Contrast Sensitivity Across the Visual Field in Children With Amblyopia

Mariam Abdel-Kader,[1,2] Anna Kosovicheva,[3] Nikhil Chaudhry,[4] Ryan Gise,[2,5] Kimberley Chan,[2,5] Bharti R. Nihalani (Gangwani),[2,5] Eric Gaier,[2,5,6] Eric Moulton,[2,7] Ankoor Shah,[2,5] Peter Bex,[8] and Emily Wiecek[2,5]

[1]New England College of Optometry, Boston, Massachusetts, United States
[2]Department of Ophthalmology, Boston Children's Hospital, Boston, Massachusetts, United States
[3]Department of Psychology, University of Toronto, Toronto, Ontario, Canada
[4]Icahn School of Medicine at Mount Sinai, New York, New York, United States
[5]Department of Ophthalmology, Harvard Medical School, Boston, Massachusetts, United States
[6]Picower Institute for Learning and Memory, Massachusetts Institute of Technology, Cambridge, Massachusetts, United States
[7]Brain and Eye Pain Imaging Lab, Department of Anesthesiology, Critical Care and Pain Medicine, Boston Children's Hospital, Boston, Massachusetts, United States
[8]Department of Psychology, Northeastern University, Boston, Massachusetts, United States

Correspondence: Mariam Abdel-Kader, New England College of Optometry, 424 Beacon St., Boston, MA 02115, USA; mariam_abdel-kader@alumni.neco.edu.

Received: March 23, 2026
Accepted: September 18, 2026
Published: November 16, 2026

Citation: Abdel-Kader M, Kosovicheva A, Chaudhry N, et al. Contrast sensitivity across the visual field in children with amblyopia. *Invest Ophthalmol Vis Sci.* 2026;67(13):24. https://doi.org/10.1167/iovs.67.13.24

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.

Methods. Contrast sensitivity was measured at 0°, 4°, and 8° of retinal eccentricity in children ages 6 to 16 years with anisometropic amblyopia ($n = 10$) or 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°.

Conclusions. Our findings confirm that both anisometropic and strabismic amblyopic children 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.

Keywords: amblyopia, contrast sensitivity, psychophysics, pediatric vision, visual field

Amblyopia is the leading cause of uncorrectable vision loss in children, with a prevalence of 2% to 4% worldwide.[1,2] Amblyopia is clinically defined as an interocular best-corrected visual acuity (BCVA) difference of two or more lines of Snellen visual acuity.[3] In addition to reduced visual acuity, individuals with amblyopia experience many other visual deficits, such as a reduction in contrast sensitivity, binocular vision, and sensorimotor function.[4,5] These deficits have been primarily evaluated in central vision in adults, and much less is known about peripheral deficits in children with amblyopia.[1]

Early work by Bradley and Freeman[6] showed that adults with anisometropic amblyopia exhibit reduced contrast sensitivity in the amblyopic eye at high spatial frequencies, with variable contrast deficits at lower spatial frequencies. This has been supported by several studies that have found similar patterns of high spatial frequency loss in amblyopic eyes.[7–10] Rogers et al.[11] revealed a linear relationship between visual acuity and contrast sensitivity: As acuity worsened, contrast sensitivity also decreased. Peak contrast sensitivity was shifted to lower spatial frequencies in participants with more severe amblyopia.[11]

Copyright 2026 The Authors

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License.

Downloaded from iovs.arvojournals.org on 11/16/2026

Few studies have evaluated contrast sensitivity deficits in amblyopia in the peripheral visual field. A study by Thomas[12] evaluated contrast deficits in three strabismic amblyopic adults at fovea, 2°, 5°, and 10° of retinal eccentricity. 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.[12] Another study by Katz et al.[8] measured contrast sensitivity in amblyopia at 0°, 10°, and 20° of retinal eccentricity in three individuals with amblyopia and found contrast sensitivity deficits in central vision of the amblyopic eye, with decreasing deficits in the periphery.

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.[5,13] Many of these 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 different regions across the visual field.[14,15]

A more complete understanding of contrast deficits across the visual field in children with amblyopia can inform the development of emerging dichoptic therapies and improve efficacy by targeting treatment to the 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.[8] This study aimed to better characterize contrast sensitivity deficits in children and define the pattern of these deficits across the visual field.

