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Fellow Eye Deficits in Amblyopia

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Abstract

Amblyopia is a neurodevelopmental disorder of the visual system, as a result of discordant visual experience during infancy or early childhood. Because amblyopia is typically defined as monocularly reduced visual acuity accompanied by one or more known amblyogenic factors, it is often assumed that the fellow eye is normal and sufficient for tasks like reading and eye-hand coordination. Recent scientific evidence of ocular motor, visual, and visuomotor deficits that are present with fellow eye monocular viewing and with binocular viewing calls this assumption into question. This clinical update reviews the research that has revealed fellow ocular motor and visual deficits and the effect that these deficits have on an amblyopic child's visuomotor and visuocognitive skills. We need to understand how to prevent and rehabilitate the effects of amblyopia not only on the nonpreferred eye but also on the fellow eye.

Keywords

Amblyopia; Fellow eye; Ocular motor; Motion perception; Motor skills

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Amblyopia is a neurodevelopmental disorder of the visual system, as a result of discordant visual experience during infancy or early childhood. The most common causes of amblyopia are strabismus and anisometropia. It is widely appreciated that stereoacuity is disrupted in amblyopia and that interocular suppression plays a central role in causing amblyopia. Yet, because amblyopia is typically defined as monocularly reduced visual acuity accompanied by one or more known amblyogenic factors, it is often assumed that the fellow eye is normal and sufficient for tasks like reading and eye-hand coordination. Recent scientific evidence of ocular motor, visual, and visuomotor deficits that are present with fellow eye monocular viewing and with binocular viewing that calls this assumption into question are reviewed here.

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Ocular Motor Function

Binocular vision relies on balanced visual input from the two eyes, as well as the ability of the eyes to make conjugate (saccades) and disconjugate (vergence) eye movements to fixate a target and maintain gaze. When binocular vision is disrupted early in life by strabismus, anisometropia, or amblyopia, a host of ocular motor deficits can occur, including fixation instability and abnormal saccades. In general, impaired ocular motor function is more pronounced when viewing with the non-preferred amblyopic eye, and when the visual acuity or stereoacuity deficit is severe.[1–4] Yet, ocular motor deficits are also present when viewing with the fellow eye, even in nonamblyopic individuals with strabismus.[5–7] Reduced visual acuity is present only in the amblyopic group, while both amblyopic and nonamblyopic individuals with strabismus or anisometropia have experienced binocularly discordant visual input during the critical period of visual maturation, suggesting that the fellow eye deficit is a consequence of discordant binocular experience, not the visual acuity deficit. Studies examining fellow eye deficits in amblyopic and nonamblyopic individuals with strabismus or anisometropia will be reviewed here.

Fixation Instability

During normal fixation, the eyes constantly make small, involuntary movements such as microsaccades, slow drifts, and tremors that aid in maintaining fixation and that prevent image fading.[8–11] Remarkably, binocularity in the normal visual system is not affected by these eye movements. When these involuntary eye movements become excessive, instability occurs in the ability to maintain fixation and visual function is disrupted. Fixation instability is a common consequence of strabismus, anisometropia, and amblyopia. Instability associated with these pediatric eye conditions is typically composed of abnormal fixational saccades, ocular drift, and fusion maldevelopment nystagmus.[1,4,8–10,16] Although amblyopia is associated with larger fixation instability, amblyopia is not a necessary condition for instability as it is also found in nonamblyopic strabismus.[1–6,13,17]

Research to date has typically focused on fixation instability under monocular viewing conditions with inconsistent results. Some studies have reported fellow eye stability with the amblyopic eye occluded.[2,3,16,17] Other studies report increased fellow eye instability compared with controls.[4,6,13,18,19] However, smaller angles of deviation and preserved binocular function may lessen instability.[13] Unpublished monocular viewing data from the cohort described by Kelly et al[20] also show amblyopic and nonamblyopic children with strabismus and/or anisometropia have larger fellow eye instability than controls (Figure 1).

Only a handful of studies have examined the fellow eye under binocular viewing conditions in strabismic and amblyopic adults, with inconsistent results. Gonzalez et al[17] found that the fellow eye was relatively stable in adults with strabismus and/or anisometropia compared with controls. In contrast, Ciuffreda et al[5] found fellow eye instability in amblyopic adults with strabismus; however, instability was absent in a small group of three amblyopic adults without strabismus.

