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Increased oculomotor deficits during target blanking as an indicator of mild traumatic brain injury

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Abstract

Given the susceptibility of cerebellar-cortical tracts to shearing injury from traumatic brain injury (TBI), we investigated impairment in the generation of predictive eye movements and its relationship to cognitive deficits in mild TBI patients using a smooth pursuit target-blanking paradigm. Compared to a target-tracking paradigm without blanking, this paradigm more greatly necessitates the generation of predictive eye movements, which are subserved by brain regions involved in cognitive processing. Mild TBI patients showed impaired prediction of target trajectories during target blanking, demonstrated by generation of saccades at earlier and more variable time points, as well as greater and more variable oculomotor error compared to controls. In addition, California Verbal Learning Test (CVLT-II) scores related to working memory, learning, and executive function were more highly correlated with oculomotor variability during target blanking than during target tracking. Our results suggest that a disruption of cerebellar-cortical connections in TBI may account for both oculomotor and cognitive impairment, and that measures of predictive eye movements during target blanking may be a sensitive metric of cognitive deficits after mild TBI.

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Keywords: Mild TBI; Diffuse axonal injury (DAI); Predictive smooth pursuit eye movement; Variability; Cognitive deficits

Overlapping neural pathways of cerebellar and cortical regions, including the frontal eye fields (FEF), supplementary eye fields (SEF), prefrontal cortex (PFC), and parietal cortex, have been implicated in both predictive smooth pursuit eye movements (SPEM) [24,20,39] and cognitive processes, including attention, working memory, and learning [35,27,19,23], suggesting that these systems may be functionally associated. Supporting this hypothesis, several studies have shown that predictive SPEM is modulated by cognitive expectations, attention, and learning [14,5,17,2].

Mild traumatic brain injury (TBI) leads to oculomotor deficits [8,38,37] increased intra-individual variability [36,38,29] and cognitive impairment, including attentional and executive deficits [18,33,26,4]. These deficits may jointly result from disruption of cerebellar-cortical tracts due to diffuse axonal injury

(DAI), a hallmark of TBI [28,1,21,9,31]. We investigated the utility of applying predictive SPEM testing with target blanking to the assessment of cognitive deficits after mild TBI. During smooth pursuit of a predictable moving target, the eyes often anticipate the target's trajectory [14,20]. In our previous study [38], mild TBI patients showed reduced target anticipation, increased oculomotor error, and increased variability of oculomotor error during target tracking, and these deficits were correlated with deficits in California Verbal Learning Test (CVLT-II) measures related to working memory, learning and executive attention [7].

When the target is visible during smooth pursuit, both retinal and cortical inputs play significant roles in programming predictive SPEM. However, when a target is temporarily extinguished, the smooth pursuit system relies solely on cortical input in order to extrapolate the virtual trajectory [24,3]. Therefore, the target-blanking paradigm may be a more sensitive indicator of the intactness of cerebellar-cortical pathways after mild TBI compared to our previous SPEM tracking paradigm without blanking [38]. Thus, we would expect predictive eye movements

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ring target blanking to correlate more strongly with cognitive measures than during target tracking.

We hypothesized that, due to DAI, which may particularly disrupt cerebellar-cortical tracts because of the highly distributed networks [1,9,31,28], mild TBI patients would show impairments in prediction during target blanking, and that these impairments would be associated with cognitive deficits, as measured by CVLT-II performance.

TBI: Twenty-six patients with mild TBI (Glasgow Coma scale (GCS) score 13–15 at time of injury) between ages 18–60 were tested. Twenty chronic patients (mean age 40.00 ± 11.19 years) were tested within two years after injury (range 6 weeks–24 months) and six acute patients (mean age 31.17 ± 14.32 years) were tested within 14 days after injury (range 8–12 days). Conditions for inclusion were blunt, isolated TBI, post-traumatic amnesia (PTA), and non-intoxication. Patients were excluded on the basis of multiple TBI with loss of consciousness (LOC), pregnancy, drug or alcohol abuse, neurological or psychiatric diagnosis, or seizures and general anaesthesia within two weeks following trauma. Acute and chronic patients were combined into one group for all analyses.

Control: The control group consisted of twenty-six subjects between ages 18–60 without prior history of TBI, drug or alcohol abuse, or neurological or psychiatric diagnosis. There was no significant difference in mean ages between groups (TBI: 31.12 ± 11.78 years, Control: 30.85 ± 13.02 years, $p=0.08$).

