Adaptation of Reactive Saccades in Normal Children

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PURPOSE. To compare the amount, the retention, and the extinction of saccadic adaptation in two groups: 9 adults (23–36 years old) and 9 children (11–14 years old).

METHODS. The paradigm used was a classical double-step target to elicit the shortening of saccade gains in response to a 2° backward step (20% of target eccentricity). Two conditions were run in the pre- and postadaptation phases without and with postsaccadic visual feedback, to allow examination of the retention and the extinction of saccadic adaptation.

RESULTS. Adaptation of reactive saccades occurred in children as well as adults. Both groups showed a progressive shortening of saccade amplitude and good retention. The main difference concerned the speed of extinction, i.e., return to baseline, which was slower for children.

CONCLUSIONS. Cerebral structures involved in human short-term adaptation of reactive saccades are functional in regard to adaptive shortening of saccade amplitude. Divergent patterns in the extinction of adaptation between children and adults suggested that lengthening of saccade gain is not yet well established in children. Further investigation is needed to clarify whether processes responsible for backward adaptation are mature before those for forward adaptation. (Invest Ophthalmol Vis Sci. 2011;52:4813–4818) DOI:10.1167/iovs.10-6626

Saccades are fast eye movements used to rapidly change the direction of fixation. Under normal conditions, human subjects make accurate saccades, with a small systematic undershoot.1 Accuracy is assessed by the baseline gain, the ratio between saccade amplitude and target eccentricity (usually around 0.90).2 A high degree of saccade accuracy is maintained throughout the lifetime by the central nervous system via adaptive mechanisms that are able to override diseases, pathologic processes, and physiological changes in mechanical properties of the oculomotor plant due to development, aging, or injury. Saccade adaptation was first reported in patients with abducens palsies.3 In the laboratory, saccade adaptation has been studied using the so-called double-step target paradigm introduced by McLaughlin.4 The adaptation of saccades with a given vector is easily obtained due to (1) the target’s step during the execution of saccades toward it and (2) the repetition of this step over several trials. The iterative backward or forward intrasaccadic stepping of a saccade target induces a progressive gain decrease (smaller saccades) or increase (larger saccades). Since this paradigm was introduced, it has been used by many researchers to explore saccadic adaptation capabilities in humans as well as in monkeys to reveal the neural substrate(s) of saccadic adaptation mechanisms.5 Recent studies have presented evidence suggesting that the cerebellar oculomotor vermis is a critical site of saccadic adaptation.6,7 Cortical areas such as the parietal cortex and the frontal/prefrontal cortex are also involved in the saccade adaptation neural network.5

The examination of saccadic adaptation in children may help to identify and characterize the neural networks involved, given that the cortical structures involved in saccade execution are still developing during childhood. Indeed, an fMRI study8 that investigated the brain activity of subjects between 8 and 30 years old during saccadic tasks showed that the related activation of frontal, parietal, striatal, and thalamic regions increases progressively with children’s age. The maturation of the brain is also known to be completed during adolescence.9

The goal of the present study was to examine the characteristics of saccadic adaptation in children, which is little documented. In the one study on this topic, the adaptation of reactive saccades was explored in a group of 39 children aged from 8 to 19 years. The children made rightward horizontal saccades to a target that began at 12° of eccentricity and stepped backward 3° during the saccade, for 200 trials.10 Twenty-six of them showed a gain decrease. By comparing the pre- and postadaptation phases, both with visual feedback from the target without the step, full extinction of adaptation was observed in six of the children who adapted. No correlation with age or sex was found, and the authors concluded that the neural substrates of mechanisms involved in saccadic adaptation are already functional in 8-year-old children.

Two other studies dealing with backward adaptation of 10° reactive saccades have been conducted in children with neurologic diseases such as dancing eye syndrome11 and Williams-Beuren syndrome.12 Their adaptive capabilities were compared with normal children. In both pathologies, saccade accuracy is poor, most likely as a consequence of cerebellar impairment.5 However, in spite of their saccadic dysmetria, all except one of the children with dancing eye syndrome (out of 7) showed an adaptive modification of gain after a 2° step, with no return to baseline during the 25 postadaptation trials.11 In the other study,12 24 patients with Williams-Beuren syndrome (10–26 years old) were compared with nine controls (13 to 30 years old). After a baseline phase, a 3° step was used for 80 trials. Both patients and controls showed backward adaptation, but unfortunately no postadaptation phase was run in this study.