Lastly, vergence instability (i.e., variability in ocular alignment over time) has been assessed. During fixation, ocular alignment varies due to microsaccades and drift. While

microsaccades are typically conjugate, the horizontal component of drift has less conjugacy, and sometimes has a “wave-like” appearance with alternating periods of convergence and divergence.[8] Vergence instability has been reported while viewing with two eyes in adults with large angle exotropia, suggesting an association of suppression with disconjugate eye movements.[6] Recently, Kelly et al[20] reported increased fixation instability and vergence instability, regardless of current amblyopia, in strabismic and anisometropic children compared with controls (Figure 2). Fellow eye instability was not associated with amblyopic eye visual acuity or stereoacuity. However, vergence instability was related to worse stereoacuity and to a larger suppression scotoma as measured by Worth 4-dot at 7 different distances[21,22] (Figure 2). When interocular suppression was artificially reduced via contrast-rebalancing, fellow eye instability was significantly improved in strabismic amblyopes.[22]

The presence of ocular motor deficits during binocular viewing indicates a fellow eye impairment since the amblyopic eye is likely suppressed, with the fixating fellow eye driving vision. Fellow eye instability during monocular and binocular viewing suggests that discordant binocular visual experience, even in the absence of amblyopia, interferes with the development of ocular motor control. Instability points to a common neural mechanism that controls the stability of both the fellow and amblyopic eye. Further, the presence of vergence instability in amblyopia may limit the potential for recovery of binocular vision.

Abnormal Saccades

Saccades are small, high velocity conjugate eye movements that direct the fovea onto a target of interest. The distinction between saccades and fixational microsaccades is somewhat artificial. Fixational eye movements include microsaccades, which are small involuntary saccades that cannot be differentiated from saccades according to their magnitude or any physical characteristic.[23] Given the fixation instability of the fellow eye in strabismic and amblyopic individuals, we might also expect abnormalities in fellow eye saccades.

A limited number of studies exist that compare fellow eye saccades with the amblyopic eye occluded to controls, as most compare amblyopic to fellow eye viewing. While one study reported similar fellow eye saccadic latencies as controls,[24] another found latencies to be delayed and more variable.[7] During binocular viewing, saccadic latency is also delayed and more variable in amblyopic adults.[7,25] Further, the typical pattern of faster saccades during binocular viewing compared with monocular viewing (i.e., binocular advantage) is absent in amblyopic adults.[25,26] Lastly, binocular coordination is impaired such that saccades are disconjugate.[27,28]

Abnormal saccades in amblyopic individuals relative to controls can be exacerbated by more severe amblyopia and by a lack of stereopsis.[24,25–27] Latencies of saccades are longer in visually normal children compared with adults, suggesting an immaturity of the eye movement system during development and susceptibility to dysfunction as a result of early abnormal visual experience.[29] The presence of abnormal fellow eye saccades in amblyopia points to a disruption in ocular motor control, and provides further evidence that binocular discordant input impacts the development of visual pathways associated with the fellow eye.

Motion Perception

Although the subnormal or nil stereoacuity that typically accompanies amblyopia is the most studied binocular outcome measure for amblyopia treatment, measurement of stereoacuity does not allow the effects of discordant binocular visual experience on the amblyopic and fellow eyes to be examined separately. Minor monocular fellow eye deficits have been reported for several aspects of visual function, and stronger fellow eye deficits have been reported for motion perception tasks that rely on binocular cortical regions.[30] In non-human primates, motion-sensitive brain regions such as the middle temporal visual area (MT) contain neurons that respond to motion presented to either eye.[31–34] The human homolog (MT+ or V5) is activated by several types of motion stimuli that require integration of local motion signals.[35,36] Early discordant visual experience may disrupt the development of this brain region and lead to impaired motion processing in the fellow eye as well as the amblyopic eye.

Impaired monocular motion processing has been demonstrated using dynamic random-dot patterns composed of signal dots that move coherently in one direction mixed with noise dots that move randomly. Performance is measured by determining the smallest percentage of signal dots necessary to accurately determine the coherent motion direction (global motion task;[37] Figure 3 left; e-supplement video) or the orientation of a shape created by signal dots moving in opposite directions inside and outside the shape (motion-defined form task;[38] Figure 3 **right;** e-supplement video).

Fellow eye global motion deficits have been reported in several studies of amblyopic children and adults,[39–41] but there also are several studies that report few or no fellow eye deficits.[42,43] (Figure 4) The discrepancies in results of various global motion studies appear to depend on the specific spatiotemporal parameters of the visual stimuli, requiring prior identification of the parameters that revealed an amblyopic eye deficit in order to observe the fellow eye deficit.[41]

Up to 40% of amblyopic children have been reported to have a fellow eye deficit in perceiving motion-defined form.[43–45] Unlike global motion, fellow eye motion-defined form deficits are reported consistently across studies of amblyopic children and adults, despite some variations in visual stimuli. (Figure 4) Fellow eye motion-defined form deficits have been reported to be resistant to rehabilitation by patching,[44] but were responsive to binocular amblyopia treatment. [46]

Reading

Reading is a vision-reliant ability fundamental to academic achievement. Saccades during reading allow us to move forward and regress backward through text. Fixations, or pauses, occur during reading as decoding of phonemes occurs. Increased fixation duration or abnormal saccades could result in slower reading that, in turn, could be detrimental to academic performance and learning. Most studies of reading by amblyopic children have been conducted with monocular viewing, comparing amblyopic eye versus fellow eye reading performance to determine whether the treated amblyopic eye provides a useful

