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ORIGINAL PAPER

Instability of visual error processing for sensorimotor adaptation in schizophrenia

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Abstract Saccadic adaptation can be used to study disturbances of sensory processing and motor learning. We investigated whether patients with schizophrenia can adjust saccadic amplitudes to account for an increase in visual error while the saccade is in flight, and whether they transfer this change to a visuo-manual localization task. Fourteen patients (mean 37.1 years) and 14 healthy controls (mean 35.1 years) performed 200 adaptation trials of 10° with target shifts of 4° in the outward direction. We determined the percent amplitude change during adaptation and adaptation speed. In addition, subjects localized a stimulus that was flashed 50 ms after saccade target onset to measure the transfer of change in visual space perception to visuo-manual coordination. Eye movements were recorded at 1000 Hz. Saccade amplitudes increased over adaptation trials by 11 % ($p < 0.001$) similarly in both groups. Amplitude variability during adaptation was higher in patients (1.06° ± 0.32°) than in controls (0.71° ± 0.14°; $p = 0.001$), while adaptation speed was slower in patients (0.02 ± 0.03) than in controls (0.11 ± 0.11; $p = 0.01$). Other pre- and post-adaptation saccade metrics did not differ between groups. The adaptation process shifted localization of the flashed target in the adaptation direction similarly in both groups. The use of error signals for the internal recalibration of sensorimotor systems and the transfer of this recalibration to visual space perception appear basically unimpaired in schizophrenia. Higher amplitude variability in patients suggests a certain instability of saccadic control in cerebellar systems. Patients seem to rely on visual error processing in frontal circuitry, resulting in slower adaptation speeds, despite unimpaired adaptation strength.

Keywords Saccades · Amplitude variability · Efference copy · Prediction error · Visuo-manual transfer · Visual space perception

Introduction

Saccadic eye movements enable us to immediately shift the fovea, the part of the retina with the highest visual acuity, to a location of interest. Saccades are generated in a ballistic way, implying that their amplitude has to be calculated before saccadic onset using the predicted landing position of the saccade. The predicted landing position is based not only on the spatial percept of the target, but also includes an efference copy, i.e., an internal copy of the saccade motor command, encoding size and direction for the upcoming saccades [1]. Due to high velocities of up to 900°/s, no visual feedback is available during saccades. Instead, post-saccadic visual information about the difference between eye and target position, and a prediction error derived from the efference copy, is assumed to drive adjustments in sensorimotor control for subsequent saccades [2].

The neural brain systems subserving visually guided saccades have been extensively studied in both humans and non-human primates. This research has shown that saccadic networks are widely distributed, involving subcortical (superior colliculus, brain stem, striatum, thalamus, and

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cerebellum) and cortical areas (primary visual, extrastriate, and parietal cortex, frontal (FEF), as well as supplementary eye fields [3–5]). Efference copies of the oculomotor command are processed by several different neural pathways. One pathway extends from brainstem neurons to cerebellar oculomotor areas, a second path reaches from superior colliculus to FEF [6], while a third connects the cerebellum to FEF [7]. Given this knowledge, it is possible to assess the integrity of sensorimotor circuitry in patients with severe mental disorders, such as schizophrenia, by testing the accuracy and timing of saccades. Most clinical studies on saccades in schizophrenia have examined psychopharmacologically treated patients, showing that saccadic latency, accuracy, and dynamic characteristics are generally preserved. Some studies have reported dysmetric and hypometric saccades, suggesting disturbances in sensorimotor circuitry for saccade generation [8, 9].