In the above-mentioned studies, no direct comparisons with adults were done, and their main focus was determining whether backward adaptation occurs, without assessing retention or extinction. Nevertheless, retention is a strong way to assess whether adaptive sensory-motor mechanisms have been engaged1 and requires measuring whether the saccade gain change persists after adaptation in trials without visual feedback.
Here we sought to take these criticisms into account. The backward adaptation of reactive saccades (10° rightward horizontal, step 2°) was investigated in two groups: one of nine normal children (11–14 years old) and one of nine young adults (24–37 years old). The novelty of our design was to test the retention and extinction of the shortening of saccade amplitude created by the 120 adaptation trials. This was done by comparing pre- and postadaptation trials where the target was either extinguished during saccade execution or left illuminated.

**METHODS**

**Subjects**

Nine children between 11 and 14 years of age and nine young adults participated (see Table 1 for individual characteristics). All (except c6) had normal binocular vision (60 seconds of arc or better), evaluated with the TNO (test of Netherlands Organisation) for stereoscopic depth discrimination. This test is based on random dot stereograms that contain a distribution of binocular disparity; it uses retinal disparities ranging from 15 to 480 seconds of arc. Visual acuity of both eyes was normal (≥20/25) for all subjects. The study adhered to the principles of the Declaration of Helsinki and was approved by our Institutional Human Experimentation Committee. Informed consent was obtained from the children’s parents after an explanation of the experimental procedure. Three children and three adults (two authors) were familiar with eye movement recording. Other participants were trained with 20 trials identical with the first 20 preadaptation trials.

**Stimuli**

Stimuli were 0.4° × 0.4° white squares on a medium gray background. The fixation stimulus was located left of screen center (−6°) and equidistant from the top and bottom. Saccade targets appeared 10° to the right of the fixation point.

**Instruments and Eye Movement Recording**

The experimental sessions took place in a dimly lit room. Subjects were seated 57 cm away from the screen, and their heads were kept stable with a submaxillar dental print and forehead rest. The stimuli were presented on an monitor (Iiyama HM240DT; Iiyama, Nagano, Japan) with a refresh rate of 170 Hz and a resolution of 600 × 800 pixels. Eye movements were monitored by an infrared tracker (Bouis Oculomotor; Karlsruhe, Germany) with an absolute resolution of 0.1° of visual angle and linear output over 12°. Viewing was binocular, but only the movements of the right eye were monitored and calibrated. The signal from the oculometer was sampled every 2 ms. Saccade onset was detected online with an in-house program (using LabView 7.1; National Instruments, Austin, TX) based on velocity (>40°/sec), acceleration (>3000°/sec/sec), and minimal displacement (0.15°) thresholds. After saccade onset detection, offset was defined as the moment the velocity fell below 15 deg/second. During trials in which the target changed during saccade execution, that is, extinction or step, the change was initiated after the detection of saccade onset, and occurred with a maximum delay of 16 ms, in the first part of the saccade.

**Procedure**

Each session began with a full calibration procedure during which subjects had to make a saccade to five bars presented successively from left (−6°) to right (+6°) of the screen center in steps of 3°. If the variability of signaled eye position at each bar did not exceed a threshold (0.4 volts, approximately 0.1°) and if they were linear, the calibration was considered successful and the experiment started. Each experimental trial started with a calibration check. A bar appeared on the left of the screen, and subjects were required to fixate on it. If the recorded value was different from that in full calibration by more than ± 0.1°, the calibration was repeated. Otherwise, the left calibration bar was extinguished and replaced by the fixation square. The fixation square disappeared after a random duration (800–1200 ms in steps of 20 ms), and the saccade target appeared (gap-0 paradigm). Subjects were instructed to saccade toward it as quickly and accurately as possible. Once subjects had made their saccade, they were able to blink freely for at least 2000 ms before pushing on a button to initiate the next trial. The target was available for 2000 ms in trials using postsaccadic visual feedback, except in noVF trials (see below), in which the target was turned off during saccade execution and was not presented throughout the rest of the trial. Thus, the screen was blank after target extinction for a variable delay, depending on the moment at which the button was pressed (with a minimum delay of 500 ms for noVF trials).

