Original Article

Saccadic Adaptation in Children

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ABSTRACT

Saccades are fast-orienting eye movements. Saccadic adaptation, a form of motor learning, is a corrective change in the amplitude of saccades in response to error. The aim of the study was to ascertain whether saccadic adaptation occurs in typically developing children. We recorded saccades with an infrared eye tracker in 39 children, aged 8 to 19 years, at baseline to 12-degree horizontal target steps and after an adaptive task. During the adaptive task, a saccadic hypometric error was induced. This task consisted of 200 12-degree target steps that stepped backward 3 degrees during the initial saccade and without the participants' awareness. The initial saccade triggered the back-step. This paradigm required a corrective reduction of the amplitude of the initial saccades in response to the induced error. Saccadic adaptation was achieved in 26 participants, whose mean saccadic amplitudes decreased by 13% ($P < .05$). Saccadic adaptation was not influenced by age. We conclude that children as young as 8 years old have established functions of the neural circuits responsible for the motor learning required for saccadic adaptation. (*J Child Neurol* 2006;21:1025–1031; DOI 10.2310/7010.2006.00238).

Saccadic amplitude inaccuracies occur as a result of aging and disease.[1,2] Saccadic adaptation, a form of oculomotor learning, is a process by which errors in the saccadic amplitude can be gradually corrected.[3] Errors in saccadic amplitude can be induced experimentally by repeatedly changing the position of the target during a saccade.[4,5] This paradigm initially produces a corrective saccade after every intrasaccadic target jump. Saccades are too brief for visual feedback to guide the eye to the new target position during the initial saccade in response to the induced error.[6] However, after as few as 150 such trials, subjects unknowingly adjust the amplitude of their saccades to land closer to the new target position to correct the error.[4,7–9]

Saccadic adaptation is involuntary.[7,10] Subjects are unaware of the intrasaccadic target jump because vision is suppressed during saccades.[11–13] The retinal error signal (distance between the fovea and target image), rather than a motor error signal (saccadic amplitude required to accurately foveate the target), drives the adaptive process.[14–16] Saccadic adaptation is specific to the direction and amplitude of the target movement.[5,7,17–19] Saccadic adaptation is best demonstrated by 10% to 40% reductions in target amplitude[20] and is reversible.[5,20]

Saccades have been studied extensively in adult human and nonhuman primates. Less research on eye movements has been done in children. Thus, limited knowledge on the course of normal development of eye movements in general and on saccadic adaptation in particular is available on children.[21] The aims of this study were to investigate whether saccadic adaptation occurs in typically developing children, to measure the magnitude of any adaptation, and to determine the effects of age and gender on saccadic adaptation.

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Dr Eizenman is the developer of the tracker. He has shares and interest in El-Mar Inc., the manufacturer of the eye tracker.

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METHODS

Participants

Thirty-nine typically developing children (21 boys) between 8 and 19 years of age (mean age 13 years 8 months, SD 3 years 6 months) were recruited by local advertising. The study was in accord with the Declaration of Helsinki guidelines, and ethical approval for this project was obtained from the Research Ethics Boards of The Hospital for Sick Children and the University Health Network, Toronto. Written consent and assent were obtained from the participants and their legal guardian. Participants with best-corrected monocular visual acuity of 20/40 were selected and excluded if they had developmental delay or diagnosed learning difficulties; visual field defect on visual field confrontation testing; peripheral cranial nerve III, IV, or VI palsy; nystagmus; ear disease; or diagnosed ocular, neurologic, or psychiatric disorders or were on medication with drugs that might interfere with eye movements (eg, sedatives or anticonvulsant medication).

