

## Deficits in predictive smooth pursuit after mild traumatic brain injury

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Received 24 January 2006; received in revised form 24 February 2006; accepted 27 February 2006

### Abstract

Given that even mild traumatic brain injury (TBI) may produce extensive diffuse axonal injury (DAI), we hypothesized that mild TBI patients would show deficits in predictive smooth pursuit eye movements (SPEM), associated with impaired cognitive functions, as these processes are dependent on common white matter connectivity between multiple cerebral and cerebellar regions. The ability to predict target trajectories during SPEM was investigated in 21 mild TBI patients using a periodic sinusoidal paradigm. Compared to 26 control subjects, TBI patients demonstrated decreased target prediction. TBI patients also showed increased eye position error and variability of eye position, which correlated with decreased target prediction. In all subjects, average target prediction, eye position error and eye position variability correlated with scores related to attention and executive function on the California Verbal Learning Test (CVLT-II). However, there were no differences between TBI and control groups in average eye gain or intra-individual eye gain variability, or in performance on the Wechsler Abbreviated Scale of Intelligence (WASI), suggesting that the observed deficits did not result from general oculomotor impairment or reduced IQ. The correlation between SPEM performance and CVLT-II scores suggests that predictive SPEM may be a sensitive assay of cognitive functioning, including attention and executive function. This is the first report to our knowledge that TBI patients show impaired predictive SPEM and eye position variability, and that these impairments correlate with cognitive deficits.

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**Keywords:** Mild TBI; Smooth pursuit eye movement; Shearing injury; Attention; Executive function; Variability; Diffuse axonal injury (DAI)

Traumatic brain injury (TBI) is the leading cause of death and disability in young people in the US. Of the 1.5 million brain injuries occurring annually, 85% are classified as “mild” [3]. Even mild TBI can produce extensive diffuse axonal injury (DAI) [1,32], leading to cognitive deficits [31,35]. Several studies have indicated that TBI patients are impaired in attentional and executive functions, including learning, working memory and executive control [4,5,28,40]. In addition, studies have demonstrated increased performance variability after TBI [17,42], which is potentially indicative of attentional deficits associated with cerebellar dysfunction [7,13]. However, conventional neuroimaging and neuropsychological measurements have thus far been unreliable in detecting structural abnormal-

ities and cognitive deficits after mild TBI [35,36]. Therefore, patients who sustain a head injury classified as mild TBI may have varying degrees of brain injury severity and symptoms.

Smooth pursuit eye movements (SPEM) are controlled by neural circuitry [11,24,33,39] overlapping with neural pathways involved in attention, anticipation and executive function [13,20,26,29,37], suggesting an association between SPEM and cognitive processes. During tracking of a predictable target, the cerebellum programs SPEM based on both retinal and extra-retinal signals, including cortical input [11,30,33,39,41], enabling the production of predictive SPEM. This compensates for delays (~100 ms) in the processing of visual feedback [21,25]. Behavioral [2,19,22,23,27,39] and fMRI [39] studies show that predictive SPEM is modulated by higher cortical functions such as attention, anticipation and learning. Due to extensive white matter connectivity between cerebral and cerebellar regions, the overlapping pathways involved in predictive

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SPEM and cognitive functions may be particularly susceptible to damage from DAI [1,18,36,38].

We hypothesized that predictive SPEM would be impaired in TBI patients, and that impairments would correlate with deficits in attention, anticipation and executive function. Correlations between these measures would suggest that predictive SPEM may be a sensitive metric of cognitive functioning. While several studies have demonstrated oculomotor deficits in TBI patients [15,16,47], to our knowledge no studies have shown specific deficits in predictive SPEM after mild TBI.

To explore this issue, a predictable sinusoidal stimulus, circular target tracking, was used to investigate deficits in predictive SPEM in mild TBI patients. Performance was monitored over a 50 s period. However, the tracking period was subdivided into epochs of 12.5 s to provide a thorough analysis of changes in performance over time. Performance on the California Verbal Learning Test (CVLT-II) [9] was studied to investigate whether there is an association between deficits in predictive smooth pursuit and cognitive functioning. To determine whether potential differences in CVLT-II performance between TBI patients and controls may be accounted for by IQ differences, the Wechsler Abbreviated Scale of Intelligence (WASI) [45] was also administered.