"spare eye" in the event that the fellow eye can no longer be used for reading.[47–49] More recently, monocular reading speed for the fellow eye has been studied; the fellow eye has decreased reading speed in strabismic and anisometropic amblyopia relative to monocular reading in visually normal children.[48,50,51] Slower monocular reading with the fellow eye is associated with an abnormally increased number of saccades.[51]

With binocular viewing by amblyopic children and adults, there are several reports of slow reading of sentences and words, and slow reading of paragraphs of text projected on a distant screen.[48–50] More recently, natural, binocular silent reading of age-appropriate print paragraphs at habitual reading distance has been assessed in amblyopic children.[52,53]Amblyopic children with strabismus or anisometropia read slowly compared with controls and nonamblyopic strabismic children.[52,53] Unlike the earlier studies, this more recent work clearly identified that amblyopia alone was sufficient to cause slow reading; reading performance was not related to severity of amblyopia, nor to diagnosis (strabismic, anisometropic). Further, data from Kelly et al[51] reported similar reading rate and number of forward saccades for binocular reading versus fellow eye reading in amblyopic children, suggesting that rather than binocular inhibition (i.e., amblyopic eye interference during binocular reading), slow reading is due to a fellow eye deficit even when reading with two eyes (Figure 5).

Importantly, reading comprehension did not differ significantly between amblyopic children and controls, nor did fixation duration or number of regressive eye movements.[52–54] Taken together, these findings suggest that amblyopic children did not read slowly because they had dyslexia or a learning disability. In fact, amblyopic children show low (<5%) prevalence for specific reading disability on the Wide Range Achievement Test II (WRAT II), similar to control children.[55] Slow reading likely results from the fixation instability associated with amblyopia and binocular dysfunction and the resultant vergence instability.[19] A larger number of forward saccades occurs (per 100 words) in amblyopic children during reading compared with nonamblyopic strabismic children and controls.[52] Moreover, there is a strong relationship between slow reading and fixation instability of the fellow eye in children with anisometropic amblyopia.[53]

Because perceived scholastic competence is a key determinant of self-esteem in school-aged children, slow reading may be expected to influence a child's self-perception. A recent study reported that school-age children with amblyopia had significantly lower scores than control children for self-perception of scholastic competence.[56] Moreover, their lower self-perception of scholastic competence was associated with a slower reading speed. [56]

Visually-Guided Motor Skills

Coordination between eye movements and hand movements is essential in object manipulation. Normal binocular vision during childhood provides important sensory input for optimal development of eye-hand coordination. Reduced visual acuity, impaired depth perception, and abnormal ocular motor function typically found with binocularly discordant visual experience may have significant effects on the development of visually-guided motor skills.

A limited number of studies have investigated fine motor ability in children and adults with strabismus and/or anisomteropia during fellow eye viewing. These studies show deficits in the speed and accuracy of manual dexterity tasks.[57–61] One study found that strabismic and anisometropic children ages 5–11 years spent almost double the amount of time in the final approach to objects during a reach-to-grasp task compared with controls, and that they made 1.5 to 3 times more errors.[57] These findings were echoed in another study, but only for younger children (5–6 years old) and not with older children (7–9 years old).[58]

Similar to fellow eye viewing, adults and children with amblyopia due to strabismus and/or anisometropia are slow to reach and grasp objects, and to place pegs in holes and thread beads under binocular viewing conditions.[59,60,62,63] Kelly et al[64] have also reported deficits in manual dexterity tasks (unimanual, bimanual, drawing trail) of the Movement Assessment Battery for Children (MABC-2) in amblyopic and nonamblyopic children age 3–12 years treated for strabismus and/or anisometropia. (Figure 6) In a recent publication, Kelly et al[65] reported that school-age children with strabismus and/or amblyopia required 28% more time than controls to transfer answers from a test booklet to a multiple-choice answer sheet, which could translate into poorer academic performance. Fine motor skills improved with binocular, contrast re-balanced game treatment,[66] and with improved stereo acuity in a small cohort of children who patched.[58] Motor skills as an amblyopia treatment outcome measure may be beneficial.

Impaired kinematics appears to be behind the fine motor deficits found in amblyopic children and adults, both for fellow eye viewing and for binocular viewing. Reach planning and execution are slower, and the precision of reaching and grasping is reduced, although compensatory strategies may improve performance with age.[26,57,58,60,67,68] During reaching, amblyopic adults have reduced peak acceleration and prolonged acceleration indicating their ability to use vision to plan movements is impaired.[69] Amblyopic children also show longer movement times, lower reach velocity, and a longer duration of contact prior to lifting an object.[60] Temporal eye-hand coordination in amblyopic adults is associated with increased corrective saccades, a compensatory strategy to increase reach accuracy; particularly among adults with nil stereoacuity.[70,71] Visuomotor deficits appear more closely associated with stereo deficits than with the severity of visual acuity deficit.[59,61,68] This is supported by the finding of fine motor deficits in nonamblyopic individuals with deficient stereoacuity or strabismus,[61] and of normal reach-related saccades with monocular in normal controls.[26,62]

In turn, impaired motor skills may affect self-perception of scholastic and social competence. Using the Self-Perception Scale for Children, Birch et al[56] recently reported that children with amblyopia had significantly lower scores than control children for social and athletic competence domains. Lower self-perception of social and athletic competence was associated with worse aiming and catching performance on a standardized test of motor skills, the Movement ABC-2.