Equipment and Procedures

We recorded saccades with the El-Mar eye tracker (El-Mar Inc., Downsview, ON, Canada), an infrared eye tracking system that determines the horizontal and vertical eye positions from the relative positions of multiple corneal reflections and the center of the pupil.[22,23] The optical components of the tracker are mounted on a lightweight spectacle frame that weighs ≈ 300 g. The video image is sampled at 120 Hz. The system accuracy is 0.5 degrees, with a linear visual range ±40 degrees horizontally and ±30 degrees vertically. The system is free from drift and has a resolution (ie, minimum detectable movement) of 0.1 degrees. Horizontal and vertical head movements were recorded using a magnetic head tracker (Flock of Birds™, Ascension Technology Corp., Burlington, VT).

Each participant was seated on a chair, with their eyes in the central position, facing the center of a 45 cm computer monitor (Samsung, SyncMaster 900 NF) located 57 cm from the participant's cornea. The visual target displayed on the computer monitor was a 2 mm white square light that subtended 12 minutes of arc. Stimulus luminance was 4.1 cd/m^2. The background monitor luminance was 0.001 cd/m^2. The laboratory background was dark. Participants' performance and alertness were monitored by television and by an oscilloscope display of horizontal and vertical eye movements to provide feedback during the task.

Each eye was calibrated, with the fellow eye occluded, by saccades to 14 fixation light points, arrayed along the horizontal and vertical axes, and separated by a 3.3-degree visual angle. The participant's head was stabilized using a chin rest and adjusted so that the eyes were in the central position when looking at the center of the array. Eyeglasses were removed prior to testing because they interfere with the function of the eye tracker. The uncorrected visual acuity in all cases was adequate for seeing and responding to the stimuli.

An eye patch was used to cover the nonpreferred (nonsighting) eye.[24] Movements of the viewing eye were measured. The target was programmed to step randomly at intervals from 0.8 to 1.2 seconds. Targets stepped rightward from points distributed randomly on the mid–horizontal axis after each trial to points 12 degrees horizontal to the starting point. Fifty target steps were presented before the adaptive task, and 50 target steps were presented after the adaptive task. During the adaptive saccadic task, the same target steps were presented randomly, but when the initial rightward saccade reached a velocity of 25 degrees/second, the target position was moved 3 degrees backward (leftward) (Figure 1). Therefore, the task induced saccadic hypometric errors.

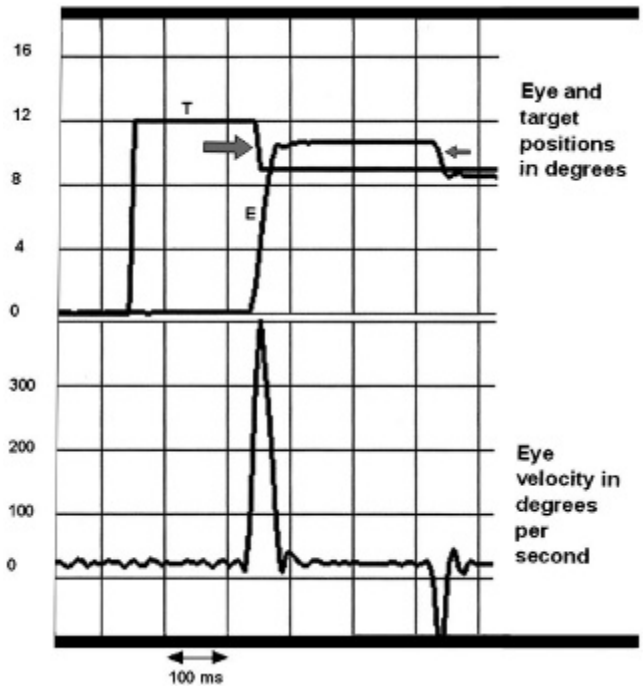

Figure 1. Saccadic adaptation using a target back-step paradigm. A horizontal right eye saccade is shown in response to visual target displacement of 12 degrees during the early stages of saccadic adaptation. Horizontal eye (E) and visual target (T) positions are displayed (*top*). Upward represents rightward eye movement. The *lower part* of the figure shows eye velocity. Time is on the x-axis in milliseconds. The target is displaced back by 3 degrees (*rightward arrow*) during the initial saccade. The ''apparent'' induced hypermetria of the initial saccade relative to the final target position is corrected by a small backward saccade (*leftward arrow*).

