Effects of Anisometropic Amblyopia on Visuomotor Behavior, I: Saccadic Eye Movements

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Purpose. Impairment of spatiotemporal visual processing is the hallmark of amblyopia, but its effects on eye movements during visuomotor tasks have rarely been studied. Here the authors investigate how visual deficits in anisometropic amblyopia affect saccadic eye movements.

Methods. Thirteen patients with anisometropic amblyopia and 13 control subjects participated. Participants executed saccades and manual reaching movements to a target presented randomly 5° or 10° to the left or right of fixation in three viewing conditions: binocular, amblyopic, and fellow eye viewing. Latency, amplitude, and peak velocity of primary and corrective saccades were measured.

Results. Initiation of primary saccades was delayed and more variable when patients viewed monocularly with their amblyopic eye. During binocular viewing, saccadic latency exhibited increased variability and no binocular advantage in patients (i.e., mean latency was similar to that during fellow eye viewing). Mean amplitude and peak velocity of primary saccades were comparable between patients and control subjects; however, patients exhibited greater variability in saccade amplitude. The frequency of corrective saccades was greater when patients viewed with their fellow eye than it was with binocular or amblyopic eye viewing. Latency, amplitude, and peak velocity of corrective saccades in patients were normal in all viewing conditions.

Conclusions. Saccades had longer latency and decreased precision in amblyopia. Once saccades were initiated, however, the dynamics of saccades were not altered. These findings suggest that amblyopia is associated with slower visual processing in the afferent (sensory) pathway rather than a deficit in the efferent (motor) pathway of the saccadic system. (*Invest Ophthalmol Vis Sci.* 2010;51:6348–6354) DOI:10.1167/iovs.10-5882

Amblyopia is a spatiotemporal impairment of vision caused by inadequate stimulation of the eyes during early childhood that cannot be corrected by optical means.[1] It affects 3% to 5% of the population.[2] Amblyopia arises from a disruption of binocular input during the critical period for the development of binocularity. It is associated most commonly with early childhood strabismus, anisometropia, or both (i.e., mixed-mechanism)[3] and, more rarely, with image degradation such as congenital cataract. The visual and sensory perceptual deficits in patients with amblyopia have been well documented. They include reduced visual acuity and contrast sensitivity,[4] deficits in global form detection,[5] spatial distortions and temporal instability,[6,7] spatial and temporal crowding,[8] abnormal global motion detection[9,10] and deficits in motion-defined form extraction[11] during amblyopic eye viewing. Importantly, these deficits are present not only during amblyopic eye viewing, they are also evident to a lesser extent during fellow eye viewing.[4,12] Poor or absent stereopsis is also common in amblyopia.[13]

The senses serve to collect sensory information, and the brain's primary task is to perform sensorimotor transformation, a process that involves combining and integrating sensory information from all available modalities for the preparation and execution of purposeful action. Although sensory and motor processes are often considered separately, they are really two facets of a single system, and they cannot be appreciated fully in isolation. Despite extensive evidence of spatiotemporal visual deficits in amblyopia,[2–11,14–16] it is surprising that very few investigators have examined how amblyopic visual deficits impact ocular motor functions such as saccades.

Saccades are fast eye movements that bring the image of an object of interest onto the fovea. During everyday manual activities, saccades move the eyes toward task-relevant objects before hand movement,[17,18] so that optimal visual information pertaining to these objects can be extracted to improve the accuracy and precision of reaching[19] and manual manipulation.[20] Typically, we make approximately 2 to 3 saccades per second, and >85% of natural saccades are <15° in amplitude.[21] Primary saccades tend to be slightly hypometric and are usually followed by secondary corrective saccades after approximately 100 to 250 ms.[22,23]

Two previous studies have examined saccadic control in patients with amblyopia. Schor[24] examined saccades in five patients using a square-wave stimulus that stepped at 0.25 or 0.5 Hz. No difference in latency was found between the amblyopic and fellow eyes, but there was increased variability in latency during amblyopic eye viewing. Ciuffreda et al.[25] tested 11 patients with amblyopia using unpredictable targets presented at eccentricities ranging from 0.25° to 8.5°. They found increased latencies of primary saccades in the amblyopic eyes, but corrective saccades had normal latency. These studies, however, have several major limitations. First, both studies included a heterogeneous group of patients who had strabismic, anisometropic, or mixed-mechanism amblyopia. Second, neither study included a control group with normal vision. Third, only saccadic latency was reported, and it is unknown whether other dynamic parameters of saccades, such as ampli-

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tude and peak velocity, were also affected. Fourth, information on the dynamics or frequency of corrective saccades was not provided.