Methods

Participant Characteristics

Participants ages 6 to 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 who 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 a 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 BCVA of 20/20 or better in each eye with no other ocular abnormalities. Summary demographic information is included in the Table, and more detailed clinical information is listed in Supplementary Table S1. 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 tenets of the Declaration of Helsinki.

Experimental Methodology

We used a psychophysical protocol similar to that of Wiecek et al.,[5] which evaluated binocular imbalance in amblyopia at central and peripheral retinal locations. Customized MATLAB R2018a software (MathWorks, Natick, MA, USA) was implemented using the Psychtoolbox 3.0.14 interface to present stimuli on a gamma-corrected 42LM6200-UE 8-bit monitor (57° × 32°; LG Corporation, Seoul, South Korea). Luminance resolution was increased (to 12 bits over a 4 × 4-pixel area) with a noisy-bit algorithm.[16] Participants were positioned 105 cm from the display and wore corrective lenses as required for BCVA. Ocular dominance in control participants was measured using the hand-in-hole test, as described in Lopes-Fereirra et al.[17] An eye tracker (Gazepoint, Vancouver, BC, Canada) was used to monitor fixation, and the test eye was calibrated using a nine-point calibration system monocularly with 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 10 Auckland Optotypes.[18] The optotypes were band-pass filtered with a log cosine filter with a peak spatial frequency at five cycles per optotype[19] and a 1-octave bandwidth (Fig. 1C). Stimuli were presented monocularly with varying contrast levels against a mean luminance background (65.6 cd/m^2) at central fixation or 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.[20] 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

Table. Subject Clinical Characteristics

Group	N	Female, n (%)	Mean Age (Y)	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

SER, spherical equivalent refraction; AE, amblyopic eye; NDE, non-dominant eye; FE, fellow eye; DE, dominant eye.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026

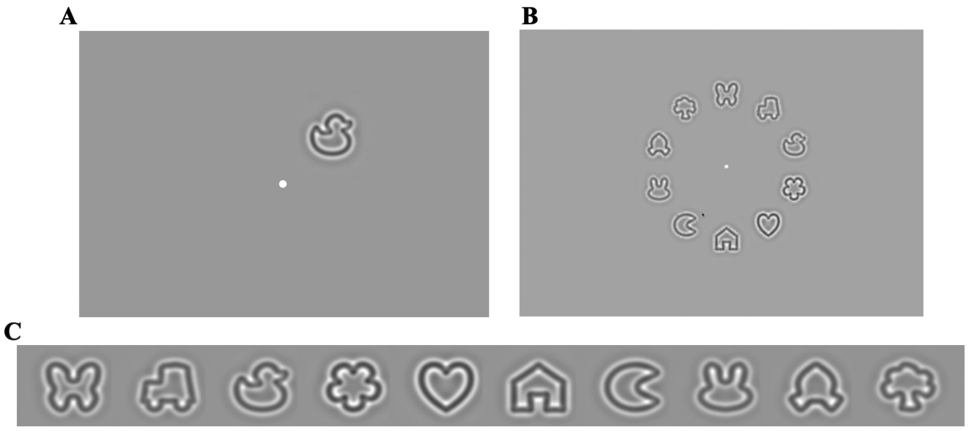


Figure 1. Visualization of screen during experimental trials. (**A**) An example stimulus at 4° eccentricity that the subject had to identify while maintaining central fixation. (**B**) The 10 alternative forced-choice option response screen for the subject to choose from to identify the stimulus following presentation. (**C**) The 10 bandpass-filtered Auckland Optotypes used in this experiment.

per degree [cpd] centrally, 2 cpd at 4°, and 1 cpd at 8° eccentricity).[21,22]

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 presentation of a response screen with all 10 optotypes (Fig. 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,[23] with one independent staircase per location. QUEST parameters were set to 50% performance criterion, slope β = 3.5, lapse rate δ = 0.01, and guess rate γ = 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 was calculated from the mean of the QUEST posterior probability density function. Figure 1 shows an example of the visual stimulus at 4° eccentricity (Fig. 1A), a response screen (Fig. 1B), and the 10 bandpass-filtered Auckland Optotypes that were used (Fig. 1C).

