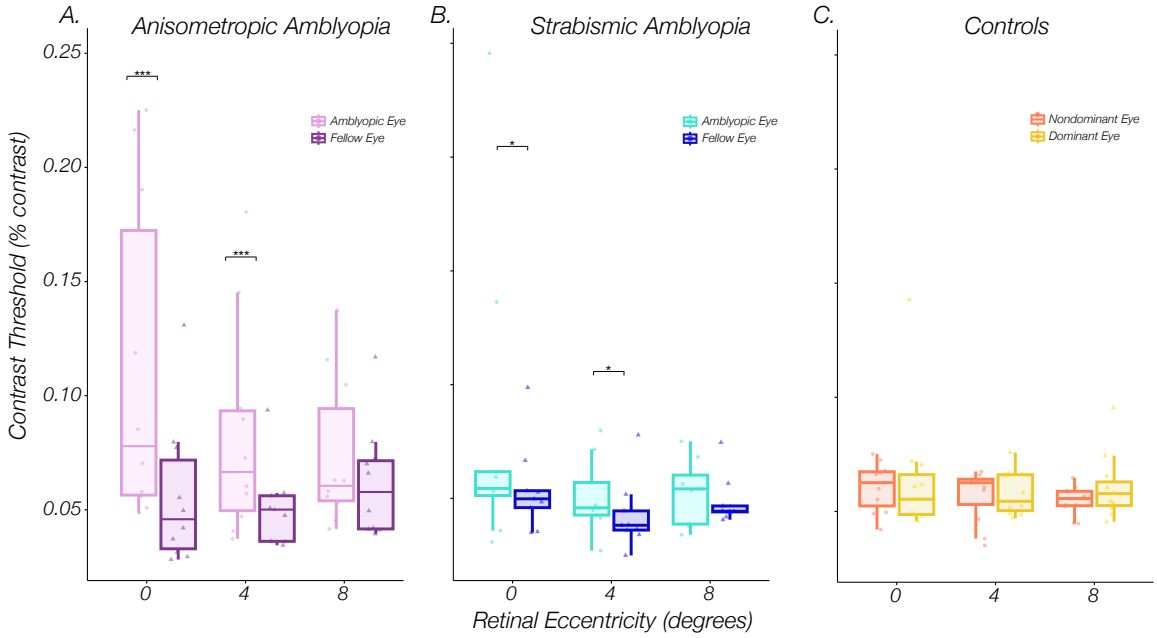


Figure 2: Comparison of contrast sensitivity threshold between eyes in each group tested. Panel A is the anisometropic amblyopia group, B is the strabismic amblyopia group, and C is the control group. The amblyopic eyes or non-dominant eyes of controls is represented by circles and the fellow eyes or dominant eyes of controls is represented by triangles. *** indicates $p < 0.001$, ** indicates $p < 0.01$.

We also compared contrast sensitivity performance of amblyopic eyes in the strabismic and anisometropic groups with the non-dominant eyes of the control group at each eccentricity, as shown in Figure 3. The amblyopic eye in the anisometropic group had significantly reduced contrast sensitivity as compared to the non-dominant eye of the control subjects at 0° and 4° of retinal eccentricity (b=0.054, p=0.002 and b=0.038, p=0.02) with no significance found in the periphery at 8° (b=0.022, p=0.20). The amblyopic eye of the strabismic group showed no significant difference when compared to the non-dominant eye of the control group at all three retinal eccentricities tested (b=-0.015; p=0.48, b=-0.004;

p=0.92, and b=0.006; p=0.86 for 0°, 4°, and 8°, respectively). However, when comparing the amblyopic eyes between strabismic and anisometropic groups at each retinal eccentricity, the anisometropic group showed significantly reduced contrast sensitivity at 0° and 4° of retinal eccentricity (b=0.038; p=0.01 and b=-0.033; p=0.01 respectively), despite no difference in interocular best-corrected visual acuity difference between amblyopic groups (Table 1).