In this article, we investigated the effects of anisometropic amblyopia alone on the amplitude, peak velocity, and latency of both primary and corrective saccades and compared them with those of control subjects with normal vision during both binocular and monocular viewing. Because we were interested primarily in goal-directed visuomotor behaviors, saccadic eye movements were measured during a visuomotor task that involved looking at and reaching to a visual target. We found that saccade initiation was prolonged during amblyopic eye viewing and that patients with anisometropic amblyopia had no binocular advantage. In addition, these patients failed to generate more corrective saccades despite a decrease in precision of saccade amplitude during both amblyopic eye and binocular viewing.

Patients, Materials, and Methods

Participants

Thirteen patients with anisometropic amblyopia were recruited (five men; age 27 ± 8.8 years; see Table 1 for clinical characteristics). All participants underwent a standard visual assessment, including visual acuity using the Snellen chart, a prism cover test to document eye alignment, measurement of refractive errors, and stereoacuity using the Titmus test. Anisometropic amblyopia was defined as amblyopia in the presence of a difference in refractive error between both eyes of ≥1 diopter (D) of spherical or cylindrical power.[14,15,26–29] All patients had visual acuity between 20/30 and 5/400 in the amblyopic eye and 20/20 or better in the fellow eye and an interocular acuity difference ≥2 lines. Four patients were orthophoric, and nine had monofixation syndrome,[30] defined as microtropia ≤8 PD (as a result of a foveal scotoma arising from anisometropia; it was not the cause of the amblyopia), inability to bifixate, and presence of fusional vergence. Nine patients had fine to gross stereopsis, and four had no stereopsis. Thirteen control subjects (six men; age 27.8 ± 6.7 years) who had normal or corrected-to-normal visual acuity (20/20 or better) in both eyes and stereoacuity ≤40 seconds of arc were also recruited. Exclusion criteria were any ocular cause for reduced visual acuity, previous intraocular surgery, or any neurologic disease. All participants were right-handed. The study was approved by the Research Ethics Board at the Hospital for Sick Children, and all protocols adhered to the guidelines of the Declaration of Helsinki. Informed consent was obtained from each participant.

Apparatus

The visual target was a white square (visual angle, 0.5°) presented on a black background generated by a custom-written computing program (MatLab; MathWorks, Natick, MA) and presented on a CRT computer monitor using the visual stimulus generator (ViSaGe; Cambridge Research Systems, Cambridge, UK). Testing was conducted in a dimly lit room. Eye movements were recorded binocularly at 200 Hz using a video-based pupil/iris tracking system (Chronos Vision, Berlin, Germany). This system has a maximum resolution of 6 minutes of arc over a range of ±20°; linearity is <0.5% for both horizontal and vertical eye movements. Before each experiment, horizontal and vertical calibrations were performed for each eye using fixation targets at five locations at 0° and ±10° horizontally and vertically.

Experimental Conditions and Procedure

Participants were seated at a table with their heads stabilized on a chin rest. At the start of each trial, the index finger of the right hand was placed on the table at their sagittal midline 28 cm from the screen. Participants fixated a cross on the screen that was centered vertically at their eye level and horizontally along their midsagittal plane. After a variable delay of 1.5 to 3 seconds, the fixation cross was extinguished and the target appeared (i.e., there was no temporal gap between fixation and target) randomly at four eccentricities at ±5°or ±10° from central fixation in the horizontal plane. The participants were instructed to look at and point to the target as quickly and as accurately as possible using their right index finger. In 50% of the trials, the target was switched off at the onset of hand movement, approximately 350 ms after target onset. For the other 50% of the trials, the target remained on the screen. Trials with and without visual feedback of target were randomized on a trial-by-trial basis.