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

Statistical Analysis

Participant responses for each polar angle were collapsed within each eccentricity and fit with a cumulative Gaussian function using MATLAB software to obtain one contrast sensitivity threshold (μ) and slope (σ) estimate for 0°, 4° and 8° eccentricities. Contrast sensitivity threshold ranged between 0 and 1 (0%–100% contrast), with higher thresholds corresponding to lower sensitivity. Larger σ values indicated a shallower psychometric function and were considered a descriptive secondary measure that corresponds to the precision of the estimate. All statistical analysis was completed with RStudio 2024.04.2+764 (R Foundation for Statistical Computing, Vienna, Austria) 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 among group, eye, and eccentricity and a random effect for subjects. A type III ANOVA model using Satterthwaite's method was used to assess significance of the individual coefficients in the mixed-effects model. We performed pairwise comparisons to compare effects of cohort (typically sighted, anisometropic amblyopia, and strabismic amblyopia) and eye (fellow/dominant vs. amblyopic/non-dominant). This analysis computed the 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.[24] 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

Threshold was the main outcome measure and was evaluated across group, eye, and eccentricity. To evaluate threshold across group, eye, and eccentricity, statistical analysis was completed using a linear mixed-effects model (Equation 1). Pairwise comparisons were performed using estimated marginal means with Tukey honestly significant difference (HSD) correction for multiple comparisons.