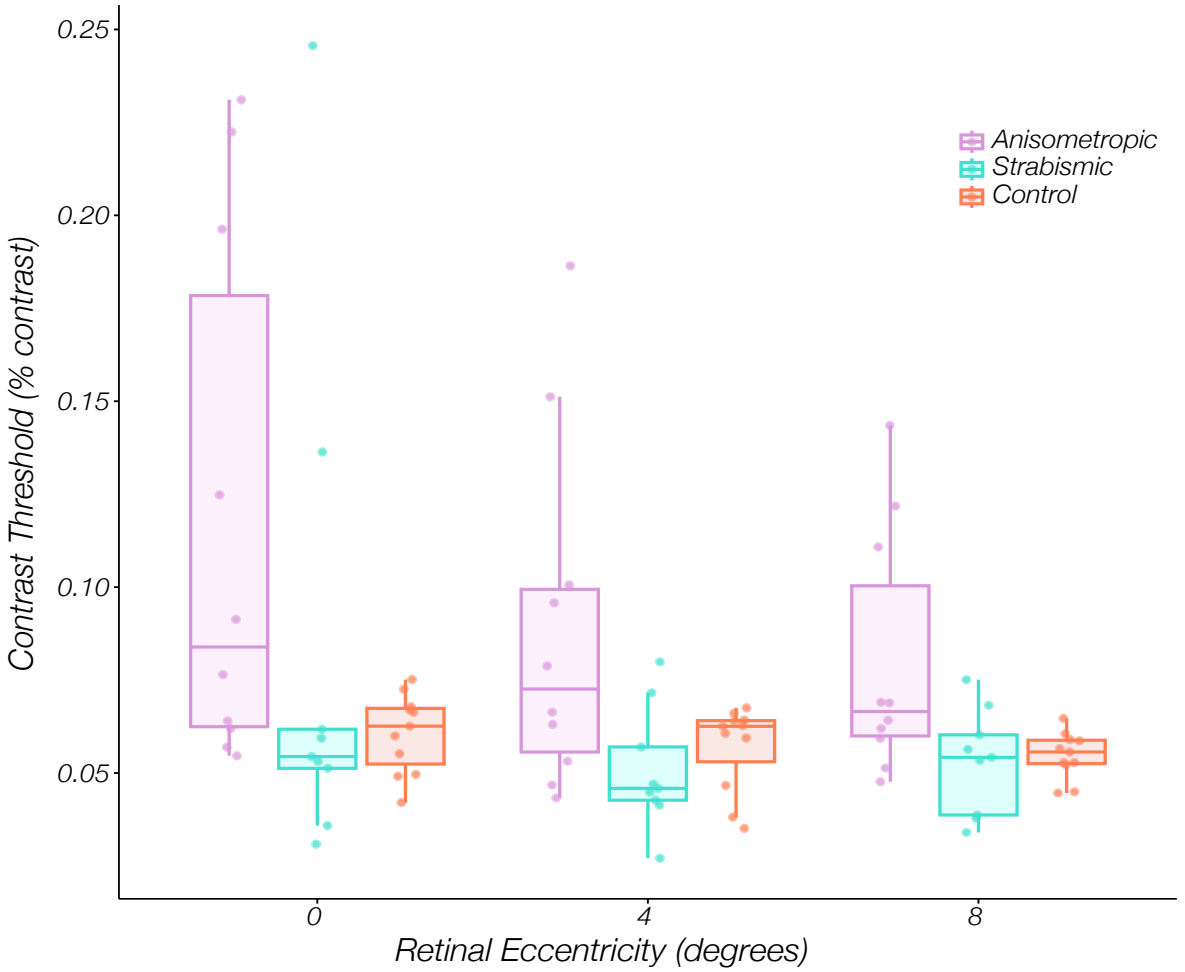


Figure 3: Comparison of contrast sensitivity threshold for each group tested in the amblyopic eye of anisometropic or strabismic amblyopes or the non-dominant eye in the control group across all eccentricities tested.

Monitoring Fixation

We monitored fixation throughout the experiment using a Gazepoint eye tracking device, and compared the BCEA for subjects in each group during the time frames in which stimuli were presented (Castet & Crossland, 2012). We used a linear mixed effects model and Type III ANOVA to evaluate the impact of group, eye, and eccentricity on fixation stability, as assessed by BCEA (Equation 2) with a random effect for subject.

$$[2]\ BCEA \sim Group * Eye * Eccentricity + (1|Subject)$$

Based on this model, there was no significance found between groups $F(2, 69.41) = 0.02$, $p = 0.983$, eye $F(1, 108.65) = 1.19$, $p = 0.279$, and eccentricity $F(1, 108.50) = 0.03$, $p = 0.853$. All interaction terms including both 2 and 3-way interactions were also not statistically significant, indicating that fixation stability did not differ across, groups, eyes or eccentricities tested and that fixation stability was consistent across all subjects and eyes that were tested.

Discussion

This study provides insight into the pattern of contrast sensitivity deficits in children with anisometropic and strabismic amblyopia and is the first to quantify peripheral contrast sensitivity in a cohort of children with amblyopia. Using age-friendly optotypes in a psychophysical experiment, we found that children with amblyopia primarily experience centralized contrast sensitivity loss in their amblyopic eye when comparing performance to their fellow eye or to the non-dominant eye of typically sighted controls. Additionally, these centralized amblyopic contrast sensitivity deficits were more pronounced in participants with anisometropic compared to strabismic amblyopia.