Twenty-one patients with mild TBI (Glasgow Coma Scale scores 13–15 at time of injury) between ages 15 and 60 were recruited for this study; 6 patients were tested within 10 days after injury (acute group, mean testing time after injury = 8.8 days), and 15 patients were tested within 5 years after injury (chronic group, mean testing time after injury = 2.3 years). Conditions for study inclusion were blunt, isolated TBI, post-traumatic amnesia, and non-intoxication at the time of testing. Patients were excluded on the basis of prior TBI with loss of consciousness, pregnancy, drug or alcohol abuse, neurological or psychiatric diagnosis, seizures or general anesthesia within two weeks of testing. All patients had significant symptoms of TBI, scoring  $\geq 2$  on the Head Injury Symptom Checklist [14]. The control group ( $n = 26$ ) consisted of subjects without a history of TBI between ages 15 and 60 and with the same inclusion criteria used for the TBI group. Mean ages and mean years of education for the TBI and control groups were matched (TBI: age  $35.7 \pm 11.8$ , education  $13.2 \pm 3.4$  years; Normal: age  $28.4 \pm 13.2$ , education  $14.1 \pm 2.8$  years; Mann–Whitney (MW) test,  $p = 0.24$  (age),  $p = 0.34$  (education)). Before each testing session, an eyechart was used to verify that all subjects had normal or corrected-to-normal vision.

Eye movements were recorded by a human infrared video-based eye-tracking system (Eyelink II) with 500 Hz temporal resolution. The target stimulus, a red circle of  $0.2^\circ$  diameter, was presented on a black computer screen. Subjects were seated in a darkened room 40 cm from the computer monitor, with heads stabilized via a bite bar system. Calibration based on nine central and peripheral locations was performed before each session, and also ensured that all subjects had a full range of oculomotor movement. The task consisted of two blocks of 20 (for TBI subjects) or four blocks of 20 (for control subjects) continuous cycles, in which the stimulus moved in a clockwise circular trajectory of  $7.0^\circ$  radius at a rate of 0.4 Hz. A larger number of

blocks was tested for the control group to evaluate test-retest reliability. If subjects expressed signs of fatigue or discomfort, they were encouraged to take a break. Standardized instructions were followed for the administration of the CVLT-II and WASI.

The signals representing eye and target movements were low-pass filtered at 50 Hz and stored on a computer for further analysis. Eye and target velocities were obtained by a two-point digital differentiation. Saccades were detected based on a velocity threshold criteria ( $100^\circ/\text{s}$ ), counted and removed. A linear interpolation technique was used to bridge the gaps produced by removal of saccades. An index for target prediction was obtained by identifying the maximum from a cross-correlation analysis between the response and the stimulus velocity. Positive indicates phase lead, while negative indicates phase lag. Eye position error was defined as the summation of the horizontal and vertical distance between eye and target position, and was averaged across the entire cycle. Intra-individual variability of eye position was defined as the standard deviation of eye position error. Eye gain was defined as the ratio between eye and target velocity. Intra-individual variability of eye gain was defined as the standard deviation of eye gain.

The TBI group showed decreased target prediction (increased phase lag) compared to the control group during the first five cycles (approximately 12.5 s duration) of each block; TBI:  $-8.8 \text{ ms} \pm 13.5 \text{ ms}$  versus controls:  $+4.4 \text{ ms} \pm 5.7 \text{ ms}$  (MW,  $p < 0.001$ ). No differences in target prediction (ANOVA;  $p > 0.05$ ) were found between blocks in either the TBI or control group, which allowed the averaging of target prediction ( ) across blocks (Fig. 1). Although both TBI and control groups exhibited some degree of target prediction, indicated by maximum greater than  $-100 \text{ ms}$ , the standard SPEM latency [21,25], 43% of TBI patients showed deficits in target prediction relative to control subjects during the first five cycles, indicated by a mean 2.5 standard deviations below the mean of controls. There were no differences between acute and chronic TBI patients on any measures (MW,  $p > 0.05$ ). Therefore, the groups were combined for all analyses.

