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Eye movement and visuomotor arm movement deficits following mild closed head injury

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Summary

Based on increasing evidence that even mild closed head injury (CHI) can cause considerable neural damage throughout the brain, we hypothesized that mild CHI will disrupt the complex cerebral networks concerned with oculomotor and upper-limb visuomotor control, resulting in impaired motor function. Within 10 days following mild CHI (Glasgow Coma Scale 13–15, alteration of consciousness <20 min), we compared 30 patients (15–37 years) and 30 matched controls on different types of saccades, oculomotor smooth pursuit (sine and random), upper-limb visuomotor performance and several neuropsychological tests known to be sensitive to head trauma. Simple reflexive saccades were not impaired, whereas, on the antisaccade task, the CHI group demonstrated prolonged saccadic latencies, a marginally higher number of directional errors and poorer spatial accuracy. The CHI group exhibited more directional errors and impaired motor accuracy on memory-guided sequences of saccades and produced fewer self-paced saccades within 30 s. Most measures of sinusoidal and random oculomotor smooth pursuit showed no deficits, with the exception of a prolonged lag on random smooth pursuit in the CHI group. While arm movement reaction time and arm steadiness were not impaired, the CHI group showed decreased arm movement speed and decreased upper-limb motor accuracy. Conversely, after controlling for IQ, the CHI group had few head trauma-related neuropsychological deficits. These results indicate that multiple motor systems can be impaired following mild CHI and that this can occur independently of neuropsychological impairment. Our study also indicates that quantitative tests of oculomotor and upper-limb visuomotor function may provide sensitive markers of cerebral dysfunction, suggesting the potential use of such tests to supplement patient assessment. To our knowledge, this study is the first to demonstrate the presence of oculomotor or visuomotor deficits following mild CHI.

Keywords: closed head injury; saccades; oculomotor smooth pursuit; upper-limb visuomotor; neuropsychological function

Abbreviations: CHI = closed head injury; CVLT = California Verbal Learning Test; DLPFC = dorsolateral prefrontal cortex; FEF = frontal eye field; GCS = Glasgow Coma Scale; LED = light-emitting diode; LOC = loss of consciousness; PASAT = Paced Auditory Serial Addition Task; PEF = parietal eye field; PPC = posterior parietal cortex; PTA = post-traumatic amnesia; SDMT = Symbol Digit Modalities Test; SMA = supplementary motor area; TMS = transcranial magnetic stimulation; TMT = Trail Making Test; WASI = Wechsler Abbreviated Scale of Intelligence

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Introduction

Around 80% of head injury admissions are categorized as mild (Jennett, 1996), and there is increasing evidence that even mild closed head injury [CHI; rating of 13–15 on the Glasgow Coma Scale (GCS) with a brief loss of consciousness (LOC)] can cause considerable neural damage throughout the brain, in the form of either focal lesions or diffuse axonal injury (Jane *et al*., 1985; Levin *et al*., 1992; Mittl *et al*., 1994; Servadei *et al*., 1994; Kant *et al*., 1997; Hofman *et al*., 2002; Lorberboym *et al*., 2002; Chen *et al*., 2003). A significant proportion of mild CHI patients experience

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disabling persistent post-concussional complaints beyond the first weeks following the injury, sometimes for months or even years (Bohnen and Jolles, 1992; Mallinson and Longridge, 1998; Wrightson and Gronwall, 1998; Ponsford *et al*., 2000). At 6 months post-injury, ~30% of mild CHI patients meet the criteria of the International Classification of Diseases (World Health Organization, 1992) for the diagnosis of post-concussion syndrome (Mittenberg and Strauman, 2000; Mittenberg *et al*., 2001). Unfortunately, initial clinical measures of trauma severity such as GCS, duration of LOC and degree of post-traumatic amnesia (PTA) are unable to predict the development of post-concussion syndrome. Several studies have investigated both the use of early post-CHI symptoms (e.g. headache, dizziness or nausea) and the potential of biochemical markers to predict outcome following mild CHI, but have so far failed to produce measures which are both specific and sensitive enough to predict adverse outcomes reliably (De Kruijk *et al*., 2002; Savola and Hillbom, 2003). Similarly, evidence from neuropsychological assessment has been generally unsatisfactory and is vulnerable to pre-morbid intelligence and other factors such as age, level of education, state of employment or socio-economic status (Taylor *et al*., 1996; Binder *et al*., 1997; Reitan and Wolfson, 1997; Ponsford *et al*., 2000; Dikmen *et al*., 2001; Wallesch *et al*., 2001).