The experiments were performed under three viewing conditions: binocular eye (BE), monocular amblyopic eye (AE), and monocular fellow eye (FE). For control subjects, viewing was binocular, monocular left eye, and monocular right eye. Data were collected in blocks for each viewing condition, and the order of viewing conditions was randomized across subjects. Participants completed 10 trials in each combination of the experimental conditions for a total of 240 trials. Practice trials were completed before the experiment was begun to familiarize the participants with the experimental procedure.

Statistical Analysis

Eye position data were low-pass filtered using a second-order, dual-pass Butterworth filter with a cutoff frequency of 50 Hz. Eye velocity was obtained using a two-point differentiation method. A custom-written computing program (MatLab; MathWorks) program was used to iden-

Table 1. Clinical Details of Studied Patients with Amblyopia

Patient	Age (y)	Visual Acuity		Refractive Error		Stereoacuity (sec arc)	Monofixation
		RE	LE	RE	LE		
1	33	20/15	20/30	−0.75	+2.00	140	Orthophoria
2	29	20/50	20/15	+2.50 + 0.75 × 50	+0.25	3000	Monofixaton (RE)
3	47	20/40	20/15	+2.75 + 2.25 × 60	Plano	400	Monofixation (RE)
4	14	20/50	20/15	+3.25 + 1.25 × 90	+2.00	50	Orthophoria
5	18	20/15	20/40	Plano	+2.00 + 0.25 × 130	60	Ortophoria
6	20	20/15	20/50	Plano	+1.50	120	Orthophoria
7	35	20/15	20/60	−4.25	−0.75	3000	Monofixation (LE)
8	36	20/15	20/400	−5.25	−12.00	Negative	Monofixation (LE)
9	25	20/40	20/15	+1.00 + 0.25 × 22	Plano	400	Monofixation (RE)
10	28	20/15	20/200	+4.00	+6.00 + 1.75 × 90	Negative	Monofixation (LE)
11	28	20/20	20/400	Plano + 0.50 × 90	+6.50	Negative	Monofixation (LE)
12	21	20/30	20/15	+1.50	Plano	3000	Monofixation (RE)
13	20	5/400	20/20	−2.00	−3.00 + 0.75 × 15	Negative	Monofixation (RE)

LE, left eye; RE, right eye.

tify primary saccades using a velocity threshold of 20°/s. All trials were inspected visually to ensure that saccades were identified correctly by the computer program. Outcome measures were primary saccade latency, amplitude, and peak velocity. The mean and the variability (SD) of each outcome measure were analyzed using separate repeated-measures mixed ANOVA, with one between-subjects factor (group [control subjects and patients]) and three within-subjects factors (viewing condition [binocular, monocular fellow eye, and monocular amblyopic eye; for control subjects, monocular left and right eye viewing]), target location [±5°, ±10°]), and visual feedback of target [on, off]). To investigate further the effects of severity of amblyopia on saccade performance, a repeated-measures ANOVA was performed on each outcome measure. The ANOVA included severity of amblyopia (mild, 20/30–20/60; severe, 20/200–5/400) as a between-subjects factor and viewing condition as a within-subjects factor.

All trials were inspected for the presence of secondary saccades, which were marked manually for each trial. Secondary saccades that occurred within 250 ms of the primary saccades were defined as *corrective* saccades.[22,31,32] The overall frequency of corrective saccades (pooled from all three viewing conditions) and the frequency of corrective saccades made during each viewing condition were compared between patients and control subjects using Pearson's χ^2 statistic. Latency, peak velocity, and amplitude of corrective saccades were each analyzed using a repeated-measures mixed ANOVA with group as a between-subjects factor and viewing condition as a within-subjects factor.

All statistical analyses were performed using the SAS 9.2 software package (SAS Institute, Cary, NC). The significance level was set at $P < 0.05$. Any significant main effects and interactions were analyzed further using post hoc pairwise comparison *t*-tests. Preliminary analysis showed that visual feedback of target had no significant effect on any outcome measures. Therefore, the results reported herein are pooled across the two visual feedback conditions (on and off).