Participants were unable to detect the target back-step when the target screen was covered by neutral density filters. Without the filters, some participants were aware of the target back-step during a pilot study. Participants were presented with 200 adaptive target steps over four successive sessions (50 adaptive target steps per session).

The stimulus, head, and eye movements were digitized for off-line analysis. Eye position signals were not filtered, but stimulus, head, and eye velocity signals were filtered using a 5-point Savitsky-Golay differentiator.

Initial saccades were included in the analyses only if they had a minimum velocity > 100 degrees/second, were in the same direction as the target displacement, the eye position trace shifted < 0.5 degrees from baseline during the 200 milliseconds prior to target displacement up to saccade onset, and if saccades occurred within a latency of 70 to 450 milliseconds to ensure that only visually directed nonanticipatory saccades were included. Adaptive saccades were included only if the target back-step occurred during or before the end of the initial saccade. The beginning and end of saccades were marked automatically by interactive software when eye velocity crossed 30 degrees/second. Each second of data was displayed on a computer monitor so that the automatic markings could be edited by cursors.

Mean horizontal head position associated with the saccades was checked for each participant before, during, and after the saccades to ensure that no head movements, defined as mean horizontal head rotation ≥ 0.5 degrees, confounded the data by inducing the vestibulo-ocular reflex or changed the size of the required saccade.

Analyses

For each participant, mean saccadic amplitude gain (G), defined as saccadic amplitude/target amplitude, and standard deviation were calculated for initial saccades at (1) baseline (preadaptive phase), which consisted of 50 target steps without back-steps (G1); (2) the last 50 of the 200 targets with back-steps (end of the adaptive phase) (G2); and (3) after the adaptive targets (postadaptive phase), which consisted of 50 target steps without back-steps (G3).

Saccadic latencies were defined as the time interval between target displacement and the beginning of the saccade at the point when the eye velocity trace exceeded 30 degrees/second at (1) baseline (preadaptive phase) (L1); (2) the last 50 of the 200 targets with back-steps (end of the adaptive phase) (L2); and (3) after the adaptive targets (postadaptive phase) (L3).

Analyses were done with the *Statistical Package for the Social Sciences* (SPSS Inc, Chicago, IL). Normality of data distribution was tested using the mean, median, standard deviation, skewness, kurtosis, box plots, normal Q-Q plots, and Shapiro-Wilk test. Group gain changes were investigated by comparing G2 and G3 with G1 using paired, two-tailed Student *t*-tests. To investigate individual variability in saccadic adaptation, G2 and G3 were compared with G1 for each participant using paired, two-tailed Student *t*-tests. This comparison allowed the calculation of the percentage of participants who adapted (ie, had a significant saccadic gain reduction from baseline). To quantify the amount of adaptation, the mean relative change in saccadic amplitude gain was calculated at the end of the adaptive phase (Ge) (ie, [G1–G2]/G1) and the postadaptive phase (Gp) (ie, [G1–G3]/G1) for each participant. This method has been used previously.[9,14,16,25] The same procedure was repeated in the participants who adapted. Group latency changes were investigated by comparing L2 and L3 with L1 using paired, two-tailed *t*-tests. To quantify the change in latency following adaptation, the mean relative change in latency was calculated at the end of the adaptive phase (Le) (ie, [L1–L2]/L1) and the postadaptive phase (Lp) (ie, [L1–L3]/L1) for each participant. The same procedure was repeated in the participants who adapted.

The effect of age and the number of adaptive saccades on the magnitude of saccadic adaptation were investigated using the two-tailed Pearson's correlation test for normally distributed data or Spearman's correlation rank test for nonparametric data. Gender differences were investigated using independent two-tailed Student *t*-tests. Further analyses were done using linear stepwise regression models to investigate the strength of association of saccadic adaptation magnitude with age, gender, and the number of adaptive saccades and to ascertain which of those explanatory variables were most strongly associated and therefore potentially predictive of saccadic adaptation. Logistic regression was used to investigate the same factors, with adaptation status as the dependent variable. The probability of F to enter was ≤ 0.05 and to remove ≥ 0.1. The best model was selected based on the adjusted R^2 and a P value $< .05$.