The saccade adaptation paradigm enables more specific measures of how visual–spatial information is processed in order to drive motor control and allows one to assess how adequately error signals are used for internal recalibration of sensorimotor systems [10]. In this paradigm, the post-saccadic visual error is artificially increased while the saccade is in flight. Thus, the motor program for saccade execution, generated based on presaccadic target location on the retina, will be inadequate. Over the course of adaptation, the oculomotor system registers the increase in the post-saccadic visual error and consequently prediction error. The saccade behavior is adapted accordingly by increasing or decreasing saccade amplitude, depending on the target shift direction [2, 11]. There is an increasing body of evidence suggesting that the efference copy that drives the prediction error signal also changes during adaptation in accordance with the changes in the oculomotor command induced by the adaptation process [7, 12]. Saccadic amplitudes can be either lengthened or shortened depending on whether the target is moved in an inward or outward direction from the original target [13, 14]. Inward and outward adaptation differs in the mechanisms that operate at various stages of oculomotor transformation. Inward adaptation is more easily achieved, faster, and more efficient than outward adaptation due to the dynamics of the oculomuscular apparatus. Inward adaptation is thought to rely predominantly on cerebellar gain learning mechanisms using the cerebellar efference copy to adjust for small errors in motor performance. In contrast, outward adaptation is thought to induce larger changes in the internal target representation and the motor command. In addition to cerebellar mechanisms, outward adaptation recruits additional neural networks, most notably prefrontal and parietal cortical areas, which process efference copy signals originating from cerebellum and colliculus superior and feeding into FEF [13]. Recent imaging studies support a model of saccade adaptation that takes place at different levels of cortico-cerebellar processing [15, 16].

Two previous studies have investigated saccade adaptation in schizophrenia using an inward adaptation paradigm [17, 18]. Picard and colleagues reported reduced saccade adaptation rates but an unimpaired adaptation time course in 12 medicated patients with schizophrenia who also showed pronounced neurological soft signs [17]. In contrast, Coesmans and colleagues observed unimpaired adaptation rates in 39 patients (13 patients were antipsychotic-free), but found that adaptation speed was slower [18]. The influence of antipsychotic medication on the adaptation process was found to be negligible [18]. Both authors argue that these results likely arise due to cerebellar dysmetria, which reflects disturbances in cortico-subcortical-cerebellar circuitry implicated in schizophrenia [19, 20].

In the present study, we used an outward adaptation paradigm to study whether patients with schizophrenia were able to sufficiently integrate an artificially increased post-saccadic error in order to remap and recalibrate oculomotor commands, resulting in an increase in saccade amplitudes. We also conducted an additional localization paradigm to investigate the influences of adaptation on trans-saccadic localization. The outward adaptation process should lead to an outward shift in the localization of a target that is briefly presented before the execution of the saccade [21, 22]. It has been suggested that trans-saccadic perception of such a localization target uses the adapted efference copy to remap presaccadic target position to its predicted post-saccadic location [1, 23]. Alternatively, saccade adaptation may involve brain structures that support both visual localization and saccade targeting [22, 24]. Here, we examined whether the transfer of adaptation-induced changes in the motor command to other sensorimotor circuitry is altered in schizophrenia.

Methods

Participants

We included 14 patients from in- and outpatient services of the Department of Psychiatry and Psychotherapy at the University of Muenster who met the DSM-IV criteria for schizophrenia [25]. Diagnoses were determined at consensus conferences using all available clinical data. At the time of testing, patients were on stable antipsychotic medication for at least four weeks. Fourteen healthy control participants all without a history of Axis I disorders according to the Structured Clinical Interview for DSM-IV [26] or any known history of psychotic disorder in first-degree relatives were also included. The two groups did not differ in age,

Table 1 Sociodemographic and clinic characteristics of patients with schizophrenia and health controls

	Patients ($N = 14$)	Healthy controls ($N = 14$)
Age, years (mean ± SD)	37.1 ± 10.1	35.1 ± 13.0
Gender (N male|female)	6|8	8|6
Verbal IQ (mean ± SD)	102.3 ± 17.1	107.6 ± 12.1
Years of education (mean ± SD)	13.4 ± 2.9*	16.3 ± 1.7*
Dominant eye (N left|right)	5|9	4|10
Illness duration, years (mean ± SD)	8.7 ± 9.4	n.a.
Chlorpromazine equivalents**	369.0 ± 208.8	n.a.
Delusions, all types (N current|lifetime)	3|13	0|0
Delusions of alien control (N current|lifetime)	1|4	0|0
Hallucinations (N current|lifetime)	0|5	0|0
Formal thought disorder (N current|lifetime)	1|7	0|0
Negative symptoms (N current|lifetime)	9|10	0|0

* $t_{26} = -3.3$, $p = 0.003$, ** according to Andreasen et al. [28], *n.a.* not applicable

pre-morbid verbal IQ (MWTB, [27]), gender distribution, or dominant eyes. The control group did, however, have more years of education than patients (Table 1).