Results

Primary Saccade Latency

Representative eye velocity tracings from individual trials for a control subject during binocular and left eye viewing and for a patient during binocular and amblyopic eye viewing are shown in Figure 1. Saccade latency was increased and more variable in the patient during both binocular and monocular (amblyopic eye) viewing conditions than in the control subject. Similar findings were also observed in the other 12 patients. Analysis on group mean saccade latency showed a significant main effect of viewing condition (binocular, monocular fellow eye, and monocular amblyopic eye; for control subjects, monocular left and right eye viewing; $F_{(2,48)} = 17.70$; $P < 0.0001$). A significant interaction was also found between group (patients and control subjects) and viewing condition ($F_{(2,48)} = 7.46$; $P = 0.0015$). Post hoc tests revealed that mean saccade latency increased significantly when patients viewed with their amblyopic eye (236 ± 72 ms) than with their fellow eye (191 ± 38 ms) or both eyes (186 ± 48 ms). In contrast, control subjects had significantly shorter saccadic latency when viewing binocularly (175 ± 32 ms) than monocularly with either eye (left eye, 192 ± 29 ms; right eye, 195 ± 31 ms). Saccade latency was comparable between control subjects during monocular viewing and between patients during binocular or fellow eye viewing (Fig. 2). No other significant main effect or interaction was observed for mean saccade latency.

For variability (i.e., SD) in saccade latency, an overall main effect was found for group ($F_{(1,24)} = 12.71$; $P = 0.0016$), with patients (41 ± 26 ms) exhibiting significantly greater variability than control subjects (28 ± 11 ms). The interaction between group and viewing condition was also significant ($F_{(2,48)} = 5.50$; $P = 0.0070$). As shown in Figure 3, post hoc tests revealed that the variability in saccade latency increased when patients viewed with their amblyopic eye (54 ± 30 ms) rather than with their fellow eye (36 ± 18 ms) or with both eyes (35 ± 22 ms). Post hoc tests also indicated no significant differences between viewing conditions for the controls (binocular, 27 ± 12 ms; left eye, 27 ± 10 ms; right eye, 30 ± 10 ms). No other significant main effect or interaction was observed for variability in saccade latency.

Primary Saccade Amplitude

As expected, an overall main effect on mean amplitude was found for target location ($F_{(3,72)} = 632.19$; $P < 0.0001$), with saccades to the 10° target reaching higher amplitude than those to the 5° target in both patient and control groups. An overall main effect of viewing condition was also found for mean saccade amplitude ($F_{(2,48)} = 5.43$; $P = 0.0075$). Post hoc tests indicated that mean saccade amplitude was higher during binocular viewing than during monocular viewing. No other significant main effect or interaction was observed for mean saccade amplitude. Of particular note, mean saccade amplitude was not significantly different between patients and control subjects in any of the viewing conditions ($F_{(2,48)} = 0.20$; $P = 0.8154$). A main effect for group, however, was found for variability in saccade amplitude ($F_{(1,24)} = 4.83$; $P = 0.0379$); saccade amplitude was more variable in the patient group (approximately 1°) than in the control group (approximately 0.70°) in all three viewing conditions (Fig. 4).

Primary Saccade Peak Velocity

As expected, an overall main effect on mean peak velocity was found for target location ($F_{(3,72)} = 327.28$; $P < 0.0001$), with saccades to the 10° target reaching higher peak velocity than those to the 5° target in both patient and control groups. An overall main effect of viewing condition was also found on mean peak velocity ($F_{(2,48)} = 8.84$; $P = 0.0005$). Post hoc tests indicated that mean peak velocity was higher during binocular viewing than during monocular viewing. Mean peak velocity in patients was comparable with that in control subjects in all viewing conditions. There was no interaction between group and viewing condition ($F_{(2,48)} = 0.26$; $P = 0.7712$), indicating that mean peak velocity was higher in both patient and control groups during binocular viewing. No other significant main effect or interaction was observed for mean peak velocity.

Corrective Saccades

The frequency of corrective saccades was significantly greater in patients (9.35%) than in control subjects (6.94%) ($\chi^2_{(df=1)} = 12.19$; $P = 0.0005$). We also found differences between patients and control subjects when the frequency of corrective saccades was analyzed separately across viewing conditions. Control subjects executed corrective saccades with comparable frequency when viewing monocularly (right eye, 7.02%; left eye, 7.60%) and binocularly (6.22%; $\chi^2_{(df=2)} = 1.61$; $P = 0.4464$). In contrast, patients executed corrective saccades more frequently when viewing with the fellow eye (14.23%) than during binocular (6.73%) or amblyopic eye viewing (7.06%; $\chi^2_{(df=2)} = 43.67$; $P < 0.0001$). There were no differences in latency, peak velocity, or amplitude of corrective saccades between patients and control subjects.