Eye movements were recorded by a human infrared eye tracking system (Eyelink II) with 500 Hz temporal resolution. The target stimulus, which was created in a Python program, was presented on a computer screen 40 cm from the subject. Before testing, an eyechart was used to verify that all subjects had normal or corrected-to-normal vision. Subjects were seated in a darkened room, and their heads were stabilized via a bite bar system. Calibration based on 9 points, including center and peripheral, was performed before each session, which also ensured that all subjects had a full range of oculomotor movement. The paradigm consisted of three blocks of 35 clockwise circular trajectories (7 degree radius, 0.4 Hz frequency), each with a period of target blanking (excluding the first cycle). Target blanking trajectories occurred at a fixed location (0°) but the duration varied randomly between 30° and 135° (208 ms and 938 ms) within subjects to avoid predictability of target re-onset. If subjects expressed signs of fatigue or discomfort, they were encouraged to take a break, after which the session was resumed.

Cognitive functioning was assessed by administering the second edition of the California Verbal Learning Test (CVLT-II) [7], a standard cognitive test measuring both recall and recognition memory of two word lists over several immediate and delayed memory trials. Standardized instructions were followed for test administration.

The signals representing eye and target movements were low-pass filtered at 50 Hz. All measures were averaged over the three blocks. For each trial, eye position over a 300ms period from the onset of target blanking was investigated. As an index of the ability to generate predictive SPEM, the time to the first saccade after target blanking was calculated by using saccade detection criteria (difference between eye velocity and target velocity

$\geq 40^\circ/\text{s}$). Horizontal oculomotor error (difference between eye position and virtual target position) during target blanking until the initial saccade was also calculated. Intra-individual oculomotor error variability was defined as the standard deviation of the difference between virtual target position and eye position during target blanking until the initial saccade.

Oculomotor error and intra-individual oculomotor error variability before target blanking were defined as the average and standard deviation of the difference between horizontal target position and horizontal eye position during the 300 ms period prior to target blanking, in order to match the period during target blanking that was analyzed. Saccades were detected based on a velocity threshold criteria (difference between eye velocity and target velocity $\geq 40^\circ/\text{s}$), counted and removed prior to calculating oculomotor error. A linear interpolation technique was used to bridge the gaps produced by removal of saccades.

A non-parametric Mann–Whitney *U*-test was used to determine differences between groups, as equality of variances could not be assumed. Linear regression analyses were used to determine the relationships among different measures.

TBI patients demonstrated a shorter mean time (TBI: 147.44 ± 27.17 ms, Control: 165.49 ± 31.95 ms, $p=0.02$) to an initial saccade, as well as greater intra-individual variability in this measure (TBI: 107.25 ± 13.60 ms, Control: 97.29 ± 23.31 ms, $p=0.03$). In addition, TBI patients showed greater oculomotor error (absolute value) before and during target blanking (TBI before: $0.72 \pm 1.52^\circ$, Control before: $0.36 \pm 1.34^\circ$, $p=0.02$; TBI during: $1.93 \pm 2.01^\circ$, Control during: $0.60 \pm 1.69^\circ$, $p=0.04$), as well as greater intra-individual oculomotor error variability during target blanking (TBI during: $1.88 \pm 1.11^\circ$, Control during: $1.19 \pm 0.90^\circ$, $p=0.004$), although not before target blanking (Table 1). Both groups demonstrated greater oculomotor error variability during target blanking than before target blanking; however, the TBI group showed a greater increase than the control group (TBI: $0.72 \pm 0.87^\circ$, Control: 0.28 ± 0.74 , $p=0.04$) (Table 1). Before target blanking, TBI patients demonstrated phase lag, indicated by negative average values of oculomotor error, while control subjects demonstrated phase lead, indicated by positive values (Table 1), which demonstrate anticipation of the target trajectory. These results were in agreement with our previous findings [38].

Correlations between eye movement measures before and during target blanking were analyzed in the TBI group alone as well as in the entire group of subjects. Phase (lead or lag) prior to target blanking was correlated with the time to initial saccade during target blanking in TBI patients ($r=0.4$, $p=0.04$). Oculomotor error before target blanking was correlated with oculomotor error variability during target blanking in the combined group ($r=0.54$, $p<0.01$), and oculomotor error variability before target blanking was correlated with oculomotor error during target blanking in both the TBI group alone and in the entire group (TBI: $r=0.62$, $p<0.01$; Combined: $r=0.59$, $p<0.01$).

The TBI group scored significantly lower than controls on CVLT-II measures related to executive functioning, including working memory, learning, and executive attention (the ability to resolve conflict among competing response alternatives) (Table 2). In the TBI group, the variability in time to the initial

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Table 1
Mean values for eye movement measures before and during target blanking in TBI and control subjects

Eye measure	TBI (n = 26)		Control (n = 26)		p-value
	Mean	S.D.	Mean	S.D.	
Time to first saccade (ms)	147.44	27.17	165.49	31.95	0.02
Variability of time to first saccade (ms)	107.25	13.60	97.29	23.31	0.03
Oculomotor error DB (deg)	-1.93	2.01	-0.60	1.69	0.04
Variability of oculomotor error DB (deg)	1.88	1.11	1.19	0.90	0.004
Oculomotor errorBB (deg)	-0.72	1.52	0.36	1.34	0.02
Variability of oculomotor error BB (deg)	1.16	0.58	0.92	0.46	0.13
Increase in oculomotor error variability DB (deg)	0.72	0.87	0.28	0.74	0.04

DB: during blanking, BB: before blanking.