$$Threshold \sim Group \times Eye \times Eccentricity + (1|Subject) \quad (1)$$

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026

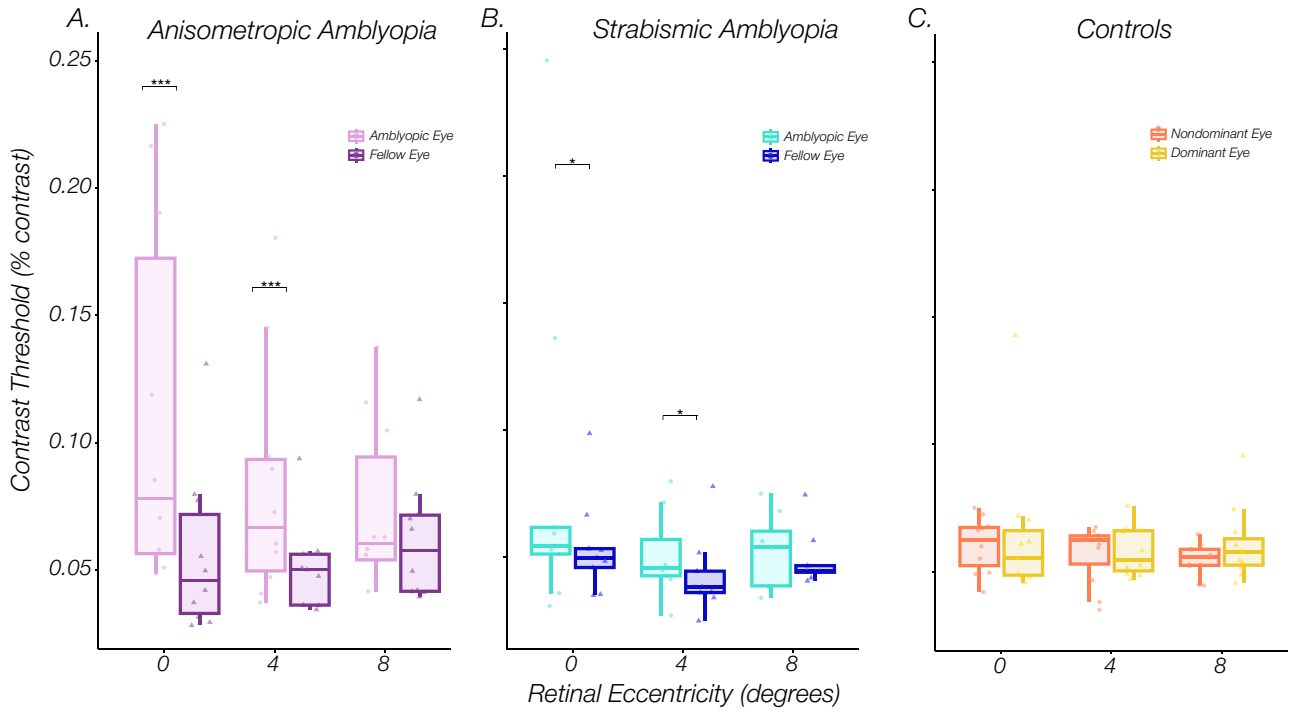


Figure 2. Comparison of contrast sensitivity threshold between eyes in each group tested. (**A**) Anisometropic amblyopia group. (**B**) Strabismic amblyopia group. (**C**) Control group. The amblyopic eyes or non-dominant eyes of controls are represented by *circles*, and the fellow eyes or dominant eyes of controls are represented by *triangles*. $^{**}P < 0.01$, $^{***}P < 0.001$.

This model revealed a significant main effect of group on contrast thresholds ($F_{2, 45.5} = 3.17$, $P = 0.05$), eye ($F_{1, 141} = 20.66$, $P < 0.001$), and eccentricity ($F_{1, 141} = 8.10$, $P = 0.005$). 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 change 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 three-way interaction among 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 ($P < 0.001$ for the anisometropic group; $P = 0.045$ for the strabismic group), and no significance was 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° ($\beta = 0.055$, $P < 0.001$) and 4° ($\beta = 0.034$, $P < 0.001$), with no significant difference found at 8° of retinal eccentricity ($\beta = 0.012$, $P = 0.22$).

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

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

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 ($\beta = 0.054$, $P = 0.002$ and $\beta = 0.038$, $P = 0.02$, respectively), with no significance found in the periphery at 8° ($\beta = 0.022$, $P = 0.20$). 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: 0° ($\beta = -0.015$, $P = 0.48$), 4° ($\beta = -0.004$, $P = 0.92$), and 8° ($\beta = 0.006$, $P = 0.86$). 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 ($\beta = 0.038$, $P = 0.01$ and $\beta = -0.033$, $P = 0.01$ respectively), despite no difference in interocular BCVA difference between amblyopic groups (Table).