Effects of Visual Acuity

There was no significant main effect of severity of amblyopia on saccade performance, indicating that patients with mild amblyopia (20/30–20/60; $n = 9$) had saccadic deficits similar to those of patients with severe amblyopia (20/200–5/400; $n = 4$), that is, longer saccade latencies and increased variability in

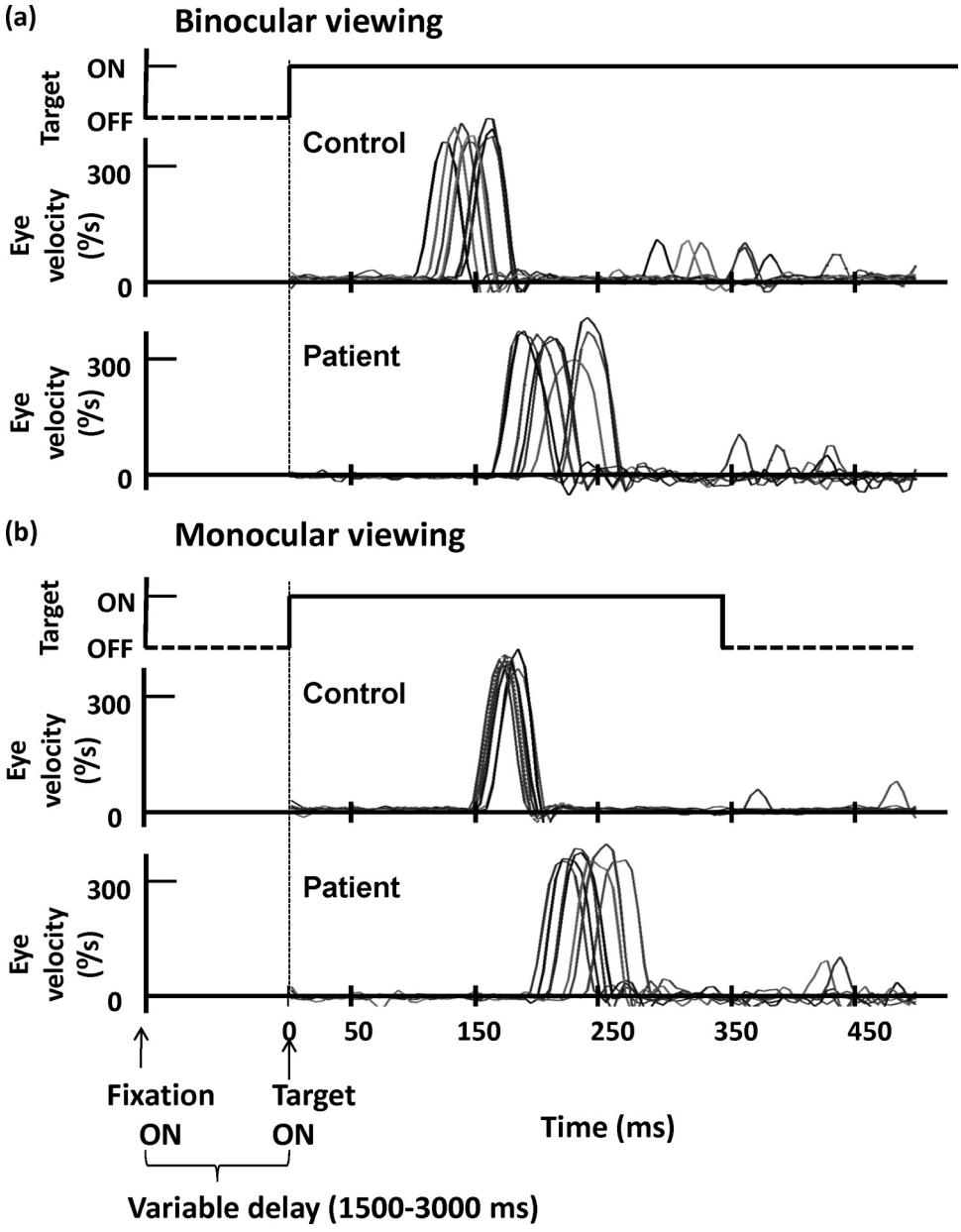

FIGURE 1. Representative eye velocity profiles from individual trials from a control subject and a patient with anisometropic amblyopia when they made saccades to a 10° target to the right during binocular viewing (**a**) and monocular viewing (**b**) with the left eye in the control subject and with the amblyopic eye in the patient. The target was displayed on the monitor throughout the trial (**a**) or disappeared at the onset of hand movement (**b**). Visual feedback of the target had no significant effect on any saccade outcome measures. Saccade latency was increased and more variable in the patient during both binocular and monocular (amblyopic eye) viewing conditions than in the control subject.

latency and amplitude of saccades compared with those of control subjects. There was no interaction between severity of amblyopia and viewing condition. It should be noted, however, that the lack of statistically significant effects of severity of amblyopia on saccade performance may well be attributed to the small number of patients in the two subgroups and to the unequal sample size between the two subgroups.

In addition, we performed a correlation analysis to determine whether there was a relationship between visual acuity and outcome measures (mean saccade latency, variability in saccade latency, and variability in amplitude). Pearson correlation coefficients were not statistically significant in any of the viewing conditions, suggesting that the saccade deficits seen in our patients were not correlated with acuity loss.