We hypothesized that quantitative computerized assessment of eye and arm movement performance might have considerable potential to contribute to improved CHI patient assessment. The cerebral structures concerned with the control of voluntary eye and arm movements are well mapped and form extensive and highly complex functional entities, incorporating cortical and subcortical structures as well as the cerebellum. Such complex anatomical structures are highly susceptible to the adverse effects of neural injury. It was our contention that oculomotor and upper-limb visuomotor function would be impaired even in mild cases of CHI, despite such deficits not being evident on standard clinical examination. Surprisingly, there have been no studies of arm visuomotor function (i.e. the voluntary execution of upper-limb limb motor tasks under controlled guidance by visual feedback) following mild head trauma, and only one study has assessed saccadic function following mild CHI. Crevits *et al*. (2000) examined latencies and response errors of single remembered saccades and antisaccades but failed to detect significant differences between the head-injured and the control group. Their study was hampered by several factors such as sample size, limited paradigm sensitivity, and selection of response errors and latencies as sole measures.

Motor deficits following mild head trauma are thus a largely under-researched area, and it was the main aim of our study to establish the incidence of any deficits in eye and visuomotor arm movement performance following mild CHI. We wished to examine whether these motor deficits would be sufficiently substantial to warrant a prospective study aiming to determine the potential clinical use of quantitative motor assessment as a supplement to established clinical and psychometric measures. We further employed standardized neuropsychological measures of attention, working memory, speed of information processing and episodic memory to compare the motor performance with the level of neuropsychological functioning. The neuropsychological testing provided the opportunity to rate our CHI group on standardized measures that have been used previously to quantify the impact of mild head trauma. Due to the functional heterogeneity of the cerebral motor systems, we anticipated that additional information on the level of cognitive deficits in our patient group would contribute to the interpretation of any motor deficits and help to ringfence the likely causes and possibly anatomical origin of any observed motor impairments.

Methods

Participants

Thirty patients (11 female and 19 male) with mild CHI (GCS score 13, two cases; 14, five cases; 15, 23 cases) were recruited from patients presenting with acute head injury to Christchurch Hospital (the principal hospital for a population pool >400 000 within the south island of New Zealand). All patients had experienced PTA (mean = 34.4 min, range 3 min–4 h) and 25 patients had a confirmed LOC (mean = 2.56 min, range 1–15 min). Mean age was 22.2 ± 7.1 years (range 15–37 years) and mean years of education was 12.8 ± 1.86. CT head scans were undertaken in seven participants and all were normal. All patients were either employed or attended institutions for secondary or tertiary education, and none was involved in litigation. Other potential participants were excluded if there was evidence of any influence of alcohol or psychoactive drugs at the time of injury, regular intake of psychoactive drugs or history of drug abuse, central neurological disorder or psychiatric condition, structural brain damage or haematoma on CT head scan (where obtained), oculomotor or somatomotor deficits upon clinical examination, presence of strabismus, visual acuity of ⩽6/12, skull fractures, or prior history of mild, moderate or severe head injury with persisting symptoms or complaints.

The number of patients without an LOC was considered too small ($n = 5$) to warrant a separate inter-group comparison between only these patients and their controls. A within-group t test analysis showed that the five non-LOC patients did not differ from the remaining 25 CHI patients regarding age ($P = 0.6$), gender ($P = 0.41$), years of education ($P = 0.44$), initial GCS score ($P = 0.22$) and duration of PTA ($P = 0.40$). Similarly, the five non-LOC patients did not differ significantly from those with LOC on any motor or neuropsychological measure. Subsequently, all patients in the study were combined into one CHI group.