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026

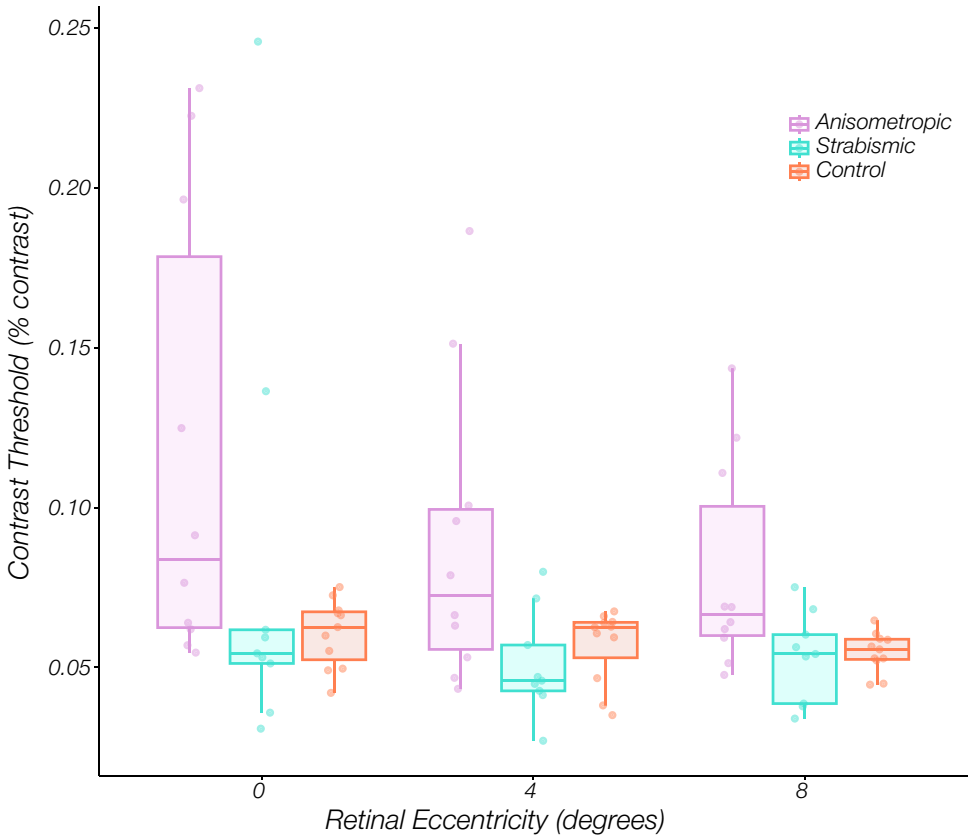


Figure 3. Comparison of contrast sensitivity threshold for each group tested in the amblyopic eye of anisometropic or strabismic amblyopes or the non-dominant eye in the control group across all eccentricities tested.

Psychometric function slope (σ) was calculated as a secondary measure, and values are reported in Supplementary Table S2. We found that σ correlated with threshold and was greater in the amblyopic as compared to the fellow eye at 0° and 4° in the anisometropic group. No consistent interocular differences in σ were observed at 8°, in the strabismic group, or between eyes of the typically sighted controls.

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.[24] 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.

$$BCEA \sim Group \times Eye \times Eccentricity + (1|Subject) \quad (2)$$

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 two- and three-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. BCEA values are shown in Supplementary Figure S1.

Discussion

This study provides insight into the pattern of contrast sensitivity deficits in children with anisometropic and strabismic amblyopia, and it is the first, to our knowledge, to quantify peripheral contrast sensitivity loss in a cohort of children with amblyopia. Using age-friendly optotypes, 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.

In line with previous studies that evaluated contrast sensitivity across the visual field in adults with amblyopia, we found that children with amblyopia exhibited contrast sensitivity deficits primarily in central vision. For example, Katz et al.[8] measured contrast sensitivity at 0°, 10°, and 20° of retinal eccentricity 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. Pointer and Hess[25] reported 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 the type of strabismus, severity of amblyopia, and spatial frequency of the stimulus. In mild cases of strabismic amblyopia, peripheral contrast sensitivity was largely

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026

spared, whereas severe cases had more extensive deficits in the peripheral field.[12,25] 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.[8,26–28] Although this study was performed monocularly, the observation of centrally localized contrast sensitivity loss follows a trend similar to other visual deficits found binocularly in amblyopes such as binocular imbalance, where deficits of interocular suppression were mostly centrally localized, becoming more balanced in the periphery.[5,13]

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.[7–10] Due to time constraints and the age of our subject population, it was not feasible to test a range of spatial frequencies with a sufficient number of trials at each eccentricity. We tested three spatial frequencies, one at each retinal eccentricity. These frequences were selected based on presumed peak contrast sensitivity at each eccentricity and accounting for known effects of cortical magnification.[21,22] Testing a larger range of spatial frequencies may have revealed a greater contrast sensitivity deficit in our strabismic group, as contrast sensitivity deficits in this group are highly spatial frequency dependent.[12] More specifically, we would anticipate that deficits in the periphery would have been more pronounced if we had tested higher spatial frequencies; however, this loss in peripheral sensitivity may have also been observed in typically sighted participants. Future work to assess spatial frequency dependent contrast sensitivity across the visual field would be helpful to gain a more complete understanding of the contrast sensitivity function across multiple eccentricities. Work that can expand upon these data by measuring mid- and far peripheral targets could provide useful information on how contrast perception may impact visual function in children with amblyopia. In this study, we were limited in testing far peripheral targets due to the age of our participants and their difficulty maintaining fixation while attending to far peripheral targets. Future studies that explore the far periphery in children with amblyopia may be beneficial to obtain a better understanding of the real-world impacts of functional deficits in the periphery. Finally, 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.