Exclusion criteria for all participants included: (1) any known systemic or neurological disease or history of head trauma with loss of consciousness >10 min, (2) any ophthalmological disease, (3) regular intake of any tranquilizers, i.e., benzodiazepines, within the two weeks prior to testing or acute intake of such medication within the last 48 h, (3) no substance dependence for at least one year and no substance abuse for at least one month according to DSM-IV criteria, and (4) corrected or uncorrected visual acuity of less than 0.8 tested by Landolt rings. All participants gave written informed consent according to the Declaration of Helsinki and its later amendments. The study was approved by the ethics committee of the University of Muenster.

Testing procedures

Subjects were seated in a darkened room (0.01 cd/m^2) with their head stabilized by a chin rest 59 cm in front of a 18″ computer monitor (36.5 × 27.5 cm corresponding to 34° × 26°, resolution 1240 × 1024 pixel, and refresh rate 100 Hz) that displayed target and localization stimuli. Care was taken to avoid any visual stimulation other than that displayed on the monitor to prevent the use of landmarks or reference for localization. Targets for saccade trials consisted of white circles (0.75°, 52.5 cd/m^2), while localization targets consisted of red vertical bars (3° × 0.3°, 12.5 cd/m^2). Eye movements were recorded by an Eyelink 1000 system (SR Research, Canada) with a sampling rate of 1000 Hz. Viewing was binocular, but only the dominant eye was recorded. A nine-point grid was used for calibration allowing for a deviation of 0.25°. Programs for stimulus generation and eye movement recording were written in MATLAB 2010b (The MathWorks, Natick, MA).

Pre-adaptation saccade trials

Each trial started with a fixation target that appeared at 4° or 6° to the left from the screen center where it remained for a randomized duration of 700–1300 ms. Simultaneous to target offset an eccentric target appeared at 10° to the right where it was visible for 500 ms. We instructed subjects to fixate the fixation target and to make a saccade to the eccentric target as soon as it appeared. The next trial started after 500 ms. We recorded a total of 10 pre-adaptation trials.

Pre-adaptation localization trials

These were identical to pre-adaptation trails, but 50 ms after appearance of the eccentric target, a red localization bar occurred for 20 ms at a distance of ±3° horizontally from the eccentric target keeping a minimum distance of 0.15°. One second after the saccade was executed, a mouse pointer occurred at the lower screen side. We instructed the participants to shift the pointer to the location where they remembered the vertical localization bar and then make a click. Whenever they had missed the localization bar, subjects should make a click at the lower screen edge. These trials were omitted from later analyses. In total, 40 localization trails were presented. In half of these trials, the eccentric target was extinguished as soon as the saccade exceeded 2° from the fixation point so that no spatial reference was visible for the following localization.

Outward adaptation trials

Adaptation trials were identical to pre-adaptation trials with the addition that whenever the saccade exceeded 2° from its starting position, the eccentric target was shifted

4° in outward direction, i.e., to the right, resulting in a total amplitude of 14°. In total, 200 adaptation trials were presented. In addition, 100 extra trials, in which targets appeared 10° above or below the fixation point, were randomly interspersed within the adaptation trials. These additional trials were included in order to reduce predictability of the horizontally presented adaptation trials.

Post-adaptation localization trials

Post-adaptation localization trials were similar to pre-localization trials. As in the adaptation trials, the target was shifted by 4° in an outward direction while the saccade was in flight so as not to attenuate the adaptation effect. Twenty de-adaptation trials followed. These were identical to pre-adaptation trials.

Neurophysiological analyses

Saccades were detected automatically (velocity >30°/s, acceleration >8000°/s^2) and were considered valid when their amplitude exceeded 5° in the rightward direction, corresponding to 50 % of the initial target step. We excluded saccades with latencies <100 ms or >400 ms and checked for blinks, which could have interacted with saccade execution. Outliers were identified on the basis of amplitudes in custom-designed bins (10, 20, 20, 30, 40, 40, and 40 trials, respectively) and excluded from analysis. The ascending number of trials per bin was chosen to permit more variability at the beginning of the adaptation phase, due to the often-reported quick change in the first tens of trials. Toward the end of the adaptation phase, the slightly larger bins provided a better identification of erroneous amplitudes that could occur due to fatigue or loss of alertness. To be identified as an outlier, saccade amplitudes had to be larger than 1.5 times the interquartile range from the outer boarder of the 25 % quantile and the 75 % quantile. This step was necessary to determine a converging exponential fit for each subject. In healthy controls, 6.5 % of saccades could not be validated, while in schizophrenia patients 13.54 % of saccades were excluded from the analyses. Basic saccadic parameters of interest included saccade amplitude and its variability, saccade latency, peak velocity, and saccade duration.