What Causes Fellow Eye Deficits in Amblyopia

Persistence of fellow eye deficits even when the amblyopic eye is occluded for monocular testing suggests that fellow eye deficits cannot be attributed to inhibition/suppression by the amblyopic eye. While binocular inhibition, i.e., seeing better with the fellow eye than with

both eyes, is related to slow reading in age-related macular degeneration,[72] binocular reading speed does not differ from fellow eye reading speed in amblyopic children (i.e., no binocular inhibition).[51]

Instead, fellow eye deficits are likely a result of binocular dysfunction. In this context, a valuable model of severe disruption of binocularity is early monocular enucleation, since only the one eye remains. The other eye cannot contribute to visual performance nor can it suppress or otherwise interfere with the visual performance of the remaining eye. Adults who experience enucleation before 5 years of age due to retinoblastoma have many of the same motion perception deficits as found in the fellow eyes of amblyopic individuals, including speed discrimination, motion-defined form perception, and the perception of motion in depth.[73–77] Further, enucleated adults display a different pattern of ocular motor function compared to binocularly intact controls; asymmetrical optokinetic nystagmus (OKN) favoring nasalward motion[78] but have normal saccades and stable fixation.[79,80] The complete deafferentation of visual input from one eye in enucleation results in the removal of binocular interactions and competition during visual development, and thus may be different than the binocularly discordant information present in strabismic and anisometropic amblyopia.

Additional support for the central role of binocular dysfunction in fellow eye deficits comes from our recent research on motion-defined form perception. Amblyopic children who have had evidence of abnormal binocularity, including nil stereo or only peripheral fusion, were significantly more likely to have a fellow eye deficit (Figure 7).

Fellow Eye Deficits and Amblyopia Treatment

Fellow eye deficits prompt us to reconsider whether amblyopia is adequately characterized as a monocular visual acuity deficit, and whether treatment by occlusion of the fellow eye, or occlusion coupled with monocular perceptual learning is optimal. Fellow eye motion-defined form perception deficits have been reported to be resistant to rehabilitation by patching[44] but responsive to binocular amblyopia treatment.[46] (Figure 7). More effective treatments for amblyopia may target suppression, by requiring integration of information between the two eyes to play dichoptic games or watch dichoptic videos. Like occlusion therapy, binocular treatment results reported to date are generally positive for young children [81–87] but less effective for older children due to poor compliance and/or intractable residual amblyopia.[88–90]

Conclusion

For many years, the fellow eye was considered to be normal and study after study assessed the child’s ability to see, read, or perform visuomotor tasks using the amblyopic eye to determine whether it was a useful spare eye. The overarching goal was to evaluate whether the monetary and personal cost of years of occlusion therapy were worth the benefit of a few lines gain in visual acuity. Now the paradigm has shifted; we have discovered that the fellow eye also has visual deficits and that these deficits affect an amblyopic child’s visuomotor and

visuocognitive skills. We need to understand how to prevent and rehabilitate the effects of amblyopia not only on the nonpreferred eye but also on the fellow eye.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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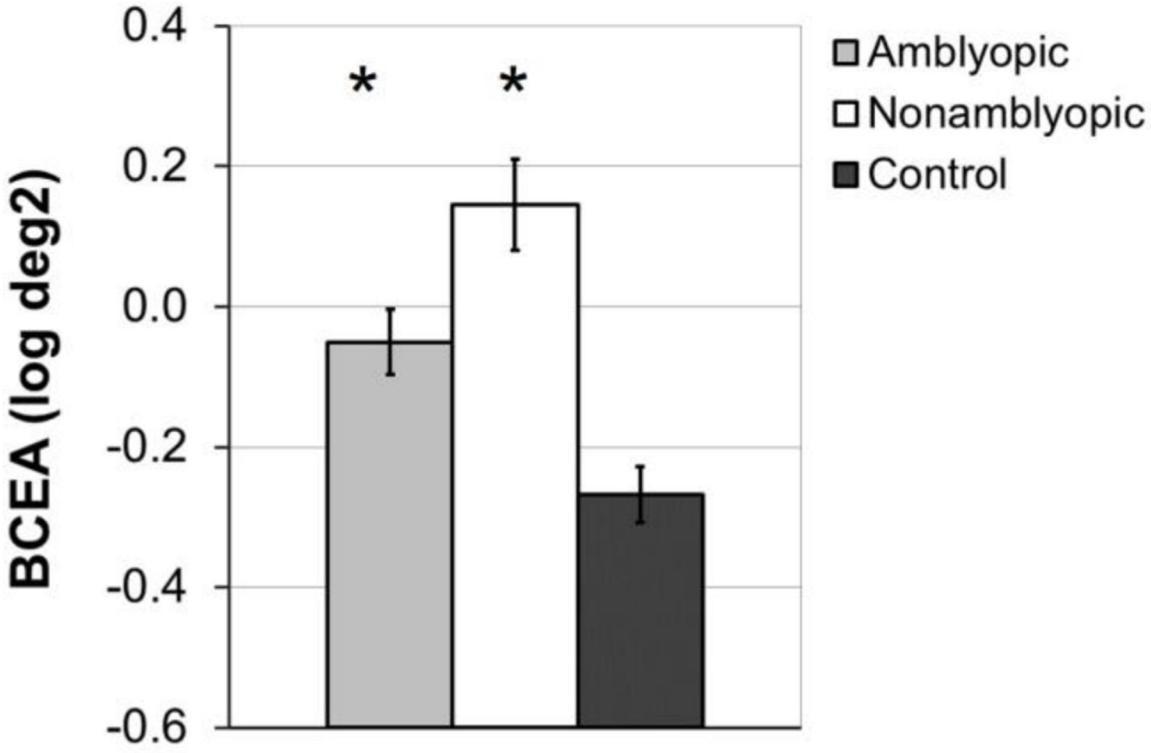