Due to issues with calibration and loss of attention in our pediatric subjects, eye tracking data were too noisy for detailed fixation stability analysis. We instead used eye tracking data as a measure of overall fixation compliance throughout the experiment to identify any systematic patterns in fixation of peripheral targets across groups and eccentricity conditions. Despite our analysis that showed no fixation difference between groups, research has shown that amblyopes do have elevated fixation instability due to the disruption of binocular experience early in life.[29] Due to the nature of this study, we did not have sensitive enough eye tracking data to detect microsaccadic differences, as fixation analysis was not one of our primary measures; rather, it was a measure of fixation compliance patterns across groups and eyes, in which we did not find any significant differences.

The findings from this study contribute to our knowledge on the pattern of visual deficits in amblyopia in the context of emerging dichoptic therapies. Although 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.[30,31] Dichoptic therapies present balanced visual stimuli by displaying a stronger stimulus to the amblyopic eye to encourage visual development.[31] Several of the U.S. Food and Drug Administration–approved therapies target different regions of the visual field, with some focused on central vision and others engaging the entire visual field.[31] For example, Luminopia (Cambridge, MA, USA), via a dichoptic virtual reality headset, targets a large area of the visual field with a low-contrast (15%) mask to the fellow eye.[14] Another modality, CureSight (NovaSight, Airport City, Israel), targets its dichoptic presentation by modulating blur rather than contrast to central vision of the fellow eye.[15] 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.[14,15,32] 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, although targeting the central visual field may be valuable for all amblyopes, slightly different approaches may be required to treat different subtypes.

Our data suggest that 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.

Acknowledgments

Supported by funding from a Knights Templar Eye Foundation Career Starter Grant and a Children's Hospital Ophthalmology Foundation Discovery Award (EW) and by grants from the National Institutes of Health (R01EY032162 to PB, F32 EY028814 to AK, and 5K23EY034212 to EW).

Disclosure: **M. Abdel-Kader**, None; **A. Kosovicheva**, None; **N. Chaudhry**, None; **R. Gise**, None; **K. Chan**, None; **B.R. Nihalani (Gangwani)**, None; **E. Gaier**, Luminopia (C, I, P), NeuroFieldz (C, I); **E. Moulton**, None; **A. Shah**, None; **P. Bex**, Luminopia (I), PerZeption (I, P); **E. Wiecek**, None

References

1. Levi DM. Rethinking amblyopia 2020. *Vision Res*. 2020;176:118–129.
2. Webber AL. The functional impact of amblyopia. *Clin Exp Optom*. 2018;101(4):443–450.
3. Maurer D, McKee SP. Classification and diversity of amblyopia. *Vis Neurosci*. 2018;35:E012.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026