The experimental session (240 trials) was composed of three stages (preadaptation, adaptation, and postadaptation) organized as follows: two preadaptation phases (with and without postsaccadic visual feedback, respectively, pre-VF and pre-noVF), the adaptation phase, and

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**Table 1. Characteristics of Child and Adult Participants**

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<tr>
<th>Children</th>
<th>Sex</th>
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F, female; LE, left eye; M, male; RE, right eye; TNO, test of Netherlands Organisation.
experimental phases: Two baselines during the preadaptation phase (VF and n°VF, with and without visual feedback of the target), the beginning and the end of the adaptation phase (Beg and End), and two conditions during the postadaptation phase (n°VF and VF, without and with visual feedback of the target). Error bars, standard deviation.

We ran a $2 \times 6$ ANOVA including group (children versus adults) and phase (pre- and post-VF, pre- and post-n°VF, beginning and end of adaptation) as factors. The first 20 trials of the adaptation phase were used for the “beginning of adaptation,” and the last 20 trials were used for both the “end of adaptation” and “post-VF,” to have the same number of observations in all the conditions tested. Latencies did not differ between adults (169 ± 22 ms) and children (170 ± 40 ms) and did not depend on phase (all $P > 0.6$). Thus, latencies remained stable throughout the session (Fig. 1). As expected, saccades were accurate; the average saccadic gains in both baseline conditions (i.e., trials with and without visual feedback of the target after saccade execution during the preadaptation phase) being similar for adults (0.92 ± 0.04 and 0.91 ± 0.07) and children (0.95 ± 0.07 and 0.92 ± 0.05) (all $P > 0.4$). Nevertheless, the intrasubject variability was higher in children than in adults for the two saccade parameters, latency ($P < 0.02$) and gain ($P < 0.005$). This difference remained stable throughout the session and did not depend on other factors.

Saccadic adaptation occurred in all the participants, as shown by progressively decreasing saccade gain over the adaptation phase (see Fig. 2 for two examples). To assess the amount of saccadic adaptation, the change in saccade gain at the end of the adaptation phase relative to the preadaptation phase was calculated for each participant (formula = (VF preadaptation gain – End. Adaptation gain) /VF preadaptation gain). As shown in Figure 3A, all subjects showed a significant decrease in saccade gain at the end of the adaptation phase relative to the preadaptation phase, as shown by the comparison performed at the individual level using a two-tailed Student’s $t$-test (all $t$-tests < 0.05). The average amount of gain change at the end of the adaptation phase was 15.7 ± 4.8% and 12.7 ± 5.9% for adults and children, respectively ($P > 0.25$). These values indicate that at the end of the adaptation phase (the 20 last trials over 120 trials with a target step of $-2^\circ$), gain had decreased in all subjects. Note that the shortening in saccade gain was significant well before the end of the adaptation phase in all participants when the intermediate period (adaptation trials 40 to 60) were compared with pre-VF baseline (all individual $t$-tests < 0.05) resulting in the average amounts of gain change of 12.5 ± 6% and 9.7 ± 6% for adults and children, respectively ($P > 0.20$). The saccade gain continued to shorten until the end of the adaptation phase (trials 100–120), but the statistical difference with intermediate adaptation trials was found in only some participants (a1, a3, a4, a8, a9, and c2, c6, c8; individual $t$-tests < 0.05). This analysis suggests different speed of saccadic adaptation among participants, with a level of adaptation reached more or less early during the phase.
To assess the retention of saccadic adaptation, the pre- and postadaptation phases without postsaccadic feedback visual target were compared. All participants showed a significant shortening in saccade gain in the posttest relative to the baseline (all individual t-tests < 0.05). The average amount of gain change [formula = (noVF preadaptation gain – no VF postadaptation gain) / no VF preadaptation gain] was 10.2 ± 4.7% and 11.9 ± 4.0% for adults and children, respectively (P > 0.4) (Fig. 3B). Interestingly, the amount of retention was smaller than the amount of adaptation (P < 0.04). Examination of Figures 3A and 3B suggests that this difference occurred more frequently in adults, but this interaction failed to reach significance (P > 0.10). This may be explained by the difference of baselines and the larger variability found in children. More importantly, these analyses indicate that the saccadic adaptation induced by the target step was maintained in subsequent trials in which no feedback from the target was given after saccade execution.