Figure 1.

Unpublished monocular viewing data from the cohort described in Kelly et al[19] showing larger mean fellow eye instability for 85 amblyopic children (light gray bars) and 55 nonamblyopic children with strabismus or anisometropia (white bars), compared with 43 normal control children (dark grey bars). Error bars represent ±SE.

*significantly different than controls (p<0.01).

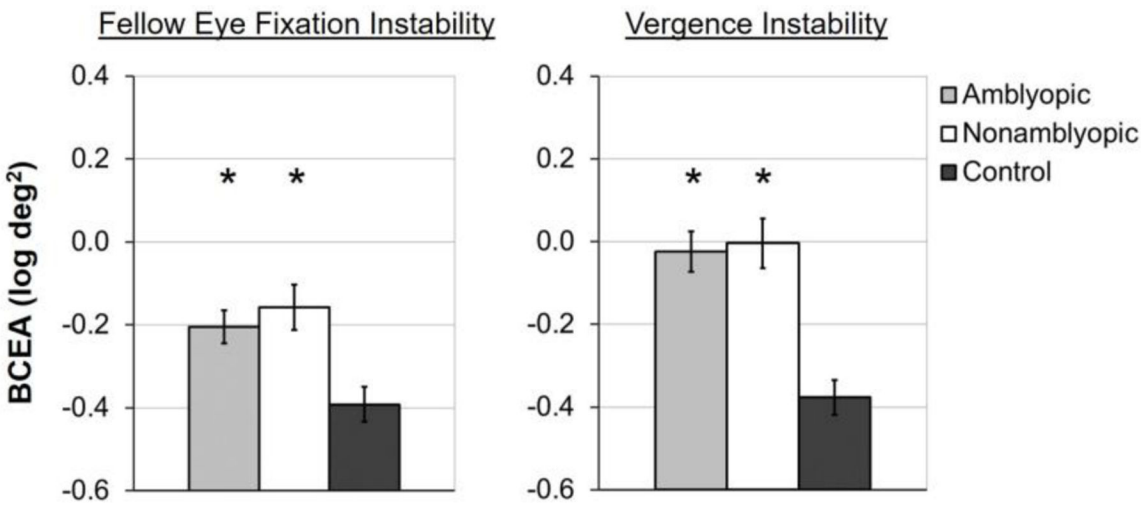

Figure 2.

Mean fellow eye instability and vergence instability during binocular viewing for 98 amblyopic children (light gray bars), 62 nonamblyopic children with strabismus or anisometropia (white bars), and 46 normal control children (dark grey bars). Amblyopic and nonamblyopic children exhibited larger fixation and vergence instability compared to controls. Error bars represent ± SE. *significantly different than controls (p<0.01).