4. McKee SP, Levi DM, Movshon JA. The pattern of visual deficits in amblyopia. *J Vis*. 2003;3(5):380–405.
5. Wiecek E, Kosovicheva A, Ahmed Z, et al. Peripheral binocular imbalance in anisometropic and strabismic amblyopia. *Invest Ophthalmol Vis Sci*. 2024;65(4):36.
6. Bradley A, Freeman RD. Contrast sensitivity in anisometropic amblyopia. *Invest Ophthalmol Vis Sci*. 1981;21(3):467–476.
7. Pang Y, Allen M, Robinson J, Frantz KA. Contrast sensitivity of amblyopic eyes in children with myopic anisometropia. *Clin Exp Optom*. 2019;102(1):57–62.
8. Katz LM, Levi DM, Bedell HE. Central and peripheral contrast sensitivity in amblyopia with varying field size. *Doc Ophthalmol*. 1984;58(4):351–373.
9. Abrahamsson M, Sjöstrand J. Contrast sensitivity and acuity relationship in strabismic and anisometropic amblyopia. *Br J Ophthalmol*. 1988;72(1):44–49.
10. Zele AJ, Pokorny J, Lee DY, Ireland D. Anisometropic amblyopia: spatial contrast sensitivity deficits in inferred magnocellular and parvocellular vision. *Invest Ophthalmol Vis Sci*. 2007;48(8):3622–3631.
11. Rogers GL, Bremer DL, Leguire LE. The contrast sensitivity function and childhood amblyopia. *Am J Ophthalmol*. 1987;104(1):64–68.
12. Thomas J. Normal and amblyopic contrast sensitivity function in central and peripheral retinas. *Invest Ophthalmol Vis Sci*. 1978;17(8):746–753.
13. Babu RJ, Clavagnier S, Bobier WR, Thompson B, Hess RF. Regional Extent of peripheral suppression in amblyopia. *Invest Ophthalmol Vis Sci*. 2017;58(4):2329–2340.
14. Xiao S, Angjeli E, Wu HC, et al. Randomized controlled trial of a dichoptic digital therapeutic for amblyopia. *Ophthalmology*. 2022;129(1):77–85.
15. Wygnanski-Jaffe T, Kushner BJ, Moshkovitz A, Belkin M, Yehezkel O, CureSight Pivotal Trial Group. An eye-tracking–based dichoptic home treatment for amblyopia: a multicenter randomized clinical trial. *Ophthalmology*. 2023;130(3):274–285.
16. Allard R, Faubert J. The noisy-bit method for digital displays: converting a 256 luminance resolution into a continuous resolution. *Behav Res Methods*. 2008;40(3):735–743.
17. Lopes-Ferreira D, Neves H, Queiros A, Faria-Ribeiro M, Peixoto-de-Matos SC, González-Méijome JM. Ocular dominance and visual function testing. *Biomed Res Int*. 2013;2013:238943.
18. Hamm LM, Yeoman JP, Anstice N, Dakin SC. The Auckland Optotypes: an open-access pictogram set for measuring recognition acuity. *J Vis*. 2018;18(3):13.
19. Solomon JA, Pelli DG. The visual filter mediating letter identification. *Nature*. 1994;369(6479):395–397.
20. Himmelberg MM, Winawer J, Carrasco M. Stimulus-dependent contrast sensitivity asymmetries around the visual field. *J Vis*. 2020;20(9):18.
21. Virsu V, Näsänen R, Osmoviita K. Cortical magnification and peripheral vision. *J Opt Soc Am A*. 1987;4(8):1568–1578.
22. Virsu V, Rovamo J. Visual resolution, contrast sensitivity, and the cortical magnification factor. *Exp Brain Res*. 1979;37(3):475–494.
23. Watson AB, Pelli DG. QUEST: a Bayesian adaptive psychometric method. *Percept Psychophys*. 1983;33(2):113–120.
24. Castet E, Crossland M. Quantifying eye stability during a fixation task: a review of definitions and methods. *Seeing Perceiving*. 2012;25(5):449–469.
25. Hess RF, Pointer JS. Differences in the neural basis of human amblyopia: the distribution of the anomaly across the visual field. *Vision Res*. 1985;25(11):1577–1594.
26. Miller EF, II. Investigation of the nature and cause of impaired acuity in amblyopia. *Am J Optom Arch Am Acad Optom*. 1955;32(1):10–29.
27. Levi DM, Harwerth RS, Pass AF, Venverloh J. Edge sensitive mechanisms in humans with abnormal visual experience. *Exp Brain Res*. 1981;43(3-4):270–280.
28. Pointer JS, Hess RF. The contrast sensitivity gradient across the human visual field: with emphasis on the low spatial frequency range. *Vision Res*. 1989;29(9):1133–1151.
29. Subramanian V, Jost RM, Birch EE. A quantitative study of fixation stability in amblyopia. *Invest Ophthalmol Vis Sci*. 2013;54(3):1998–2003.
30. Scheiman MM, Hertle RW, Beck RW, et al. Randomized trial of treatment of amblyopia in children aged 7 to 17 years. *Arch Ophthalmol*. 2005;123(4):437–447.
31. Falcone MM, Hunter DG, Gaier ED. Emerging therapies for amblyopia. *Semin Ophthalmol*. 2021;36(4):282–288.
32. Halabi MH, Aldhahwani B. The efficacy and safety of dichoptic therapy in comparison to patching in children with amblyopia: a systematic review and meta-analysis. *Acta Ophthalmol*. 2026;104(2):e139–e151.

Investigative Ophthalmology & Visual Science

Downloaded from iovs.arvojournals.org on 11/16/2026