Contrast Sensitivity Across the Visual Field

In line with previous studies that evaluated contrast sensitivity across the visual field in adults with amblyopia, we found that children with amblyopia exhibit contrast sensitivity deficits primarily in central vision. For example, Katz et al. measured contrast sensitivity at 0°, 10°, and 20° of retinal eccentricity in individuals with amblyopia, and reported that the greatest loss occurred centrally with small stimuli. Performance improved in the periphery with larger stimuli, although deficits were still evident up to 20° of eccentricity (Katz et al., 1984). Pointer and Hess concluded similar findings in adults with strabismic and mixed amblyopia, in which the difference in contrast sensitivity between amblyopic and fellow eyes decreased with increased retinal eccentricity at mid-level spatial frequencies, and were dependent on type of strabismus, severity of amblyopia, and the spatial frequency of the stimulus (Hess & Pointer, 1985). In mild cases of strabismic amblyopia, peripheral contrast

sensitivity was largely spared, whereas severe cases had more extensive deficits in peripheral field (Hess & Pointer, 1985; Thomas, 1978). Several other studies have replicated this finding in adults with amblyopia and have compared the visual function of the amblyopic central retina to the peripheral retina of the non-amblyopic fellow eye (Earl Fred Miller, 1955; Katz et al., 1984; Levi et al., 1981; Pointer & Hess, 1989). Our observation of centrally localized contrast sensitivity loss follows a similar trend to other visual deficits in amblyopes such as binocular imbalance. Studies have demonstrated interocular suppression is most pronounced in central vision and becomes more balanced in the periphery (Babu et al., 2017; Wiecek et al., 2024).

Study Limitations

The primary limitation of this study was that contrast sensitivity was obtained for a single spatial frequency at each eccentricity, and our results must be interpreted in the context of known high spatial frequency selective deficits in amblyopia (Abrahamsson & Sjöstrand, 1988; Katz et al., 1984; Pang et al., 2019; Zele et al., 2007). Due to time constraints and the age of our subject population, it was not feasible to test a wider range of spatial frequencies with a sufficient number of trials at each eccentricity to obtain more of contrast sensitivity function at each eccentricity. Testing a larger range of spatial frequencies may have revealed a greater contrast sensitivity deficit in our strabismic group given that contrast sensitivity deficits in this group are highly spatial frequency dependent (Thomas, 1978). Future work to assess spatial frequency dependent contrast sensitivity across the visual field would be helpful to gain a more complete understanding of the CSF across multiple eccentricities. Work that can expand upon these data by measuring mid and far peripheral targets may

provide useful information on how contrast perception may impact visual function in children with amblyopia. Lastly, due to the limited testing time with pediatric participants, we were unable to estimate contrast thresholds separately for the nasal, temporal, superior and inferior visual fields in individuals with strabismic amblyopia. Future studies that compare these thresholds in strabismic participants with esotropic and exotropic deviations will better inform how direction of deviation may differentially bias sensitivity loss across the visual field meridians.

Future Directions of Amblyopia Treatments

The findings from this study contribute to our knowledge on the pattern of visual deficits in amblyopia in the context of emerging dichoptic therapies. While the current standard of treatment for amblyopia consists of optical correction and patching the fellow eye (a full-field intervention) high levels of noncompliance in many pediatric patients has driven the emergence of dichoptic therapies as an alternative treatment to improve treatment outcomes (Falcone et al., 2021; Pediatric Eye Disease Investigator Group, n.d.). Dichoptic therapies present balanced visual stimuli by displaying a stronger stimulus to the amblyopic eye to encourage visual development (Falcone et al., 2021). Several of the FDA-approved therapies target different regions of the visual field, with some focused on central vision, while others engage the entire visual field (Falcone et al., 2021). For example, Luminopia, delivered via a dichoptic VR headset, targets a large area of the visual field with a low contrast (15%) mask to the fellow eye (Xiao et al., 2022). Another modality, CureSight, targets its dichoptic presentation by modulating blur rather than contrast to central vision of the fellow eye (Wygnanski-Jaffe et al., 2023). Many of the large clinical trials on these

therapies include amblyopia from many different etiologies, with mixed results on differences in improvement between anisometropic vs strabismic groups (Halabi & Aldhahwani, 2025; Wygnanski-Jaffe et al., 2023; Xiao et al., 2022). Based on our findings that contrast sensitivity in amblyopic eyes is most reduced in central vision, it may be important to focus on modulating contrast-based dichoptic therapies to the central region of the visual field in amblyopic eyes. This central deficit was most pronounced in anisometropic amblyopes as compared to strabismic, and while targeting the central visual field may be valuable for all amblyopes, slightly different approaches may be required to treat different subtypes as we obtain more data.