For each subject, we computed the mean percent change across blocks of 20 trials defined by the formula (amplitudes$_{adapt}$ − mean(amplitudes$_{pre}$))/mean(amplitudes$_{pre}$) (Fig. 1). Overall adaptation strength refers to the mean percent amplitude change in the last block, i.e., trials 181–200.

To define adaptation speed, we computed an exponential nonlinear fit for each subject. The baseline of the fit was set to the subject's mean saccadic amplitude prior to the adaptation trials. In all, data from 12/14 patients and 14/14 controls were suitable for the fitting procedure. Adaptation speed was defined as 1/b in the fitting formula $y = a \times (1 - e(-x/b))$ with a indicating the expected amplitude, b indicating the time constant, and x indicating the trial number.

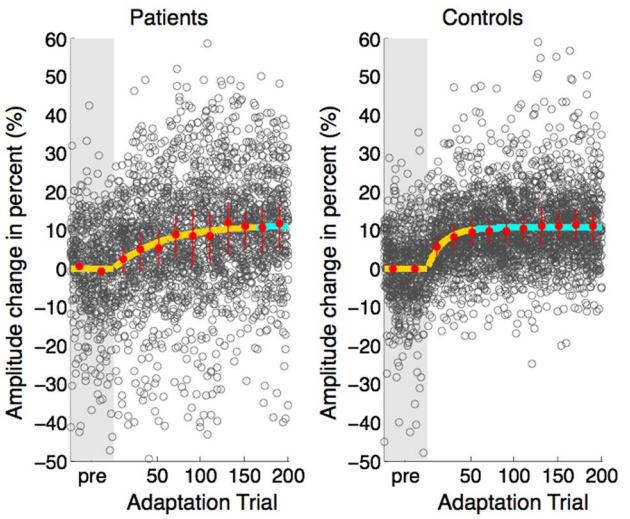

Fig. 1 Amplitude change in percent over the adaptation course of 200 trials as related to the mean amplitude in saccades prior to adaptation given for patients with schizophrenia (*left*) and controls (*right*). The *colored line* illustrates the mean fitting curve for each group. The change from *yellow* to turquoise indicates the point when 95 % of the asymptote of the fit was reached. Additionally, *red dots* indicate the group means of amplitude change with 95 % confidence intervals over blocks of 20 trials

In pre- and post-adaptation localization trials, we defined the absolute localization error as the difference between the positions of the mouse pointer and the localization target for each subject individually. Failures in execution of a localization trial were very rare and did not differ between the groups (patients: 0.0015 %, controls: 0.005 % of all localization trials). We then related the localization error of each post-adaptation localization trial to the mean pre-adaptation localization error of that subject. Mean localization errors were calculated separately for localization trials with and without reference point.

Statistical analyses

Repeated measures ANOVAs were used to compare group differences between pre-adaptation and end of adaptation trials. For the ANOVAs, "time" was included as a within-subjects factor, while "group" was included as a between-subjects factor. Analyses were carried out using the SPSS (version 23) software package (IBM Deutschland GmbH, Ehningen, Germany). For simple group comparisons,

Table 2 Saccade characteristics (means with standard deviation) before and at the end of adaptation (last 20 trials) in patients with schizophrenia ($N = 14$) and healthy controls ($N = 14$)

	Pre-adaptation		End of adaptation	
	Schizophrenia patients	Healthy controls	Schizophrenia patients	Healthy controls
Amplitude (°)	9.02 (0.74)	9.41 (0.60)	9.92 (1.07)	10.43 (0.72)
Latency (ms)	178.17 (35.67)	175.21 (50.25)	177.68 (33.20)	177.12 (37.13)
Peak velocity (°/s)	466.49 (77.23)	435.73 (43.97)	473.26 (77.88)	445.79 (47.10)
Duration (ms)	56.54 (8.07)	53.29 (8.65)	54.83 (16.09)	56.04 (7.48)