Finally, to examine the extinction of saccadic adaptation, defined as the return to baseline gain, the gain change at the end of the postadaptation phase was calculated relative to pre-VF baseline. In the group of children, eight of nine showed a shorter saccade gain at the end of the postadaptation phase relative to pre-VF baseline. In the group of adults, five of nine (a1, a2, a4, a5, a7) showed a full extinction of adaptation, signaled by the return to baseline gain, individual t-tests < 0.05. Only one child (c4) showed full extinction of adaptation, signaled by gain increasing. Except where indicated by “ns,” tested saccade gains differed from baseline at individual level (t-tests, P < 0.05).

**FIGURE 3.** Individual (gray bars) and average (hatched bars) amount of gain change (in percentages) relative to baseline for adults and children (left and right panels, respectively). Relative to baseline, a value of 0% indicates no change in saccade gain, while a positive or negative value indicates decreasing or increasing gain respectively. (A) Amount of adaptation calculated as the saccade gain of the last 20 trials of the adaptation phase relative to the preadaptation phase without visual feedback. (B) Amount of retention calculated as the saccade gain of the last 20 trials of the postadaptation phase relative to preadaptation phase, both with visual feedback from the target. (C) Amount of extinction calculated as the saccade gain of the last 20 trials of the postadaptation phase relative to the last 20 saccades of the adaptation phase. This analysis allows measurement of the beginning of extinction, signaled by gain increasing. Except where indicated by “ns,” tested saccade gains differed from baseline at individual level (t-tests, P < 0.05).
children (5.3 ± 5.6% and 6.4 ± 5.5%; \( P > 0.6 \)). Because individual analyses suggested a difference in the extinction of adaptation between children and adults, this pointed to the fact that extinction was not complete at the end of the postadaptation phase relative to the baseline for most of the children. Are their saccade gains at the end of the postadaptation phase different from those at the end of the adaptation phase? At the individual level (\( t \)-tests < 0.05), five of the eight children who did not show the extinction of adaptation (no return to baseline) had a greater saccade gain at the end of the postadaptation phase than at the end of the adaptation phase (Fig. 3D, right panel). This extinction was starting in almost all adults in the study, as shown in Figure 3D (left panel). On average, the difference between the two groups did not reach significance (\( P > 0.09 \)).

Thus, for adults as well as for children, saccade gains shortened with target step trials, an adaptation that persisted over trials where no visual feedback from the target was given. However, eight of nine adults showed a significant shortening of saccade gain at the end of the postadaptation phase relative to the end of the adaptation phase, with a complete return to baseline in five adults.

In contrast, only six of the children showed a significant shortening of saccade gain at the end of the postadaptation phase relative to the end of the adaptation phase, and only one child returned to baseline. The time course of gain change during the post-VF phase appeared different from that during the adaptation phase. Indeed, recall that six children reached a saccade gain shortening stable after 60 adaptation trials, but the same number of trials during the post-VF phase was not enough to return baseline, except for one child. These individual analyses suggest that even though children showed a trend toward disadaptation, the speed of saccade gain shortening in children may be different from that of gain shortening.

**DISCUSSION**

With a double-step target procedure, we examined the backward adaptation of reactive saccades in children. Compared with adults, children showed similar saccadic adaptation and retention, but differed regarding the extinction of saccadic adaptation.