DISCUSSION

To the best of our knowledge, this is the first study to examine saccadic eye movements systematically in patients with anisometropic amblyopia only and to compare them to movements in control participants with normal vision. The three major findings are that patients with anisometropic amblyopia exhibited a significant increase in saccade latency that was also highly variable during amblyopic eye viewing, that patients had no binocular advantage (i.e., a reduction in saccade latency during binocular viewing was not observed in patients),[33] and that patients did not generate more corrective saccades despite a decrease in precision of saccade amplitude during amblyopic eye and binocular viewing. Our results are in general agreement with those of previous studies[24,25] and extended their findings by including an age-matched control group and patients with anisometropic amblyopia only.

Increased Saccade Latency

It has been suggested that the longer saccade latency during amblyopic eye viewing results from an abnormally long spatiotemporal sensory acquisition of the target rather than from a motor deficit.[25] This is supported by a previous finding that

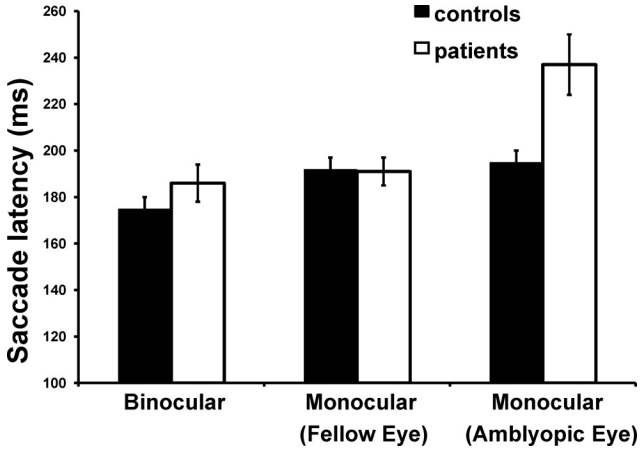

FIGURE 2. Mean latency of primary saccades for control subjects and patients in each viewing condition: binocular, monocular fellow eye, and monocular amblyopic eye viewing. For control subjects, viewing was binocular, monocular left eye, and monocular right eye. Saccade initiation was significantly delayed when patients viewed with their amblyopic eye ($P = 0.0015$). No binocular advantage was found when patients viewed binocularly compared with control subjects. Error bars, ±1 SEM.