The relationship between the ability to adapt and baseline gain was investigated by comparing baseline gain (G1) between participants who adapted and participants who did not adapt. G1 was compared using independent two-tailed Student *t*-tests. This analysis was done because it has been suggested that people with abnormal saccadic gains might have impaired saccadic adaptation.[21]

To investigate if any reduction in baseline gain might have been be caused by saccadic fatigue,[26,27] G2 (end of adaptation gain) was compared with G3 (postadaptation gain). The rationale is that G3 would be expected to be smaller than G2 if the reduction from baseline gain was caused by fatigue, whereas with adaptation acquisition and a subsequent increase in saccadic amplitude, G3 is expected to be significantly higher than G2. Fatigue in saccadic performance, defined as a decrease in saccadic amplitude after making several hundred saccades, is attributed to "mental" (ie, feeling tired) rather than peripheral nerve, neuromuscular junction, or extraocular muscle fatigue.[26,27] Parameters that were not normally distributed were analyzed using nonparametric statistical tests. For all tests, significance was defined by P values $< .05$.

RESULTS

All 39 participants completed the saccadic adaptation task. Saccadic gains and latencies at baseline, end of adaptation, and postadaptation had approximately normal distributions. None of the participants reported noticing the back-step when we asked them at the end of the task.

Saccadic adaptation, defined as a significant reduction in baseline amplitude gain following the adaptation phase (Figure 2), occurred in 26 of the 39 participants at the end of the adaptive phase. Twenty of the 39 participants retained significant saccadic gain reduction through the postadaptation phase. The group mean percentage amplitude gain reduction, Ge (standard error), was 9.3% (1.2) at the end of the adaptive task. The ideal decrease in

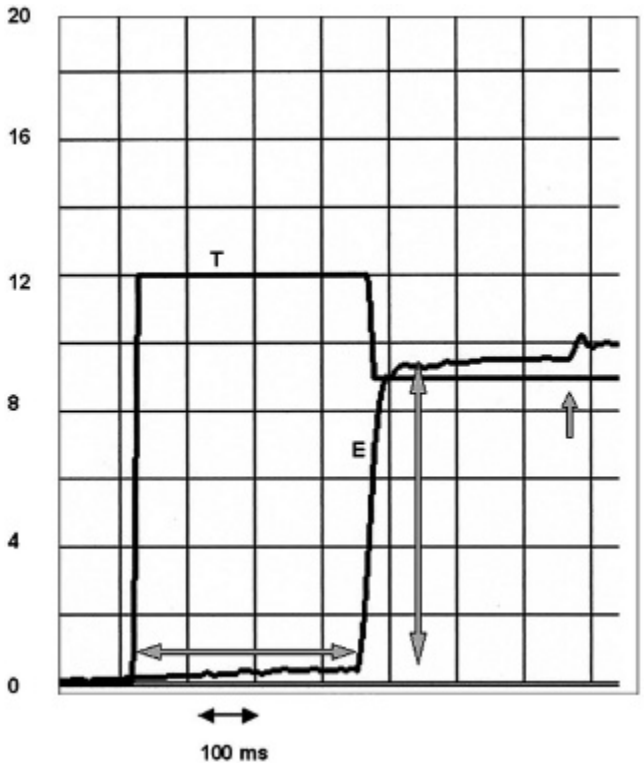

Figure 2. Saccade adaptation. Horizontal eye (E) and target (T) traces are illustrated. Upward deflection represents rightward displacement of the eye. Horizontal saccades are shown in response to visual target displacement of 12 degrees at the latter stages of adaptation, recorded from the right eye. During the initial saccade, the target is displaced back by 3 degrees. The vertical scale shows saccade amplitude in degrees. Time is displayed on the x-axis in milliseconds. The figure shows a decrease in saccade amplitude (*vertical double arrow*) and an increase in latency (*horizontal double arrow*) after 200 adaptive target steps, in comparison with the early stages of adaptation shown in Figure 1. Furthermore, the corrective backward saccade in Figure 1 is replaced by a small saccade to the right (*upward small arrow*).