Conclusion

Our data suggest centralized contrast sensitivity deficits in children with amblyopia are present across common amblyopia subtypes, and may be more pronounced in anisometropic as compared to strabismic amblyopia. These findings not only contribute to our characterization of visual deficits in amblyopia but may also be informative in the development of targeted treatment plans dependent on amblyopia etiology.

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Appendix

Supplementary Table S1. [Detailed Participant Information]

Age	Sex	Group	Interocular Acuity Difference	Amblyopic/NDE Eye Snellen Best-Corrected	Amblyopic/NDE SE RE	Treatment History	Strabismus Angle
15	M	Anisometropic Amblyopia OD	0.50 logMAR	20/70	+3.25 D	Glasses, Patching	N/A
7	M	Anisometropic Amblyopia OD	0.20 logMAR	20/30	+2.25 D	Glasses, Patching	N/A
10	M	Anisometropic Amblyopia OS	0.30 logMAR	20/40	+4.75 D	Luminopia	N/A
6	M	Anisometropic Amblyopia OS	0.20 logMAR	20/40	+0.75 D	Glasses, Patching	N/A
8	F	Anisometropic Amblyopia OS	0.20 logMAR	20/40	+0.50 D	Glasses, Patching	N/A
14	M	Anisometropic Amblyopia OD	0.20 logMAR	20/30	+1.00D	Glasses, Patching	N/A
6	F	Anisometropic Amblyopia OD	0.30 logMAR	20/40	+1.25 D	Glasses	N/A
12	F	Anisometropic Amblyopia OD	0.40 logMAR	20/50	-1.00 D	Glasses, patching	N/A
11	F	Anisometropic Amblyopia OD	0.40 logMAR	20/50	+3.00 D	Glasses	N/A
6	F	Anisometropic Amblyopia OS	0.50 logMAR	20/60	+4.50 D	Glasses, patching	N/A
16	M	Strabismic Amblyopia OS	0.20 logMAR	20/50	0.00 D	Strabismus Surgery	D: 16pd XT N: 16pd XT
14	F	Strabismic Amblyopia OD	0.40 logMAR	20/50	+4.75 D	Strabismus Surgery	D: 16pd ET N: 18pd ET

6	M	Strabismic Amblyopia OS	0.20 logMAR	20/40	0.00 D	Strabismus Surgery	D: 12pd ET N: 18pd ET
16	F	Strabismic Amblyopia OD	0.20 logMAR	20/30	+6.50 D	Strabismus surgery, Glasses	D: 14pd XT N: 18pd XT
7	F	Strabismic Amblyopia OS	0.30 logMAR	20/50	+1.00 D	Glasses	D: 20pd ET N: 20pd ET
8	M	Strabismic Amblyopia OS	0.30 logMAR	20/40	+4.50 D	Glasses, patching	D: 2pd ET N: 16pd ET
6	M	Strabismic Amblyopia OS	0.80 logMAR	20/125	+4.00 D	Glasses, patching	D: 35pd ET N: 50pd ET
6	M	Strabismic Amblyopia OD	0.20 logMAR	20/40	-1.50 D	Glasses, patching	D: 25pd XT N: 25pd XT
14	F	Strabismic Amblyopia OS	0.30 logMAR	20/40	+2.00 D	Strabismus Surgery	D: 18pd ET N: 18pd ET
15	F	Control	0.00 logMAR	20/20	0.00 D	N/A	N/A
14	M	Control	0.00 logMAR	20/20	+1.00 D	N/A	N/A
14	M	Control	0.00 logMAR	20/20	-0.75 D	N/A	N/A
9	M	Control	0.00 logMAR	20/20	0.00 D	N/A	N/A
11	M	Control	0.00 logMAR	20/20	-0.50 D	N/A	N/A
10	F	Control	0.00 logMAR	20/20	0.00 D	N/A	N/A
8	M	Control	0.00 logMAR	20/20	0.00 D	N/A	N/A
15	M	Control	0.00 logMAR	20/20	-0.75 D	N/A	N/A
6	M	Control	0.00 logMAR	20/20	+0.25 D	N/A	N/A
9	M	Control	0.00 logMAR	20/20	-5.50 D	N/A	N/A
11	M	Control	0.00 logMAR	20/20	-2.50 D	N/A	N/A