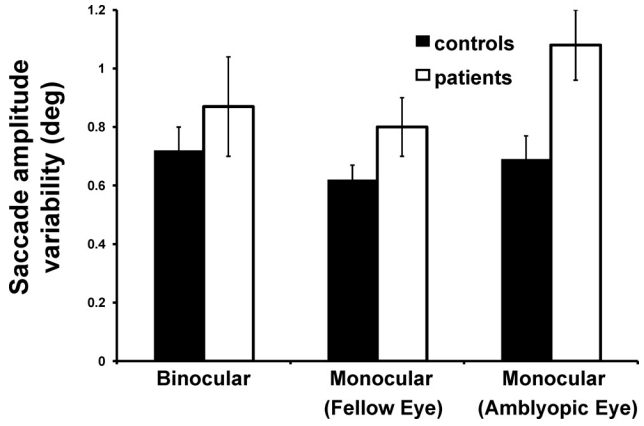

FIGURE 4. Variability in amplitude of primary saccades for control subjects and patients in each viewing condition: binocular, monocular fellow eye, and monocular amblyopic eye viewing. For control subjects, viewing was binocular, monocular left eye, and monocular right eye. Variability in saccade amplitude was significantly greater ($P = 0.0379$) in the patient group in all viewing conditions. Error bars, ±1 SEM.

when a predictable target was used, saccade latency was comparable during amblyopic and fellow eye viewing.[24] In this study, we found that once the target was detected, the dynamics of the saccades were not altered, as evidenced by normal saccadic latency when patients viewed with their fellow eye, normal latencies for corrective saccades in all viewing conditions (including during amblyopic eye viewing), and normal mean amplitude and peak velocity of both primary and corrective saccades in patients, irrespective of the viewing conditions. Our results provide further support that the increased saccade latency in amblyopia represents a slower visual processing in the afferent (sensory) pathway rather than a deficit in the efferent (motor) pathway of the saccadic system.[25]

The increased variability in saccade latency and amplitude during amblyopic eye viewing further suggested that patients had difficulty detecting and localizing the target. Variability is an inherent feature of motor control and can come from both sensory and motor processing.[34] Signal detection theory postulates that decisions are based on noisy sensory signals and that a decision about the presence or absence of a target is reached when the accumulating sensory signals reach a threshold.[35] It has been shown that patients with amblyopia exhibited a marked loss of efficiency (threshold elevation at all noise levels) and increased random internal noise.[36–38] It is possible that because the sensory signals used to program the saccadic response are degraded in amblyopia, a longer and more variable period is required for the noisy sensory signals to reach threshold, resulting in highly variable saccade latency during amblyopic eye viewing.

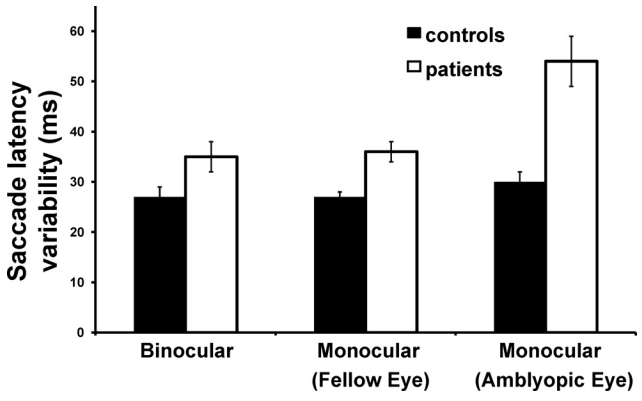

FIGURE 3. Mean variability in the latency of primary saccades for control subjects and patients in each viewing condition: binocular, monocular fellow eye, and monocular amblyopic eye viewing. For control subjects, viewing was binocular, monocular left eye, and monocular right eye. Variability in latency increased significantly ($P = 0.0070$) for patients in all viewing conditions compared with controls. Error bars, ±1 SEM.

Amblyopia has been associated with unsteady fixation,[39–45] including square wave oscillations, manifest latent nystagmus, and infantile nystagmus (Subramanian V, et al. *IOVS* 2010;51: ARVO E-Abstract 4759).[41] Analysis of eye movement data and model simulation have shown that latency to target acquisition and saccade latency are prolonged in patients with infantile and manifest latent nystagmus.[46] Although none of our patients exhibited any nystagmus, it is possible that they had unsteady fixation (e.g., drifts) that might have interfered with target acquisition and saccade initiation. The study design we used, however, precludes us from investigating this possibility. Further studies using a specific protocol designed to test fixation stability[47,48] are under way to investigate whether fixation instability in amblyopia may play a role in the increased saccade latency we documented.