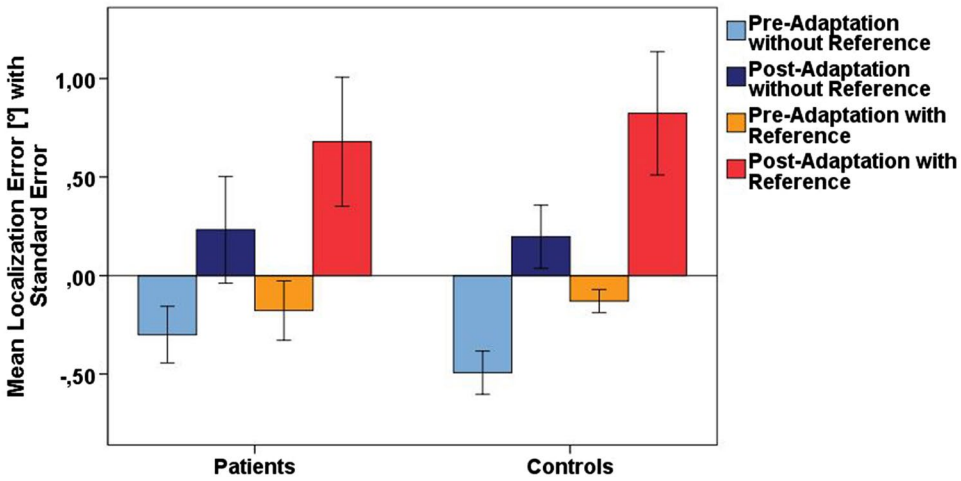

Fig. 2 Mean localization error before and after saccadic outward adaptation in patients with schizophrenia and controls. *Negative values* indicate an underestimation of the target's position, while *positive values* indicate an overestimation in direction of saccade adaptation. Results are shown for trials with (*red colors*) and without a reference point (*blue colors*)

independent sample t-tests were used. Possible effects of clinical variables or medication status on saccadic parameters were evaluated by Pearson correlations.

Results

Saccadic outward adaptation

Saccade amplitudes increased during the adaptation process significantly in both groups (*time*: $F_{1,25} = 47.29$, $p < 0.001$; post hoc: patients $t_{1,13} = -4.01$, $p = 0.001$; and controls: $t_{1,12} = -6.44$, $p < 0.001$) but did not differ between groups, nor was there an interaction of time × group (Table 2). Accordingly, overall adaptation strength from pre-adaptation to end-adaptation trials did not differ between groups (patients 10.14 % ± 9.88; controls 11.10 % ± 6.46) Fig. 1. Adaptation velocity, as defined by 1/*b* in the exponential fitting function, was lower in patients than in controls (patients 0.02 ± 0.03; controls 0.11 ± 0.11, $t_{1,24} = -2.71$, and $p = 0.012$). While controls reached 95 % of the asymptote of the mean fit after only 54 trials, patients needed 164 trials until they reached 95 % of the asymptote of their mean fit.

Amplitude variability was higher in patients than in controls across pre-adaptation and adaptation trials (*pre-adaptation*: patients 0.75 ± 0.54°; controls 0.51 ± 0.15°; *during adaptation:* patients 1.06° ± 0.32; controls 0.71° ± 0.14; and *group:* $F_{1,26} = 8.03$, $p = 0.009$). Amplitude variability increased in both groups during adaptation (*time*: $F_{1,26} = 14.11$, $p = 0.001$). Although the group × time interaction was not significant, exploratory post hoc analyses showed that amplitude variability was considerably larger in patients than in controls during adaptation ($t_{1,26} = 17.80$, $p = 0.001$). Groups did not differ statistically in pre-adaptation trials ($p = 0.13$).

Neither saccade latency, peak velocity, nor duration changed during the adaptation process. There were no group differences or interactions between group and time for these basic saccade parameters (Table 2).