gain was 25% (3-degree back-step from a 12-degree target step). In other words, participants attained 37.2% (9.3 of 25) of ideal gain reduction at the end of the adaptive task. The corresponding value of Ge (SE) for the subgroup of participants who adapted was 13.3% (0.7) at the end of the adaptive task. That is, participants attained 53.2% (13.3 of 25) of optimal gain reduction at the end of the adaptive task (Table 1 and Figure 3, A and B). No participant had a significant increase in amplitude gain.

Saccadic latency increased at the end of the adaptation task in 31 participants, which included 19 of the 26 participants who adapted. The group mean percentage latency increase, Le (standard error), was 7.8% (1.6) at the end of the adaptive task ($n = 39$). The corresponding value (standard error) for the subgroup of participants who adapted was 7.0% (2.2) at the end of the adaptive task ($n = 26$) (see Table 1 and Figure 3, C and D). The time course of the adaptation process in one participant is shown in Figure 4.

When saccadic parameters between the beginning and end of adaptation and between the beginning and postadaptation phases were compared, the group mean amplitude gain decreased, whereas mean latency increased significantly (Table 2). There was no significant effect of age (Figure 5) or gender on Ge. The number of adaptive saccades (ie, initial saccades that triggered target back-steps) did not affect Ge; participants made a mean of 139 saccades (SD 34) in response to back-stepped targets. Regression analysis showed no correlation between either Ge or the ability to adapt (ie, the dependent variables) with age, gender, or the number of adaptive saccades.

Relationship Between the Ability to Adapt and Baseline Gain

When participants were split into adapting and nonadapting groups, based on significant amplitude gain reduction from baseline amplitude gain at the end of the adaptation phase, baseline gain did not differ significantly between the adapting and nonadapting groups (Table 3).

Table 1. Mean Percentage Change (Standard Error) in Saccadic Amplitude Gain and Latency

End of adaptive phase	
All participants	
Number of participants	39
Mean % gain change (Ge)	−9.3 (1.2)
Mean % latency change (Le)	7.8 (1.6)
Subgroup of participants who adapted*	
Number of participants	26
Mean % gain change (Ge)	−13.3 (0.7)
Mean % latency change (Le)	7.0 (2.2)
Postadaptive phase	
All participants	
Number of participants	39
Mean % gain change (Gp)	−5.5 (1.2)
Mean % latency change (Lp)	5.5 (1.5)
Subgroup of participants who adapted*	
Number of participants	20
Mean % gain change (Gp)	−10.6 (1.0)
Mean % latency change (Lp)	5.2 (2.3)

Mean percentage reduction in saccadic amplitude gain from mean baseline saccadic gain at the end of the adaptation phase is denoted as Ge and the postadaptation phase as Gp. Mean percentage increase from mean baseline saccadic latency at the end of the adaptation phase is denoted as Le and the postadaptation phase as Lp.
*These participants achieved a significant gain reduction from their baseline gain on *t*-tests ($P < .05$).

Saccadic Adaptation and Fatigue

Saccadic amplitude gain in the postadaptation task (G3) was significantly higher ($P < .001$) than amplitude gain at the end of the adaptation task (G2) (see Figure 3a). This indicates that the saccadic amplitude reduction during adaptation was not caused by fatigue or that the influence of fatigue is smaller than that of adaptation.