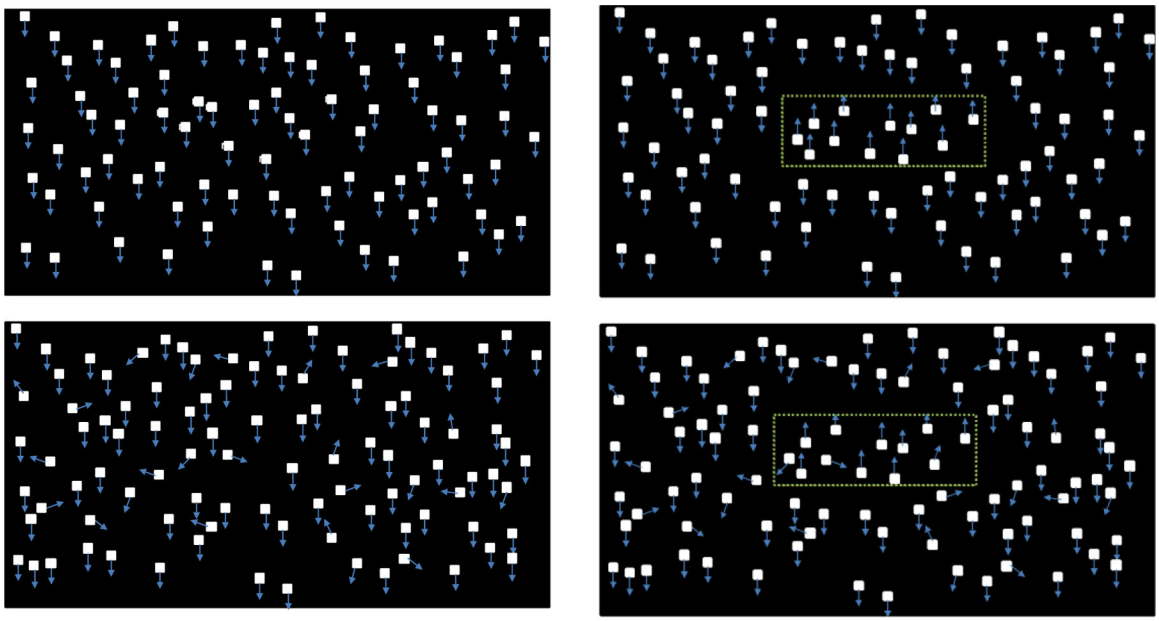

Figure 3.

Left: global motion stimuli with 100% coherence (*top*) and 75% coherence (*bottom*). *Right*: motion-defined form stimuli with 100% coherence (top) and 75% coherence (bottom). Arrows were added to illustrate the direction of motion of each dot. The dotted-line boxes were added to illustrate the target shape. Neither the arrows nor the boxes were present in the actual stimulus displays.

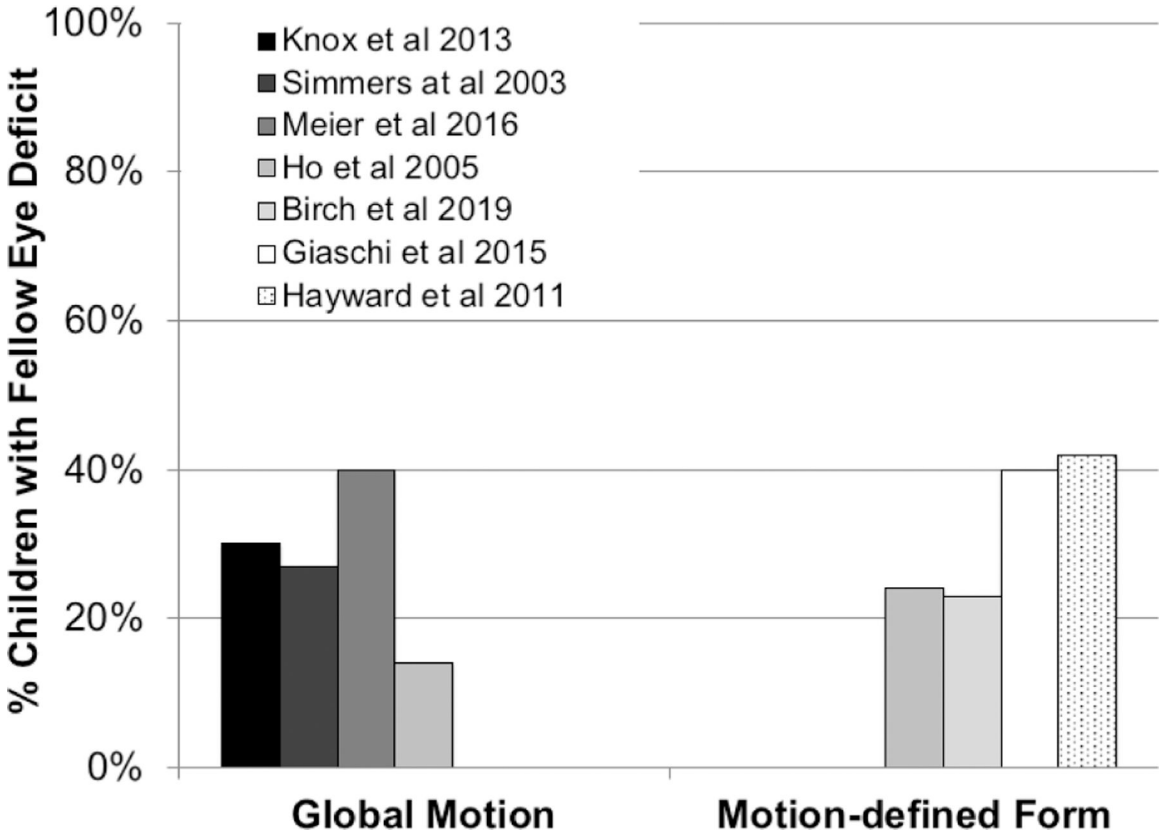

Figure 4.