Adaptation-induced mislocalization

Prior to adaptation, participants of both groups underestimated the positions of the localization targets. This occurred in trials both with and without a reference point. Saccadic adaptation induced an outward shift in the localization of the flashed target, resulting in an overestimation of its position after adaptation (*pre–post:* $F_{1,25} = 22.47$, $p < 0.001$) (Fig. 2). Generally, localization errors and their adaptation-induced changes were larger in conditions with than without a reference point (*reference point:* $F_{1,25} = 21.50$, $p < 0.001$ and *reference point* × *pre-post*: $F_{1,25} = 4.44$, $p = 0.045$). Despite these clear effects of outward adaptation on mislocalization, we did not find any differences or interactions between groups (group × reference point, group × pre–post-adaptation, or group × reference

point × pre–post-adaptation). Notably, the adaptation-induced change in mislocalization did not correlate with percent amplitude change or amplitude variability in either group.

Possible associations with demographic and clinical variables

Measures of saccade parameters, including amplitude change and adaptation speed, as well as pre-post localization errors, were not significantly correlated with age, IQ, education, duration of illness, medication status, or current and lifetime symptom expression.

Discussion

Our main results suggest that patients are able to increase saccadic amplitude on a trial-by-trial basis when the target is shifted in an outward direction immediately after saccadic onset. In this regard, adaptation strength in patients did not differ from that in controls. Adaptation speed, however, was considerably slower in the patient group, with patients requiring approximately 100 trials more than controls to reach a final amplitude increase of 10–11 %. Slower adaptation speed in patients was accompanied by higher amplitude variability, especially during adaptation. Both patients and controls were able to transfer the internal target representation to a manual target localization task. This was exhibited by an overestimation of the target location in the outward direction after saccadic adaptation. In both groups, this effect was larger for tasks in which a reference point was presented to support localization. We will discuss our results with regard to their implications for a better understanding of the altered neurophysiological mechanisms that may underlie core disease mechanisms in schizophrenia.

Generally, our findings of unimpaired basic saccade metrics are in line with previous reports from psychopathologically stable patients under antipsychotic medication [8, 9, 29]. Our results are also in line with one of the two previous reports on inward adaptation that showed equal adaptation strength but reduced adaptation speed for both medicated and unmedicated patients with schizophrenia [18]. In accordance with the latter finding, we did not find any evidence for an influence of medication status or clinical variables on either adaptation ability or saccade metrics. Contrary to the current study, Picard and colleagues [17] reported reduced adaptation strength but same adaptation speed in 12 patients with schizophrenia. The differences between the results of Picard et al. [17] and ours could potentially be explained by several factors. Firstly, Picard and colleagues only compared adaptation changes blockwise, but did not apply a fitting approach to determine adaptation speed, as was done in both the current study and by Coesman's group [18]. Employing an exponential function to fit the amplitude development over the course of adaptation supersedes the assignment of trials into arbitrarily sized blocks and allowed us to determine the adaptation speed equally based on all successful trials. Secondly, Picard and colleagues [17] only included 134 adaptation trials for analyses and excluded the last two blocks of their experiment. Although inward adaptation is faster than outward adaptation, the adaptation process in patients of the Picard study may not have been completed after these 134 trials, resulting in lower adaptation strength. Thirdly, the findings of Picard's group may be specifically true for patients who express high levels of neurological soft signs (NSS) reflecting disturbances in cerebro-cerebellar circuits [17, 20]. However, the authors did not compare their findings to patients with low NSS levels. It remains unclear whether patients with low NSS are also impaired regarding adaptation strength.

Patients may rely more strongly on frontal circuitry for adaptation

Our findings extend the previous literature by showing that patients are able to use visuospatial and predictive error signals for modifying the saccadic motor program during the outward adaptation process, which is more challenging than inward adaptation. Besides cerebellar mechanisms, outward adaptation is thought to be dependent on frontal cortical mechanisms, which include the efference copy pathway from cerebellum to FEF. Our findings of unaltered outward adaptation strength in patients suggest that these mechanisms for spatial mapping and coordination are preserved in schizophrenia. If this efference copy pathway was disturbed in patients, oversized amplitudes would have been expected during outward adaptation, as has been reported from a patient with a focal thalamus lesion [30]. This, however, was not the case in our patients.