The exact loci of the neural substrates underlying the visual deficits in amblyopia are unknown. Studies using visually evoked potentials[49] and magnetoencephalography[50] have shown increased latency of V1 activation when viewing with the amblyopic eye. In addition, imaging studies have shown that the level of activity in lateral geniculate nucleus,[51] V1, and extrastriate areas (V2, V3, V3a)[52–54] was reduced when patients viewed with their amblyopic eye compared with their fellow eye. The increased latency and decreased amplitude of neural activity in the early visual areas might explain the slower visual processing in the afferent (sensory) pathway of the ocular motor system we observed.

Lack of Binocular Advantage

A second major finding from this study is that patients with amblyopia showed no binocular advantage. In visually healthy participants, response times have been shown to be shorter

when subjects viewed binocularly rather than monocularly, even when the task involved two-dimensional displays that did not require stereopsis.[33] It has been proposed that during binocular viewing, the inputs from both eyes contain correlated stimulus signals that summate during visual processing, whereas the noise signals in the stimulus from each eye are uncorrelated, effectively cancelling each other when combined. As a result, the signal-to-noise ratio in the stimulus signals increases during binocular viewing, which, in turn, leads to more accurate, precise, and faster responses.[55] This binocular advantage, however, was not evident in people with amblyopia, as indicated by the observation that saccade latency during binocular viewing was no better than that during fellow eye viewing, possibly because of a disruption of binocular organization and a loss of binocularity in neurons in the visual cortex in amblyopia in general[56–58] and in anisometropic amblyopia specifically.[13,58] Note, however, that a recent study demonstrated that binocular summation of contrast sensitivity is normal in people with strabismic amblyopia when stimulus contrast is adjusted to equalize visibility of the gratings for both eyes,[59] suggesting that the binocular summation deficits seen in previous studies[4,13,60–64] may result from interocular differences in contrast sensitivity between the eyes.

Increased Frequency of Corrective Saccades during Fellow Eye Viewing

A small saccade hypometria (undershooting of the target) is commonly seen in people with normal vision.[21] The amount of hypometria is usually approximately 10% of the saccade amplitude for unpredictable visual targets.[23,65] When a saccade undershoots the target, people with normal vision usually make a corrective saccade with a latency of 100 to 250 ms.[22,23,31] Two mechanisms have been proposed for the generation of corrective saccades. One is based on extraretinal information derived by monitoring the efferent ocular motor command or the efference copy (or proprioceptive signals) so that a corrective saccade can be triggered if the primary saccade is inaccurate.[66,67] Another mechanism is based on retinal feedback derived by monitoring the position of the target image on the retina at the end of the primary saccade.[31,32,66,67] Evidence in favor of this mechanism is provided by the finding that the probability of the occurrence of a corrective saccade and its accuracy both increase if a visual target is available at the end of the primary saccade.[31,32,66,67]

In the present study, we found that the precision of primary saccade amplitude decreased (increased variability) in all three viewing conditions in patients with amblyopia. The frequency of their corrective saccades, however, increased only during fellow eye viewing, not during amblyopic eye or binocular viewing. It is possible that because the fellow eye had normal acuity, patients were using both retinal feedback and extraretinal information to generate more corrective saccades during fellow eye viewing. In contrast, during amblyopic eye and binocular viewing, the retinal feedback mechanism may be impaired because of less reliable retinal position error signals of the target image from the amblyopic eye,[68,69] such that less corrective saccades are generated than during fellow eye viewing.

In conclusion, we investigated the effects of anisometropic amblyopia on saccadic eye movements toward single high-contrast targets during a manual pointing task. Our results showed that saccade initiation was prolonged and was highly variable during amblyopic eye viewing, suggesting that the efficiency of the sensory (visual) component of the saccadic system was decreased for the detection (latency) and localization (amplitude variability) of the target. The dynamics of primary and corrective saccades, however, were not altered once eye movement was initiated, suggesting that the efferent (motor) component of the saccadic system is spared in anisometropic amblyopia. Our results lead to several interesting questions. For example, how does the degraded visual input in patients with anisometropic amblyopia affect the planning and execution of visuomotor skills? How do these visual deficits impact the spatiotemporal coordination between the ocular and manual motor systems during reaching? How do these changes differ among different subtypes of amblyopia? Further studies are under way in our laboratory to investigate these issues.

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