The control group consisted of subjects with no history of mild, moderate or severe head injury with persisting symptoms or complaints, no central neurological disorder or psychiatric condition, and no regular intake of psychoactive drugs or history of drug abuse. The controls were individually matched to each CHI case with respect to age (control mean 22.4 ± 7.0 years, range 15–37 years), gender and years of formal education (mean 13.2 ± 2.1). The project was approved by the Canterbury Ethics Committee and written consent was obtained from all participants.

Oculomotor testing

Equipment

Eye movements were recorded using an IRIS infrared limbus tracker (Skalar Medical, BV, Delft, The Netherlands) (Reulen *et al*., 1988). Eye position signals were low-pass filtered at 100 Hz, sampled and digitized at 200 Hz, and recorded for off-line analysis. Subjects were seated in a darkened room. Head movements were stabilized via a wax bite-bar. Eye movements were elicited by instructing the subject to follow a computer-generated stimulus [for saccades, a red square target, subtending 0.75°, front-projected onto a video screen 1.72 m in front of the subject or a light-emitting diode (LED) bar 1.5 m in front of the subject; for oculomotor smooth pursuit, a circle with a centred cross, subtending 4.82°]. The tests were generated and controlled by a PC (Muir *et al*., 2003) which also recorded the data for off-line analysis. The equipment was calibrated at the start of the session and between tests. Mean values of the key measures over all trials in a particular test paradigm were used in analyses.

Reflexive saccades

A target jumped randomly by 14, 16, 18, 20, 22 or 24° in a horizontal direction on a video screen, at intervals varying pseudorandomly between 1.0 and 2.0 s. The current (fixation) target was extinguished at the same time as the next (peripheral) target was illuminated. A tone sounded in coincidence with the target jump. The test sequence included 49 trials and the subjects were instructed to follow the targets as quickly and accurately as possible. Key measures were saccade latency (ms), saccade velocity (°/s), mean absolute position error of the final eye position [$PE_{reflexive} = |(EP_{fin} - SP)/SP| \times 100$], gain of the primary saccade ($G_p = EP_{prim}/SP$) and gain of the final eye position ($G_f = EP_{fin}/SP$), where EP_{prim} is the eye position after the initial saccade, EP_{fin} is the final eye position and SP is the stimulus position.

Antisaccades

The subject was instructed to fixate a central target. After a random time of 1.0–1.6 s, the central fixation LED was extinguished coincident with the appearance of a peripheral horizontal target at either 5 or 15° off centre. A tone sounded at the same time. The subject was instructed not to look at the peripheral target but to make a saccade in the opposite direction as quickly and accurately as possible, an equal distance from the central fixation point (Fig. 1A). Directional errors (i.e. erroneous prosaccades) had to be corrected as quickly as possible. The peripheral stimulus was extinguished after 1.5 s, and the subject refixated the central fixation stimulus, which was illuminated again. The task included 32 trials (16 left, 16 right, balanced for 5 and 15° steps). A practice test incorporating 10 antisaccade trials familiarized the subject prior to the test. Key measures were the number of directional errors (incorrect glances towards the peripheral target), antisaccade latency (ms), latency of directional errors (ms), correction time for directional errors (ms; latency from termination of the erroneous prosaccade until initiation of the correcting saccade), antisaccade peak velocities (°/s), gain of the primary antisaccade (G_p), gain of the final eye position (G_f) and the mean absolute position error [$PE_{antisaccade} = |(EP_{fin} - SP)/SP| \times 100$] for all saccades within the test. Both the final saccade gain and the mean absolute error are related, but each quantifies a different aspect of motor accuracy. The mean absolute position error quantifies the size of error, i.e. the difference between desired and actual eye position, whereas the final saccade gain assesses whether there is a general direction to that error (i.e. whether head injured and controls, while having a certain mean position error, equally over- and undershoot the desired target position, or whether mild head trauma is likely to cause abnormal hyper- or hypometria).