The lack of differences in target prediction ( ) between the TBI and control group after the first five cycles (TBI:  $-9.7 \text{ ms} \pm 12.4 \text{ ms}$  versus controls:  $-6.3 \text{ ms} \pm 4.2 \text{ ms}$ ; MW,  $p > 0.05$ ) suggests that control subjects were unable to sustain the generation of predictive SPEM, decreasing their later performance to the level of TBI patients. Control subjects showed a significant decrease in target prediction over the course of each block (ANOVA;  $p < 0.01$ ), while TBI patients showed no change in performance within each block (ANOVA;  $p > 0.05$ ). It is possible that the control subjects began each block with a higher level of attention and subsequently experienced a decrease in prediction due to a loss of attention. In contrast, the TBI patients may have been consistently inattentive throughout the block. Therefore, only the initial five cycles of each block were used for comparison analyses, as averaging over all cycles would have eliminated differences between the TBI and control group. We will explore this possibility in future dual-task studies investigating the allocation of attention over the course of the experiment.

Compared to the control group, the TBI group showed greater eye position error, TBI:  $1.7^\circ \pm 1.1^\circ$  versus controls:  $0.8^\circ \pm 0.2^\circ$ ,

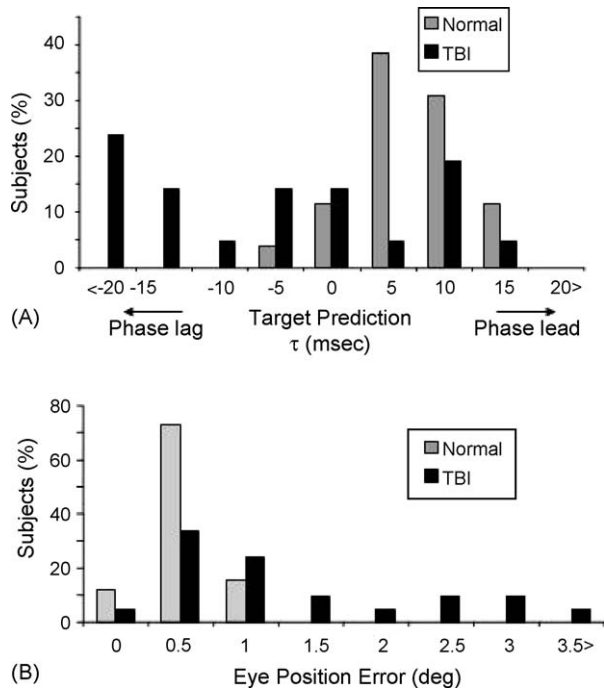


Fig. 1. (A) Histogram of average target prediction in each group (TBI ( $n = 21$ ) (black bar), Control ( $n = 26$ ) (grey bar)) during the first five cycles of each block. TBI patients showed decreased target prediction compared to the control group (MW,  $p < 0.001$ ). (B) TBI patients showed greater average position error and inter-individual variability of position error compared to the control group (MW,  $< 0.05$ ).

intra-individual variability of eye position, TBI:  $0.8^\circ \pm 0.6^\circ$  versus controls:  $0.5^\circ \pm 0.3^\circ$ , and number of saccades, TBI:  $7.4 \pm 3.2$  versus controls:  $5.2 \pm 2.1$  (MW, all  $p$ -values  $< 0.05$ ). In addition, measurements 2.5 standard deviations above the control mean were found in 48% of TBI patients for eye position error, 24% of TBI patients for intra-individual variability of eye position and 33% of TBI patients for number of saccades. Correlations were found between target prediction ( $\tau$ ) and eye position error (linear regression analysis:  $r = 0.7$ , regression coefficient (RC) =  $-9.5$ ,  $p < 0.001$ ), intra-individual variability

of eye position ( $r = 0.7$ , RC =  $-16.7$ ,  $p < 0.001$ ), and number of saccades ( $r = 0.6$ , RC =  $-2.3$ ,  $p < 0.001$ ), indicating that these indices of SPEM performance are not independent. However, no difference in average eye gain (TBI:  $1.0 \pm 0.1$  versus controls:  $1.0 \pm 0.1$ ; MW,  $p = 0.29$ ) or intra-individual variability of eye gain (TBI:  $0.06 \pm 0.06$  versus controls:  $0.03 \pm 0.03$ ; MW,  $p = 0.11$ ) was found between groups.