DISCUSSION

Recording eye movements in children is challenging. In this study, a large cohort of typically developing children were able to complete the study using a noninvasive and well-tolerated eye tracker. We found that saccadic adaptation occurs in children. Human adults and monkeys show that decreases in saccadic gain reach up to 60% of the ideal saccadic gain reduction using experimentally induced hypometria and following 200 to 400 saccades to targets with back-steps in humans[2,17] or several hundred more such targets in monkeys.[20] The magnitude of saccadic gain reduction in children in this study was 53% of the ideal saccadic gain reduction. This is comparable to data for adults. However, direct comparison with adult data is not feasible because experimental designs vary among studies. One study reported an average saccadic gain reduction that reached 49% of the ideal saccadic gain reduction in seven children of unspecified age.[21] No effects of gender or of different ages were identified in our 39 participants.

Participants attained significantly lower saccadic amplitude gains and significantly increased saccadic latency at the end of the adaptation and postadaptation phases. Increased latencies were a consistent feature at the end of both the adaptation and the postadaptation phases. Saccadic latencies are known to be shorter for smaller-amplitude targets.[28] In contrast, reduced saccadic amplitude after adaptation was accompanied by longer saccadic latencies in this investigation. Increased latency of adaptive saccades has also been noted in some monkeys.[20] However, no change in saccadic latencies with adaptation was found in a study in human adults.[7]

The spatial accuracy of the eye tracker of 0.5 degrees could produce errors in saccadic amplitude measurements. This was overcome by planning a relatively large target back-step of 3 degrees, which increased the chance for a larger saccadic amplitude reduction to occur during adaptation that would be clearly detected above the noise level of the eye tracker while still ensuring that the back-step was small enough not to be seen by the participants. Saccadic amplitude reduction did not reach statistical significance in some participants. This could be due to day-to-day variability in saccadic adaptation, as suggested in one study,[25] or the spatial accuracy of the eye tracker, which could have masked a subtle but significant decrease in saccadic gain. Planning a larger number of adaptable saccades and varying the size of the back-step might have increased the amount of gain reduction and the percentage of children who adapt.

A third of the participants who adapted increased their saccadic amplitude in the brief postadaptation phase, indicating the rapid reversibility of the adaptation process in children. Gain