Sequences of memory-guided saccades

A central fixation LED appeared for 2.0 s and then jumped to a predefined number of successive horizontal eccentric positions 5 or 15° on either side of the central fixation point, 1.0 s for each position and with the final sequence position always being the central fixation LED. The test contained six different sequences with four target positions (i.e. three steps) and five practice repetitions per sequence (Fig. 1B) (i.e. subjects viewed the current sequence five times in succession immediately before they had to perform it from memory). During practice, a tone sounded coincident with each target relocation and subjects were instructed to follow the lights and memorize the positions, order and timing in the sequence. Following completion of the last practice repetition, there was a 2 s delay (indicated by illumination of the central LED) before initiation of the memory-guided performance (indicated by extinguishing the central LED). Subjects then had to replicate the sequence in darkness and without the tone as accurately as possible in terms of positions and timing of the sequence. This was followed by the presentation of a new sequence (i.e. five practice repetitions, etc.). Prior to test recording, subjects were exposed to a training sample sequence to familiarize themselves with the paradigm and ensure that they had understood the instructions. This was achieved by observing and then performing one sequence test, which consisted of five practice repetitions and the following memory-guided performance. This sample trial was not part of the six sequences used in the actual test recording. Key measures were the number of directional errors, gain of the primary saccade (G_p), gain of the final eye position (G_f) and the mean absolute sequence position error {$PE_{sequence\,s} = [(PE_{step1S} + PE_{step2S} + \ldots.. + PE_{stepnS})/n]$}. In order to be counted as a separate step, sequence steps had to have a minimum duration of 150 ms. Omissions of steps or initiation of sufficiently sized saccades (i.e. those of size comparable with that required), but in the direction opposite to the next position within the sequence (after having reached an established final eye position in the preceding step), followed by a fixation-like pause, were counted as directional errors. The position information of directional errors was excluded from the collated saccade data, as inclusion of these data would have heavily skewed information on spatial accuracy of saccades directed to the correct destinations.

Self-paced saccades

Two LEDs at ±15° (horizontal) were illuminated simultaneously and continuously for 30 s. The subject was instructed to look back and forth between the lights as quickly and accurately as possible. Key measures were the number of refixations within 30 s and the mean intersaccadic interval (ms).

Smooth pursuit

The subject was instructed to fixate and track the centre of a horizontally moving stimulus, projected onto a video screen 1.72 m in front of the subject. The tests included sine tracking [predictable sinusoidal pattern, peak velocity 20, 40 (Fig. 1C) and 60°/s] and random tracking (random pattern, mean peak velocity 80°/s). The