To investigate the relationship between deficits in predictive SPEM and cognitive functioning following TBI, CVLT-II scores were compared between groups. The TBI group produced lower CVLT-II scores related to attention, working memory, learning, and executive control (the ability to resolve conflict among competing responses) (MW) [9] (Table 1). WASI scores were compared between groups to examine whether the observed differences in CVLT-II scores could be accounted for by IQ differences. No difference between TBI and control groups was found in either the full WASI IQ scores or the WASI Vocabulary Test scores (Full WASI IQ: MW,  $p = 0.17$ , WASI Vocabulary Test:  $p = 0.25$ ). Regression analyses between CVLT-II measures and WASI scores in the combined group of TBI and control subjects showed no correlation (all  $p > 0.05$ ), with the exception of the analysis between Immediate Recall 5 and the WASI Vocabulary Test ( $p = 0.016$ ), indicating that differences in WASI IQ scores do not strongly account for differences in CVLT-II scores between the TBI and control group. CVLT-II measures related to attention, working memory, learning and executive control correlated with eye movement measures in the combined group of TBI and control subjects (Table 2). Within each group, control subjects showed significant correlations, while TBI subjects showed a trend toward significance, which may be due to the reduced number of TBI subjects compared to control subjects.

The results of this study indicate that mild TBI patients show decreased target prediction during predictable smooth pursuit tracking, associated with increased eye position error, intra-individual eye position variability, and cognitive deficits. The finding of increased intra-individual and inter-individual SPEM variability (demonstrated by a more broadly distributed histogram in the TBI group compared to the control group (Fig. 1)) is consistent with previous demonstrations of reaction time vari-

Table 1  
Comparison of CVLT scores between the TBI and control groups

CVLT measure	Cognitive measure	TBI ( $n = 21$ )		Control ( $n = 26$ )		$p$ -value
		Mean	S.D.	Mean	S.D.	
Immediate free recall trial 1	Attention span	6.33	1.39	7.5	1.84	0.0223*
Immediate free recall trial 5	Encoding/retrieval	12.24	2.3	13.54	2.14	0.0296*
Total free recall (Trials 1–5)	Global verbal learning	49.29	9.34	57.65	9.79	0.0024*
Short-delay free recall	Executive attention	10.43	3.74	12.38	2.98	0.0226*
Short-delay cued recall	Working memory-based retrieval	11.38	3.43	13.08	2.65	0.0357*
Long-delay free recall	Encoding/retrieval	10.57	3.49	12.77	2.66	0.0077*
Long-delay cued recall	Working memory-based retrieval	10.71	3.91	13.35	2.58	0.0076*
Semantic clustering	Working memory-based encoding	1.26	1.99	1.23	1.68	0.6151
Across-trial recall Consistency	Consistency of learning strategy	76.38	14.87	85.77	7.39	0.0056*
Delayed recognition total hits	Encoding/retrieval	13.52	4.62	14.5	3.00	0.2762
Total recognition discriminability	Executive attention	2.82	1.36	3.43	0.59	0.0806

Comparison of CVLT-II scores between the TBI and control groups. TBI patients scored lower on measures related to attention, working memory, learning and executive control. Significant correlations are indicated with an asterisk.

Table 2  
Correlations between oculomotor measures and CVLT-II scores in all subjects

CVLT measure	Target prediction			Position error			Position error variability		
	<i>r</i>	Regression coefficient	<i>p</i> -value	<i>r</i>	Regression coefficient	<i>p</i> -value	<i>r</i>	Regression coefficient	<i>p</i> -value
Immediate free recall Trial 1	0.38	0.06	0.010*	0.18	−0.36	0.234	0.3	−1.12	0.040*
Immediate free recall Trial 5	0.28	0.05	0.050*	0.31	−0.81	0.034*	0.22	−1.09	0.130
Total free recall (Trials 1–5)	0.33	0.29	0.020*	0.28	−3.39	0.055	0.32	−7.06	0.030*
Short-delay free recall	0.28	0.08	0.050*	0.29	−1.13	0.049*	0.29	−2.15	0.048*
Short-delay cued recall	0.4	0.11	0.005*	0.26	−0.94	0.074	0.29	−1.94	0.047*
Long-delay free recall	0.4	0.11	0.010*	0.40	−1.47	0.005*	0.39	−2.71	0.010*
Long-delay cued recall	0.52	0.15	<0.001*	0.41	−1.65	0.004*	0.36	−2.65	0.010*
Semantic clustering	0.1	0.01	0.520	0.09	−0.18	0.545	0.05	−0.18	0.750
Across-trial recall consistency	0.24	0.24	0.110	0.28	−3.96	0.060	0.18	−4.70	0.230
Delayed recognition total hits	−0.03	−0.01	0.840	0.18	−0.79	0.225	0.02	−0.16	0.890
Total recognition discriminability	0.11	0.01	0.470	0.21	−0.26	0.151	0.13	−0.28	0.390

Correlations between target prediction, eye position error, intra-individual variability of eye position and CVLT-II measures. Significant correlations are indicated with an asterisk.

ability in TBI patients [17,42]. Intra-individual variability may result from cerebellar dysfunction [7,13], possibly associated with attentional deficits [20,26], while inter-individual variability may result from heterogeneity of brain damage (both in location and severity) in the TBI group [17,36]. Correlation studies with neuroimaging data will enable direct testing of these hypotheses.