Table 2
Mean CVLT-II scores for TBI and control subjects

CVLT-II measure	TBI (n = 26)		Control (n = 26)		p-value
	Mean	S.D.	Mean	S.D.	
Imm Recall 1	6.12	1.86	7.62	2.00	0.01
Imm Recall 5	11.69	2.51	13.19	2.15	0.03
Total Recall 1-5	48.54	10.22	56.89	10.01	0.001
SDFR	9.89	3.47	12.08	2.90	0.02
SDCR	10.62	3.53	12.65	2.87	0.03
LDFR	9.81	3.37	12.27	3.12	0.01
LDCR	10.77	3.23	12.77	2.87	0.03
FRD	1.91	0.43	2.39	0.49	0.001
CRD	2.12	0.85	2.67	0.83	0.02

Imm Recall 1: Immediate Recall Trial 1; Imm Recall 5: Immediate Recall Trial 5; Total Recall 1-5: Total Recall Trials 1-5; SDFR: Short-Delay Free Recall; SDCR: Short-Delay Cued Recall; LDFR: Long-Delay Free Recall; LDCR: Long-Delay Cued Recall; FRD: Free Recall Discriminability; CRD: Cued Recall Discriminability.

saccade and variability of oculomotor error during target blanking, as well as the increase in oculomotor error variability during target blanking compared to before target blanking, were highly correlated with most CVLT-II scores (Table 3). As an example, Fig. 1 depicts the correlation between the variability in time to the initial saccade and one CVLT-II measure, Long-Delay Cued Recall. In the control group, the variability in time to the initial saccade was correlated with most CVLT-II scores, although the correlations were lower than in the TBI group (Table 3).

This study examined the utility of applying predictive smooth pursuit testing to the assessment of cognitive deficits after mild

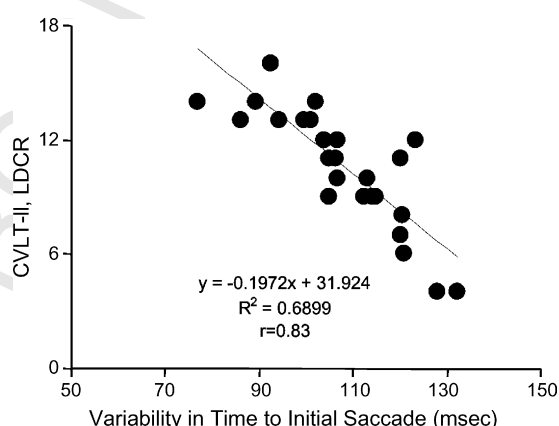


Fig. 1. Relationship between the CVLT-II Long-Delay Cued Recall score and variability in the time to initial saccade in TBI patients. Variability in time to initial saccade was highly correlated with CVLT-II performance.

TBI, using a circular pursuit target-blanking paradigm that necessitated the generation of predictive eye movements. The TBI group showed increased oculomotor deficits during target blanking, indicated by earlier generation of saccades, increased oculomotor error, and increased intra-individual variability compared to control subjects. Increased oculomotor variability was highly correlated with lower CVLT-II scores.

Studies have demonstrated that predictive SPEM during target blanking depends on input to the cerebellum from cortical areas including the FEF, SEF, PFC, and parietal cortex [24]. Therefore, we had hypothesized that a disruption of cerebellar-cortical tracts in TBI patients due to DAI [28,1,9,31] would lead

Table 3
Correlations between CVLT-II scores and oculomotor variability measures in TBI patients and control subjects