% of children with amblyopia who had a deficit in perception of global motion direction (*left*) or motion-defined form orientation (*right*) when viewing with their fellow eye, compared with controls. Thresholds for both tasks were measured by the minimum percentage of coherently moving signal dots required for accurate discrimination.

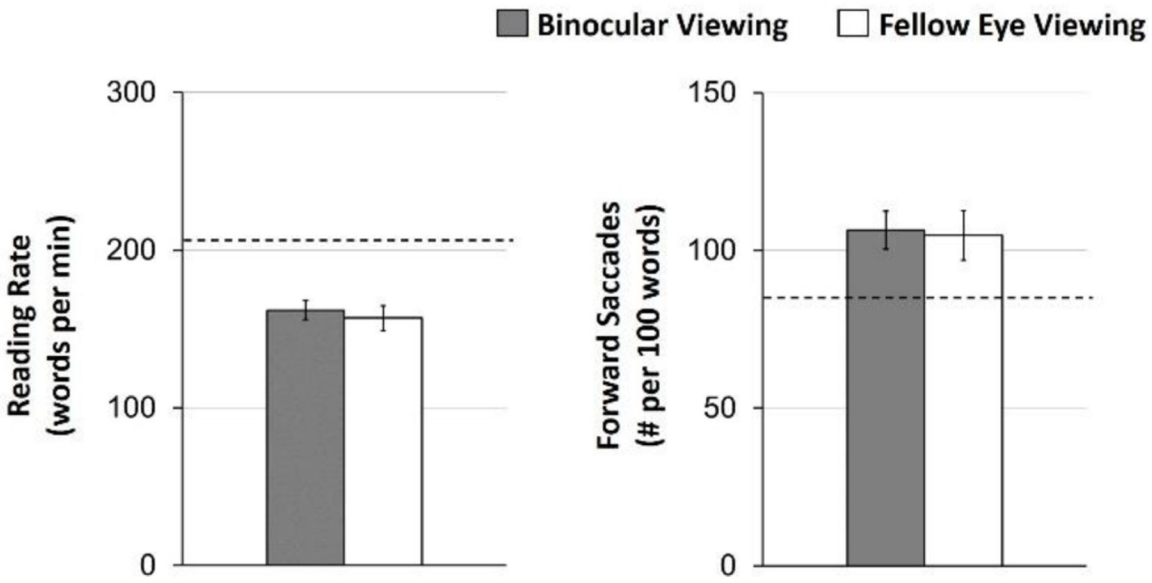

Figure 5.

Mean reading rate (words per minute) and number of forward saccades (per 100 words) assessed with the Readalyzer ® during binocular viewing (grey bars) and fellow eye viewing (white bars) for 49 amblyopic children No differences were found (ps≥0.50). Error bars represent ± SE. The dashed line represents previously published control data for binocular reading.[52]