In contrast to the current study's finding of decreased target prediction during predictive SPEM in mild TBI patients, Heitger et al. [16] reported no difference in predictive sinusoidal smooth pursuit between mild TBI patients and controls, indicated by similar phase lags, although the study did find increased phase lags on a random trajectory, as well as increased saccadic latencies, in TBI patients. However, SPEM measures were averaged over all cycles (40 s), which would have obscured differences between groups if the control subjects showed a pattern of performance similar to those in the current study, in which target prediction decreased after the first five cycles.

The finding of correlations between target prediction and other measures of SPEM suggests that target prediction is necessary in order to generate accurate and consistent SPEM. A dual-task study in normal subjects [19] demonstrated that performance of an attention-demanding secondary task during smooth pursuit leads to decreased SPEM accuracy, indicating that attention may play a significant role in the maintenance of accurate SPEM. The study did not specifically examine predictive SPEM; however, the results suggest that decreased target prediction, which the current study shows to be associated with inaccurate eye movements, may reflect decreased attentional resources. In addition, the increased production of saccades in the TBI group may have consisted mainly of "catch-up" saccades, which are produced reflexively in order to compensate for inaccurate SPEM [8], and therefore may be another index of performance inaccuracy.

The lack of a difference between groups in average eye gain and intra-individual variability of eye gain indicates that differences in target prediction and eye position error between the TBI and control group are not due simply to general oculomotor impairment or to a velocity-storage dysfunction in the smooth

pursuit system, but to specific deficits in predictive SPEM. This result is consistent with damage to cerebellar-frontal rather than cerebellar-vestibular connections [6,10,34,44]. However, it is possible that other connections, including cortico-cortical, may be affected by TBI, depending on the degree of head injury.

The TBI group's lower performance on the CVLT-II is consistent with other studies demonstrating impairments in TBI patients on the CVLT [43,46], as well as studies suggesting that TBI patients are impaired in paradigms requiring attention and executive control [28,40] and lack effective encoding strategies [4]. These impairments may be due to an inability to manipulate items in working memory [5], which has been linked to frontal lobe functioning [12]. Similarly to their oculomotor performance, the TBI group showed increased intra-individual variability in CVLT-II performance, indicated by lower scores on the measure "Across-Trial Recall Consistency", as well as increased inter-individual variability (Table 1). Although the CVLT-II measures on which deficits were observed have been linked to several components of executive function, including working memory and executive control [9], these measures do not conclusively differentiate between these processes. Therefore, further studies are necessary in order to dissociate the effects of TBI on various components of executive function.

In both TBI and control subjects, CVLT-II measures were correlated with oculomotor measures, suggesting that performance on these measures for both TBI patients and the normal population lies on a continuum, with TBI patients performing more poorly overall. We explored potential relationships between SPEM and cognitive measures, although other factors are also likely to influence SPEM performance, as DAI leads to "idiosyncratic combinations" of deficits [36]. An experiment of broader scope may permit an investigation of additional factors accounting for individual differences.

The coupling of oculomotor and cognitive measures supports the hypothesis that the prevalence of DAI [1,32,36] following mild TBI may result in impairments to both predictive SPEM and higher-level cognitive systems, which depend on extensive overlapping networks of cerebral and cerebellar regions [30,33]. Similar neural circuitry involved in predictive SPEM and atten-

tion, anticipation and executive function may allow parameters of predictive eye movements to be used as a metric of these functions in both the normal population and TBI patients, and as a sensitive assay of damage to the underlying neural connections after TBI.

## Acknowledgements

Supported by a collaborative grant from the McDonnell Foundation to the Brain Trauma Foundation, with lead investigators from Weill Cornell Medical College, UC San Francisco, and UC Berkeley.

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