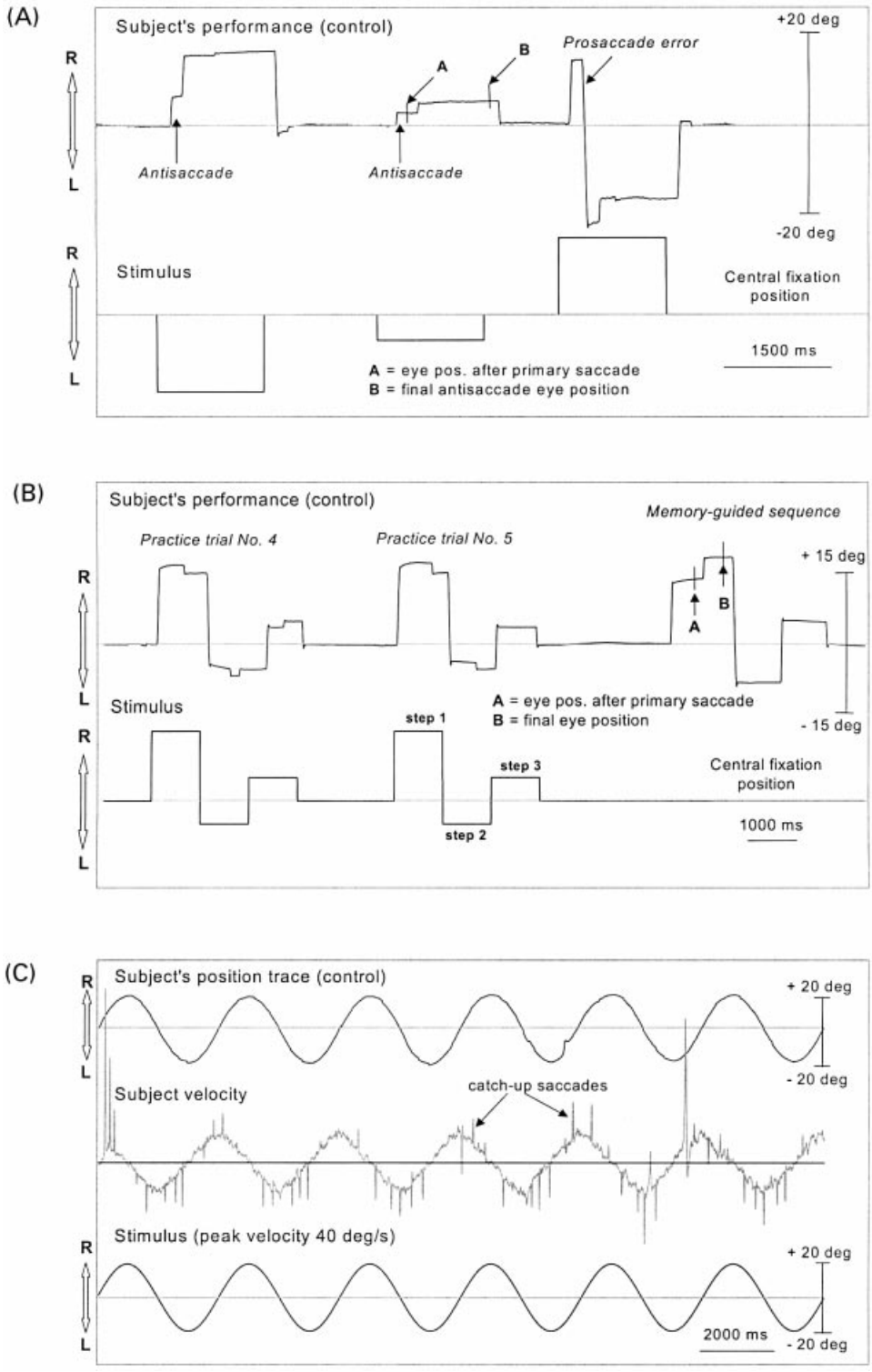

Fig. 1 Oculomotor test paradigms. Line traces represent stimulus position and eye movement performance. Timeline progresses from left to right. Upward line displacements describe stimulus/eye movements to the right, downward to the left. Sample recordings displaying stimulus and subject performance are shown for (**A**) antisaccades (including two antisaccades and one directional error, an erroneous prosaccade), (**B**) memory-guided sequences of saccades (showing two practice repeats and one memory-guided performance) and (**C**) oculomotor smooth pursuit (with the eye velocity trace before removal of catch-up saccades). Saccades show a distinct step pattern, whereas continuous oculomotor smooth pursuit is characterized by a wave-like line form.

duration of each test was 40 s. Key measures were the average eye peak velocity (°/s) after removal of all saccades from the tracking performance and the tracking lag (ms).

Upper-limb visuomotor testing

Equipment

The test setup comprised tests of visual perception, basic arm motor function and several one-dimensional (1D) visuomotor tracking tasks (Jones and Donaldson, 1986; Jones *et al*., 1993; Jones, 2000). These tests have been applied extensively to assess visuomotor arm function following stroke (Jones *et al*., 1989, 1990) and in disorders such as Parkinson's disease (Jones and Donaldson, 1989, 1995; Dalrymple-Alford *et al*., 1994; Jones *et al*., 1992, 1996; Watson *et al*., 1997) and developmental stuttering (Jones *et al*., 2002). The tests are similar to computerized tests used by others to assess upper-limb visuomotor performance in patients with focal lesions (e.g. Haggard *et al*., 1995; Liu *et al*., 1999; Gomez Beldarrain *et al*., 2002), stroke (e.g. Lynn *et al*., 1977; DeSouza *et al*., 1980; O'Dwyer *et al*., 1996), essential tremor (Schwartz *et al*., 1999) and Parkinsons's disease (e.g. Angel *et al*., 1970; Bloxham *et al*., 1984; Sheridan *et al*., 1987; Soliveri *et al*., 1997; Hocherman and Giladi, 1998; Turner *et al*., 2003).