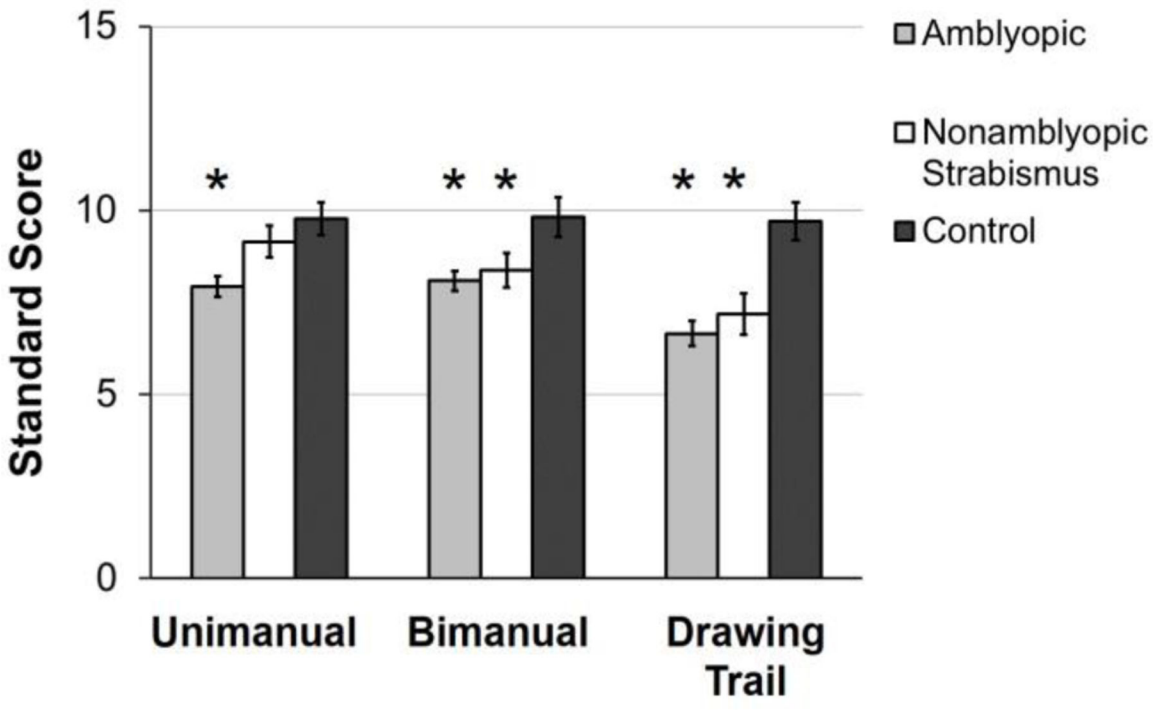

Figure 6.

Mean standard scores for manual dexterity tasks completed during binocular viewing for 129 amblyopic children (light gray bars), 47 nonamblyopic children with strabismus (white bars), and 40 normal control children (dark grey bars). Amblyopic children had lower scores for all three tasks compared to controls. Nonamblyopic children with strabismus had lower scores on the bimanual and drawing trail tasks compared with controls. Error bars represent ± SE.

*significantly different than controls (p<0.01).

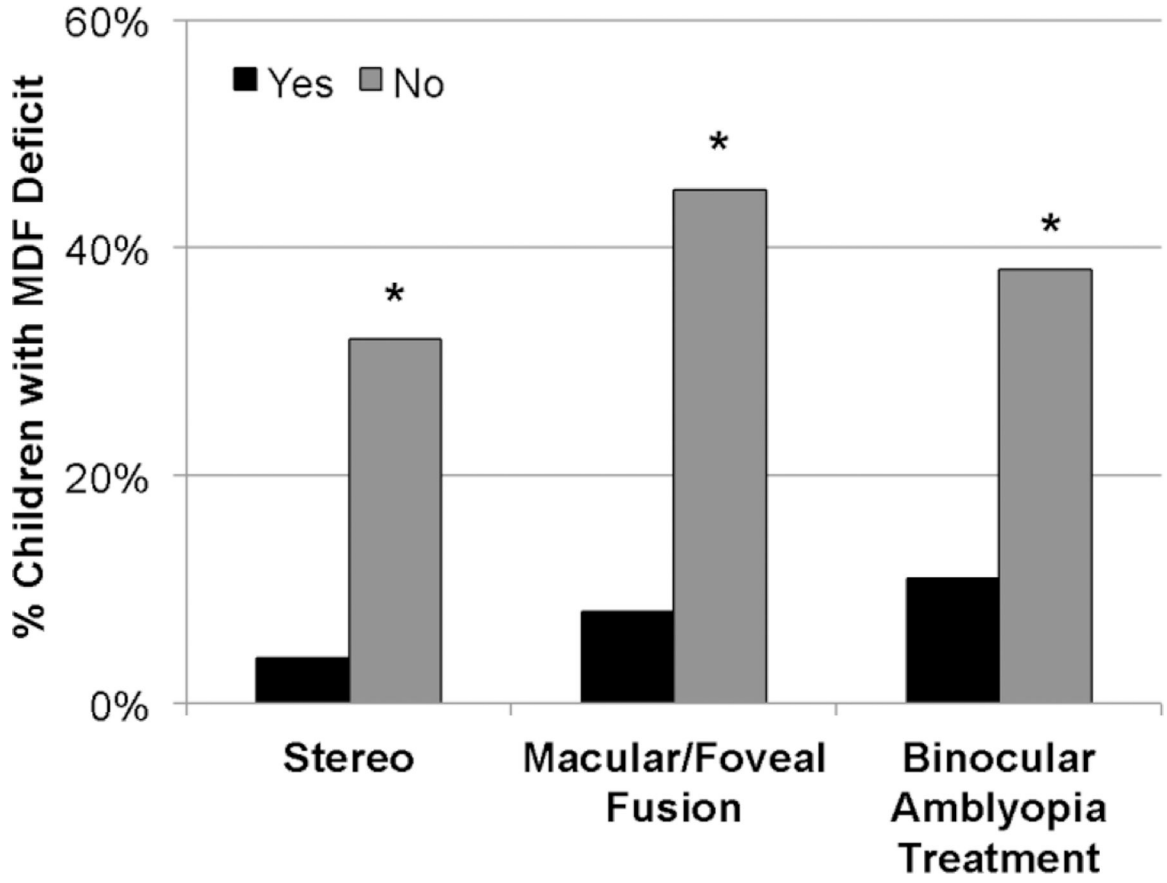

Figure 7.

% of amblyopic children who have a fellow eye deficit for motion-defined form perception in subgroups with randot stereoacuity of 1.6–2.9 log arcsecs (Yes) vs nil stereoacuity (No), with macular/foveal fusion by Worth 4-dot test (Yes) vs peripheral/no fusion, and with prior binocular amblyopia treatment (Yes) vs no prior binocular treatment (No).