	CVLT-II measures								
	Imm Recall 1	Imm Recall 5	Total Recall 1-5	SDFR	SDCR	LDFR	LDCR	FRD	CRD
Oculomotor variability BB	r = -0.33 p = 0.1	r = -0.25 p = 0.22	r = -0.29 p = 0.16	r = -0.1 p = 0.64	r = -0.37 p = 0.06	r = -0.19 p = 0.36	r = -0.25 p = 0.22	r = -0.29 p = 0.16	r = -0.16 p = 0.42
Oculomotor variability DB	r = -0.27 p = 0.19	r = -0.71 p < 0.001	r = -0.62 p < 0.001	r = -0.48 p = 0.01	r = -0.57 p < 0.001	r = -0.52 p = 0.01	r = -0.56 p < 0.001	r = -0.60 p < 0.001	r = -0.41 p = 0.04
Variability in time to saccade DB	r = -0.32 p = 0.11	r = -0.69 p < 0.001	r = -0.62 p < 0.001	r = -0.74 p < 0.001	r = -0.80 p < 0.001	r = -0.72 p < 0.001	r = -0.84 p < 0.001	r = -0.68 p < 0.001	r = -0.78 p < 0.001
Increase in oculomotor variability DB	r = -0.26 p = 0.19	r = -0.59 p < 0.001	r = -0.57 p < 0.001	r = -0.47 p = 0.02	r = -0.51 p = 0.01	r = -0.49 p = 0.01	r = -0.55 p < 0.001	r = -0.57 p < 0.001	r = -0.44 p = 0.02

impairments in prediction during target blanking. In this study, TBI patients showed increased oculomotor error and oculomotor error variability compared to control subjects during target blanking, as well as an earlier interruption of predictive SPEM saccades, possibly compensating for increased oculomotor error [25].

The time to an initial saccade was correlated with phase (lead lag) prior to target blanking, suggesting that time to an initial saccade is an index of predictive ability. Nagel et al. [24] demonstrated that activation of the parietal lobe and dorsolateral PFC is negatively correlated with the frequency of saccade generation during smooth pursuit with target blanking. As these regions are part of cerebellar-cortical circuits involved in attention [35,27], these findings suggest that reduced attention leads to increased generation of saccades. Supporting this hypothesis, there is evidence that the increased production of disruptive saccades during smooth pursuit in schizophrenic patients and their children normalizes with attention facilitation [30]. Therefore, earlier generation of saccades during target blanking may be linked to reduced or dysfunctional attention.

The intra-individual variability of all eye movement measures was higher in the TBI group, consistent with previous studies [6,38]. Greater variability of oculomotor error prior to target blanking correlated with greater oculomotor error during target blanking. In addition, greater oculomotor error prior to target blanking correlated with greater variability of oculomotor error during target blanking. As other studies indicate that smooth pursuit is modulated by attention [5,13,11], this suggests that attentional dysfunction leads to increased performance variability. Taken together, these findings suggest that oculomotor error variability during smooth pursuit may be an index of moment-to-moment attention to the task. Studies have suggested that performance variability may be related to cerebellar dysfunction [6,6], which is also associated with attentional deficits [12,16]. The relationship between attention and performance variability is consistent with previous research in TBI patients [32], as well as with studies demonstrating increased reaction time variability in children with attentional deficits [15].

While both groups demonstrated greater oculomotor error variability during target blanking compared to before target blanking, the increase in variability during target blanking was greater in TBI patients than in control subjects, consistent with studies suggesting that the impact of attentional demands on variability is increased by neurological disturbance [22,34,10].

Mild TBI patients showed deficits on the CVLT-II, consistent with other studies demonstrating the impact of TBI on CVLT performance [41,42], and on attentional and executive functions in general [18,33,26,4]. CVLT-II scores were highly correlated with oculomotor variability during target blanking, but not before target blanking. In addition, the increase in oculomotor error variability during target blanking, a measure of the impact of target blanking on smooth pursuit, was highly correlated with CVLT-II scores. These correlations are consistent with the hypothesis that predictive eye movements during target blanking are primarily dependent on cortical input [24].

Unlike the current study, our previous study [38] found that TBI patients showed greater variability of oculomotor error

compared to control subjects during target tracking, and that oculomotor error and variability of oculomotor error during target tracking were correlated with CVLT-II scores, although these correlations were lower than those found during target blanking in the current study. However, these measures were calculated over a longer time interval (12.5 s), whereas in the current study they were calculated over 300 ms. The lack of oculomotor error variability differences and the lack of correlations between SPEM in the brief 300 ms period prior to target blanking and CVLT-II performance suggest that the target-blanking paradigm is a more sensitive test of attention over brief periods. Therefore, this paradigm may be a useful assessment of moment-to-moment attention.

Mild TBI patients showed increased oculomotor deficits during target blanking, indicated by earlier generation of saccades, increased oculomotor error, and increased intra-individual variability compared to control subjects. The relationship between oculomotor variability and other performance measures prior to and during target blanking suggests that attentional dysfunction may be linked to increased variability. Our finding of correlations between the variability of predictive eye movements and cognitive functioning supports the hypothesis that deficits in predictive oculomotor functions and attention in mild TBI patients are anatomically linked, possibly resulting from common disruptions in white matter cerebellar-cortical tracts. Therefore, predictive smooth pursuit testing during target blanking may be a sensitive indicator of diffuse axonal injury and resulting attentional deficits after mild TBI.

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