The apparatus included a PC and two colour monitors: one displaying test stimuli for the subject and the other used by the assessor for task generation and analysis. Each test lasted 1–2 min. Subjects were seated in front of a colour monitor (312 × 234 mm) with an eye–screen distance of 132 cm. All of the 1D tracking tasks had a steering wheel (395 mm diameter) as the subjects' output sensor, with rotation of the wheel moving a vertical white arrow (16 mm high, 11 mm wide) horizontally on a black background (top of the arrow 58 mm from the bottom of the screen). In the tests requiring the use of the steering wheel, subjects placed their preferred hand at a fixed position on the steering wheel (10 o'clock mark for left handers and 2 o'clock mark for right handers; exceptions were the tests for arm speed, reaction time and steadiness, for which the hand was placed at the 12 o'clock mark).

The mean absolute error (horizontal distance between arrowhead and target) and mean delay between response and target were measured for all 1D tracking tasks, where

$$\text{Mean absolute error} = \frac{1}{N}\sum_{i=1}^{N} \mid (x_t - x_r) \mid$$

and $|(x_t - x_r)|$ is the absolute difference (error) between the target and tracking response, sampled at 60.34 Hz (Jones, 2000). Calculation of the cross-correlation function between the target and response provided the tracking lag (ms).

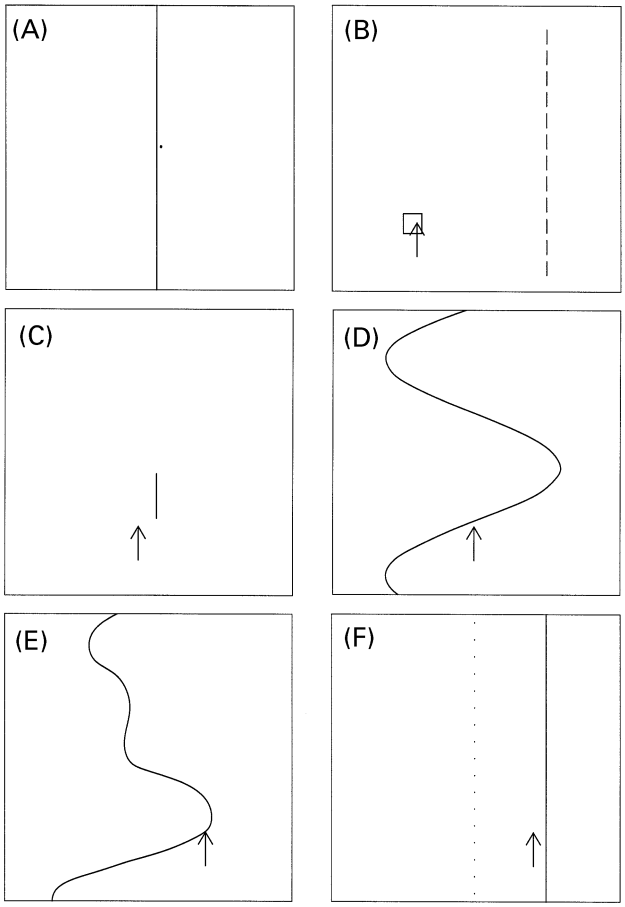

Fig. 2 Sample displays of visuoperceptual and arm visuomotor tasks. Shown are the test displays for visual resolution (**A**), static perception (**D**), dynamic perception (**E**), ballistic arm movement (**B**) and the 1D tracking tasks (**C–F**), each with movement stimulus and response arrow (the latter controlled by the subject via a steering wheel). In the ballistic arm movement task (**B**), the subject moves the arrow into the box (start position) and is instructed to wait for the go signal (i.e. after a delay of 3–7 s the dashed red line turns into a solid green line) and then move the arrow past the line as fast as possible. The 1D tracking tasks shown are sine/random tracking (**C**), preview sine tracking (**D**), preview random tracking (**E**) and step tracking (**F**). In the non-preview tasks, the vertical target line moves horizontally on the screen. In the preview tasks, the target line descends from the top of the screen, providing an 8 s preview before reaching the level of the arrow point.

