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Retention of Saccadic Adaptation in Humans

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ABSTRACT: AU: Please supply a short abstract of 2 or 3 sentences

KEYWORDS: eye movement; plasticity; retention; saccade metrics; saccade dynamics

INTRODUCTION

The accuracy of saccades directed to a visual target is maintained throughout life by adaptive mechanisms. Thus, any internal or environmental factor that compromises the accuracy of visually guided saccades stimulates adaptive mechanisms that progressively adjust saccade amplitude relative to target eccentricity (gain). Numerous studies have analyzed the functional properties and the neuroanatomical substrate of saccadic adaptation in humans and monkeys,¹ but to our knowledge no one has tested how long the adaptive changes last. The aim of the present study was to test the retention of saccadic adaptation induced by the double-step target protocol. We thus measured gain at different times after adaptation, and further investigated whether adaptation also modified saccade dynamics and affected leftward and rightward saccades by the same amount.

METHODS

Five human subjects, sitting in a dimly illuminated room with their head stabilized by a chinrest, looked at visual targets (light-emitting diodes (LEDs)) embedded in a concave spherical board centered on the subjects' cyclopean eye at a distance of 1.1 m. Targets were presented along the horizontal axis every 2.5° from -27.5° to +27.5° with respect to the board center. During the adaptation session (544 trials) divided into 10 successive blocks of trials (ada to adj), a fixation point was initially presented at a random location. It was then turned off and simultaneously a peripheral target appeared to the right or to the left. The size of this initial target step was 10° or 20° (blocks ada, adb), 7.5° or 15° or 22.5° (adc, add), 12.5° or 25° (ade to adj).

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During the saccade, the target stepped backward by 25% (ada, adb), 33% (adc, add), and 40% (ade to adj) relative to the initial target jump. The final position of the target became the initial position for the next trial. This adaptation session was preceded by a pretest session (40 trials) in which four single target steps ($\pm 10^\circ$ and $\pm 20^\circ$) were presented randomly, all initiated from the center of the board. The peripheral target was turned off at the start of the saccade. The same test session followed adaptation (= posttest) at different times: on the same day (day 0) after five adaptation blocks (ada to ade) (D0a) and after the last five blocks (adf to adj) (D0b), on day 1 (D1), day 5 (D5), day 11 (D11), and day 19 (D19).

Eye movements were recorded by the EyeLink system (SMI, Germany) at a frequency of 250 Hz. The intrasaccadic displacement or extinction of the target was triggered on the basis of a 50 deg/s velocity threshold by a PC program (DataWave, USA) that also stored the eye signals on disk. Primary saccade parameters were analyzed off-line based on a 40 deg/s velocity threshold detection.

RESULTS AND DISCUSSION

FIGURE 1 depicts the time course of the average saccade gain (rightward and leftward data pooled together) as a function of the test recording session for the five subjects (black trace). A one-way repeated measures ANOVA showed a significant main effect of the factor "test session" ($P < .001$). The gain that was very close to 1 in pre-

FIGURE 1. Time course of mean saccade gain as a function of the test session for the five tested subjects and for the control subject (*black and gray traces*). Error bars are SD. Statistically significant differences of saccade gain between posttest and pretest are shown by * ($P < .05$) or *** ($P < .001$).

FIGURE 2. Time course of mean saccade gain as a function of adaptation blocks of trials (ada to adj), separately for rightward and leftward saccades (black and gray symbols). Error bars are SD. Statistically significant differences of saccade gain between the two directions are shown by * ($P < .05$), ** ($P < .01$), or *** ($P < .001$); NS: nonsignificant.

test was significantly decreased by $15.1 \pm 3.3\%$ after the first five adaptation blocks, and was further reduced after the last adaptation blocks up to $22 \pm 4.7\%$. On day 1, saccades remained adapted with subjects exhibiting a mean amount of retention of $36 \pm 17\%$ relative to D0 gain reduction. Saccades were still hypometric on D5, with an amount of retention of $19.7 \pm 13.3\%$. Saccade gain returned to the pre-test value at D11. *Post hoc* LSD Fisher tests indicated that only the gains on D11 and D19 were equivalent to the pretest gain ($P > .05$). The control data (gray trace) collected in one subject who underwent the same number of trials, but without intrasaccadic target step, revealed no change in saccade gain after this “pseudoadaptation” session except, unexpectedly, for D1 and D5 ($P < .05$; *t*-test for independent samples). This suggests that the decrease in saccade amplitude induced in our double-step target protocol was the result of adaptation and not of fatigue.

When we considered the direction of the saccades, we observed that the gain of leftward saccades was significantly smaller than that of rightward saccades for all days except for the pretest. We thus compared the time course of adaptation for the two saccade directions by plotting the saccade gain versus adaptation block relationships (FIG. 2). A two-way repeated measures ANOVA (saccade direction $\times$ adaptation block) and *post hoc* LSD Fisher tests revealed that the two gains were similar during the first two blocks, but that from the third block until the end of adaptation, the gain of leftward saccades exhibited a significantly larger change than rightward saccades. This suggests that adaptation of leftward saccades can be stronger than ad-

FIGURE 3. Saccade main sequence. (A) Duration or (B) peak velocity as a function of saccade amplitude in pretest and in posttest D0b (*open and filled symbols*) for the five tested subjects.

adaptation of rightward saccades. In follow-up analyses, it would be interesting to relate such right-left asymmetry to the manual preference of each subject.

Finally, we examined whether adaptation also modified saccade dynamics. Adaptation led to a decrease of both saccade duration and peak velocity. However, these modifications could be accounted for by the decrease of saccade gain. Indeed, the main sequence relationships between duration or peak velocity and amplitude of saccades were not affected by our saccadic adaptation protocol (Fig. 3).

In conclusion, our study suggests that in humans, adaptive mechanisms induce long-lasting changes in visually guided saccade amplitude, probably reflecting plastic changes in the brain. However, this conclusion is challenged by the observation, made in a single subject, of day-to-day fluctuations of the saccade gain independent-

ly of any adaptation-related gain changes (control experiment). Insofar as these unexpected gain fluctuations do not consistently follow the pseudoadaptation session (possibility currently tested in other subjects), our results would also indicate that the numerous, mostly volitional saccades generated by our subjects between the test sessions do not completely de-adapt the reactive saccades tested in our study, supporting the hypothesis that adaptation of these two saccade types relies on partly separated mechanisms (see Ref. 2, for example).

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