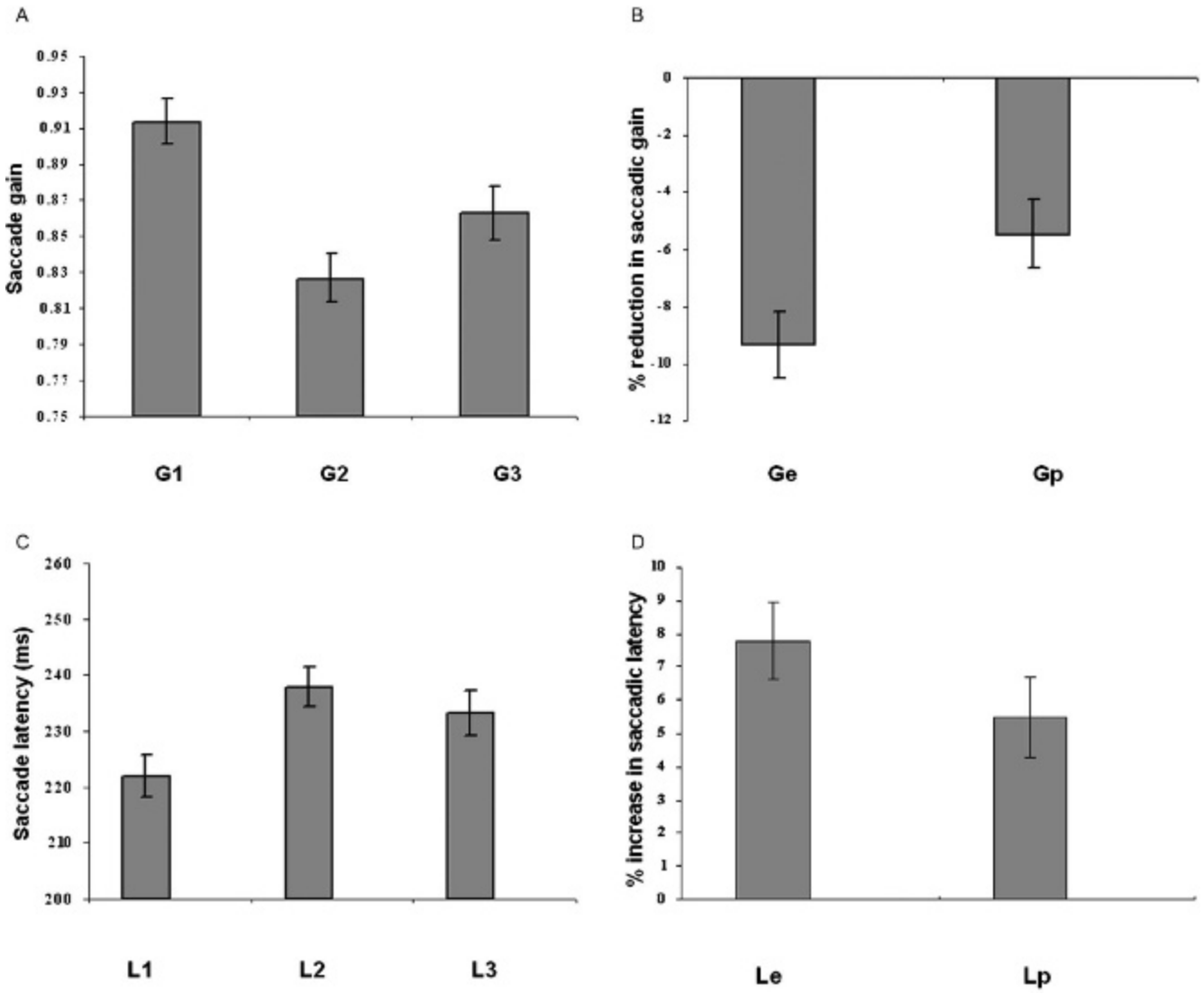

Figure 3. Changes in mean saccadic parameters following the adaptive task. *A*, Mean saccadic amplitude gain (± 1 standard error) at baseline (G1), the end of adaptation (G2), and postadaptation (G3) phases. *B*, Mean percentage reduction in saccadic amplitude gain from mean baseline saccadic amplitude gain (±1 standard error) at the end of the adaptation (Ge) and postadaptation (Gp) phases. *C*, Mean saccadic latency (±1 standard error) at baseline (L1), the end of adaptation (L2), and postadaptation (L3) phases. *D*, Mean percentage increase from mean baseline saccadic latency (±1 standard error) at the end of the adaptation (Le) and postadaptation (Lp) phases.

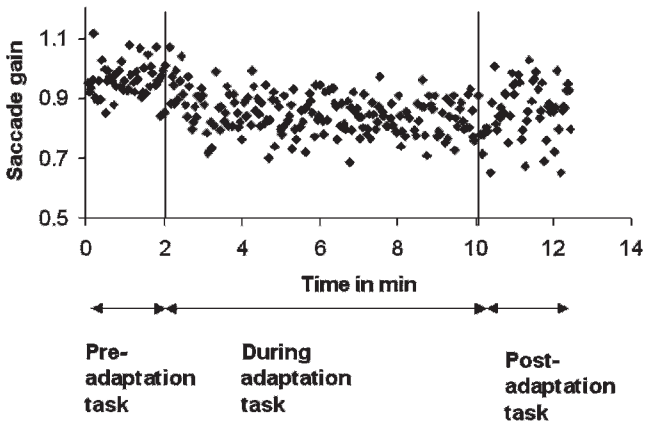

Figure 4. Time course of saccadic adaptation in one participant.

reductions can endure in complete darkness following adaptation in monkeys[20] and humans, although the process is reversible in light.[5,7] In monkeys, a recovery of saccadic size occurs after approximately the same number of saccades that were required for adaptation.[16,20]