Though adaptation strength was not affected in the patient group of the current study, they did exhibit higher saccade amplitude variability. This could be interpreted as evidence of a volatility of saccadic control due to unstable efference copy signaling for predictive coding in cerebellar systems. In accordance with this interpretation, increased saccade amplitude variability has been reported for patients with cerebellar atrophy, while mean amplitudes in these patients were not altered, analogous to findings of the current study [31]. Others have concluded impaired use of the efference copy in schizophrenia patients based on a saccadic double-step paradigm, where it was found that patients were slower and less accurate when accounting for the error of the first saccade while executing the second saccade [29]. Similarly to our study, Thakkar and colleagues observed higher amplitude variability in patients.

There are two reasons why unstable predictive coding due to unstable efference copy signaling could have resulted in slower adaptation speed. First, unstable prediction error processing in cerebellar systems would result in an increase in prediction signal noise and, therefore, slower adaptation. Second, an impairment of prediction might have generated higher amplitude variability, resulting in an increase in visual error variability. This would also serve to slow down adaptation, despite frontal compensation processes for increased prediction error coding. Others have argued that disturbances in frontal circuitry produce increased variability of saccade accuracy in schizophrenia patients during memory-guided saccades, especially in patients with negative symptoms [32]. Despite the fact that memory-guided saccades require stronger recruitment of prefrontal areas than our adaptation task, we cannot exclude the possibility that imbalances in frontal circuitry may have contributed to increased amplitude variability in patients. It should be noted, however, that patients who exhibited negative symptoms in the current study (9 of 14) did not show higher amplitude variability than those without negative symptoms, making an account based on frontal circuitry less likely.

Transfer of adaptation change to visuo-manual coordination

Two key findings support our hypothesis that spatial remapping of sensorimotor systems, which include prefrontal–parietal circuitry, is sufficiently intact in patients. Firstly, patients were not impaired in the pre-adaptation localization task. Secondly, they could also transfer the outward shift from the saccade to the localization task similarly to controls. There has been a broad discussion of whether this transfer requires an efference copy of the motor command to the adapted saccade, or whether the adaptation process induces modifications in motor parameters of visual targets in space [22–24]. If, in the latter case, saccade motor vectors are used for visual localization, adaptation-induced mislocalization should also be observable during fixation when no saccade is executed. A recent study in healthy subjects showed that post-adaptation the spatial position of a localization target was shifted even when the subject kept fixating the central fixation point [21]. This suggests that localization changes should be interpreted as a consequence of the saccade adaptation procedure per se and are cannot be explained by changes in motor performance or mismatches between the saccade and its related efference copy signal [21]. Our findings in patients indicate an unimpaired contribution of saccade motor parameters to visual space perception. The finding that localization shifts after adaptation were larger for trials with than without reference point in both groups can be explained by the fact that in post-adaptation trials the reference point, i.e., the target was also shifted in an outward direction in order to avoid attenuation of the motor adaptation effect.

Limitations

There are limitations to this study that need to be considered. First, the small sample size does not allow for further subgroup analyses. There may be additional impairments of saccadic adaptation or associations with symptom expression in patients that we did not detect. Second, we did not assess patients and controls for NSS. We were therefore unable to investigate the relationship between adaptation capability and possible other symptoms of cerebellar alterations. Third, despite the finding of a relative independence of reduced adaptation speed and increased amplitude variability from current medications in both the current study and the previous literature [18], effects of chronic medication treatment are potential confounds on performance measures that cannot be fully excluded.

Conclusions

Despite these limitations, our results add to the complex puzzle of discrete impairments and preserved neurophysiological functions in schizophrenia. Although basic metrics of reflexive saccades and the ability to adapt motor behavior to changes in visual space perception appear intact, adaptation speed slowing and greater instability in oculomotor adjustments may, nonetheless, result in disturbances of visual perception and motor control. Future studies should focus on the relationship between these disturbances and discrete distortions of visual perception that may not fulfill the criteria of core visual hallucinations but are often reported by patients and that may even indicate increased psychosis risk and prodromal symptoms, i.e., brief limited intermittent psychotic symptoms (BLIPS). Future studies should also include voluntary scanning saccades, which have been suggested to rely more strongly on frontal cortical mechanisms subserving sensorimotor adaptation.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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