The saccadic parameters recorded during the preadaptation phase, that is, baseline, are similar to those observed in previous reports in children between 11 and 14 years old. Here both children and adults presented similar latencies for reactive saccades of around 170 ms. As mentioned in the Introduction, during adolescence the brain is still maturing. Oculomotor characteristics, particularly latency, develop throughout childhood, stabilizing around the second decade of human life.15–20 Our study also showed that saccade accuracy in 11- to 14-year-old children was good, and similar to the values reported in adults. Reflexive saccades have been observed to be quite accurate in humans from 5 to 50 years old in several other studies.50,23–25

During the adaptation phase, both children and adults showed an adaptive change of saccade gain as saccade amplitudes shortened progressively in the course of the adaptation phase. After 60 step trials, all participants showed a significant gain shortening, and at the end of the adaptation phase, the reduction of saccade gain was 15.7% in adults and 12.7% in children, corresponding to a target step compensation of around 78% and 64%, respectively. Because of the high probability of the target appearing at a certain location in our procedure, gain changes could result from a cognitive strategy. The question of the nature of gain changes with the double-step procedure—sensorimotor plasticity or strategic adjustment—is a recurrent and important one. Although this hypothesis cannot be totally ruled out here, the stability of latencies across conditions and progressive changes in gain seem to speak against a cognitive hypothesis. Our data support previous findings on the adaptive capabilities of children.10–12

However, a stronger way to assess adaptive change is to examine the retention of gain change by comparing saccade gain before and after the adaptation phase in trials without feedback from a visual target.5 Using such trials, we demonstrated good retention of adaptive change gain in children as well as adults, even though saccade gains in this situation were usually smaller than those observed at the end of the adaptation phase. According to Pelisson et al.,5 retention “reflects the true saccade adaptation resulting from plasticity mechanisms.” Here we show that mechanisms of reactive saccade gain decrease are functional in children 11 to 14 years old, as found for adults here and in previous studies.22,23

The main difference between children and adults that we found concerns the extinction of saccadic adaptation. By comparing trials in which the target did not step, before and after the adaptation phase, it is possible to assess how fully saccades return to normal after the end of step-induced adaptation. A complete return of saccade gains to baseline corresponds to full extinction of saccadic adaptation. This was the case for five adults but only one child. That means that 60 trials were not enough to return to the baseline gain level for almost all children and for some adults. However, extinction had started in most participants, as shown by the comparison to the end of the adaptation phase. The comparison of saccade gain changes after the same number of trials during the adaptation and post-VF phases suggests a difference between saccade gain decrease and increase, in particular for children. Interestingly, such difference is similar to the well-known difference in gain changes observed when backward and forward adaptations are induced by target steps: The speed and amount of gain change are higher in backward than in forward adaptation.24,25 This similarity leads to the possibility of viewing the extinction of backward adaptation as simply a case of forward adaptation.24,25 These behavioral differences could then result from two separate mechanisms of short-term adaptation, responsible for gain decrease and increase.26,27 This raises the question of whether distinct neural substrates underlie the two mechanisms.27,28 The difference between children and adults we found is compatible with the speculation that these neural structures mature with different time courses. However, our study was not designed to investigate this issue. Indeed, saccade gain lengthening was not induced by a forward target step but occurred in a postadaptation phase where the target backward step stopped. Moreover, because the saccade gain lengthening occurred after the gain shortening, we could not eliminate the possibility that fatigue was responsible for the fact that gain lengthening took longer to return to baseline, in particular for children. Unfortunately, we did not examine the effect of time (i.e., trial number) on saccade gain with sessions in which no target step ever occurred. Nevertheless, the stability in saccade latency throughout the complete session that we observed for both children and adults argues against the fatigue hypothesis.1,29 Interestingly, children showed higher intrasubject variability than adults for saccadic parameters such as gain and latency. This difference was constant throughout the experiment, however, suggesting that this effect is not explained by fatigue, but may be a subtle sign of immaturity of the saccadic system.

In conclusion, our individual and group analyses provide a description of the backward adaptation of reactive saccades in children between 11 and 14 years old. Adaptation and retention occurred in all children, with a pattern similar to that of adults. Interestingly, however, individual tests revealed that...
Acknowledgments
The authors thank the participants, in particular the children and their parents for their time and enthusiasm; Laure Delpierre, Eric Orrilos, and Sylvain Haupert for their assistance; and two anonymous reviewers for their helpful comments.

References