The site of saccadic adaptation is downstream from the superior colliculus because saccades evoked by electrical stimulation of the primate superior colliculus are adapted.[29] Furthermore, saccade-related activity in the superior colliculus remains appropriate for the saccade that was required to foveate the initial target rather than for the adapted saccade.[30] The cerebellum participates in controlling saccadic amplitude accuracy.[31] Patients with cerebellar disease often exhibit saccadic dysmetria. Inactivation of cerebellar vermis lobules VI and VII, or the oculomotor region of the fastigial nucleus, results in markedly dysmetric saccades in monkeys.[32,33] Cerebellar vermis

Table 2. Mean Saccadic Amplitude Gain and Latency (SD) at Baseline, End of Adaptation, and Postadaptation Phases

Phase		P *Value*†
Baseline		
Number of saccades	37 (9)	
Mean saccadic amplitude gain (G1)	0.91 (0.08)	
Mean saccadic latency, ms (L1)	221.8 (23.4)	
End of adaptation		
Number of adaptive saccades	139 (34)	
Mean saccadic amplitude gain (G2)	0.83 (0.08)	< 0.0001*
Mean saccadic latency, ms (L2)	237.8 (22.4)	< 0.0001*
Postadaptation		
Number of saccades	35 (10)	
Mean saccadic amplitude gain (G3)	0.86 (0.09)	< 0.0001*
Mean saccadic latency, ms (L3)	233.2 (25.1)	0.001*

Mean saccadic amplitude gain at baseline is denoted as G1, end of adaptation as G2, and postadaptation as G3. Mean saccadic latency at baseline is denoted as L1, end of adaptation as L2, and postadaptation as L3.
*Denotes significant change from baseline.
†On paired Student *t*-test in comparison with baseline values.

lobules VI and VII and the caudal part of the fastigial nucleus in the cerebellum are involved in saccadic adaptation.[34–36] Positron emission tomography (PET) studies in humans show an increase in blood flow in vermis lobules VI and VII during saccadic adaptation.[34,37] Ablation of lobules VI and VII in monkeys[32,33] and lateral medullary infarcts in humans abolish saccadic adaptation.[38]

The results of the present investigation provide evidence that the function of neural structures that participate in saccadic adaptation, a form of motor learning, is established in children and adolescents.

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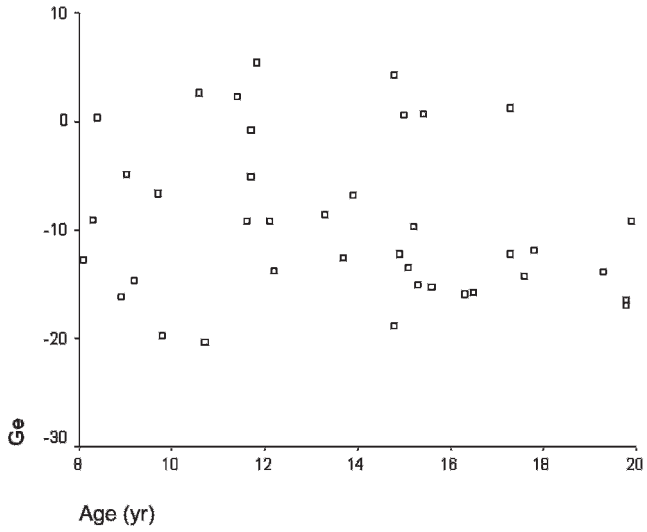

Figure 5. Percentage change in saccadic gain at the end of the adaptive task (Ge) and age. The magnitude of saccadic adaptation did not correlate with age.

Table 3. Mean Saccadic Amplitude Gain, G1 (SD) at Baseline, and the Ability to Adapt

Group	n	*G1*	P *Value*
Adapting	26	0.92 (0.06)	
Nonadapting	13	0.89 (0.1)	.362

Baseline gain did not differ significantly between the adapting and nonadapting groups.

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