Visuoperception and basic arm motor function

Visual acuity. This was tested using the patients' best eye on the Snellen chart at 6 m.

Visual resolution. The subject was asked to identify the position of a dot with respect to a vertical line on screen. Dot–line separations were in multiples of 0.27 mm (Fig. 2A). Visual resolution was defined as the minimum separation (mm) at which a subject was always able to identify the dot correctly as being off the centre of the line.

Arrow perception. The subject had to indicate the position of a dot relative to an arrow point in 13 trials (responses: 'on or 'off'). The number of correct answers was recorded.

Static perception. This test simulated the appearance of the 1D tracking tasks, but with a stationary stimulus. The subject had to indicate the position of an arrow point with respect to a static vertical line in four trials and a static sinusoidal wave in 16 trials (responses: 'left', 'right' or 'on', Fig. 2D). The test score of number of incorrect responses was converted to static perception resolution (mm; Jones and Donaldson, 1995).

Dynamic perception. This test simulated the appearance of the 1D preview tracking tasks. The subject had to determine whether an arrow point stayed perfectly on a random input descending the screen with 8 s preview time (Fig. 2E). The duration of 20 trials decreased from 10 to 2 s and various error offsets were simulated. A dynamic perception resolution was defined as the minimum spacing between the point of the arrow and the target over the 20 trials at which a subject was always able to perceive the arrow as being off the target at some stage during its descent (Jones and Donaldson, 1995).

Ballistic movement. This test measured the fastest possible arm movement in response to a non-target stimulus (no accuracy required). This required moving the arrow out of a box and across a pass-line equivalent to 90° of movement on the steering wheel in response to a random 3–7 s latency stimulus (Fig. 2B). The best reaction time and speed of movement over eight attempts were recorded.

Steady movement. This test measured the steadiness of attempted constant-speed non-pursuit movement on the steering wheel over a range of 116° (mm/s). The subject was asked to move an arrow horizontally at the same speed as a sample stimulus (a moving dot) and to maintain that speed once the stimulus disappeared. The best of eight attempts within a speed range of 17.7–34.7°/s was recorded.

1D tracking tasks

Sine tracking (non-preview). This test assessed the ability to keep an arrow point on a sinusoidal target (0.15 Hz, a straight line displacing in a lateral fashion on the screen; Fig. 2C). The task required smooth movements over a 180° range of the steering wheel.

Random tracking (non-preview). This test assessed the ability to keep an arrow point on a random target (bandwidth 0.34 Hz, a straight line displacing in a lateral fashion on the screen; Fig. 2C). The task required smooth movements over a 175° range of the steering wheel.

Sine tracking (preview). This test assessed the ability to keep an arrow point on a sinusoidal target (same thickness yellow line down the full screen; maximum displacement 96 mm). The tasks incorporated the same target displacement pattern as the non-preview sine task but provided an 8.0 s preview of the target line as it moved down the screen (Fig. 2D).

Random tracking (preview). This test assessed the ability to keep an arrow point on a randomly displacing target (same thickness yellow line down the full screen; maximum displacement 96 mm). The tasks incorporated the same target displacement pattern as in the non-preview random task but provided an 8.0 s preview of the target line as it moved down the screen (Fig. 2E).

Step tracking (non-preview). This test assessed the ability to keep an arrow point on a vertical line as it moved abruptly by way of 32 steps alternating between displacement from and return to centre screen (Fig. 2F). The subject had to use fast ballistic movements to keep the arrow aligned with the target. Spatial unpredictability was incorporated via four randomly distributed amplitude/direction movements from ('step out') and back ('step back') to the centre (centre was always indicated by a vertical line of nine dots). The task incorporated large steps (90° on wheel) and small steps (22°), to both the right and left of centre. Four randomly distributed durations between steps (2.8, 3.4, 4.0 and 4.6 s) and lack of preview ensured temporal unpredictability. The duration of the test was 120 s.

Neuropsychological tests

Attention, working memory, episodic memory and speed of information processing were assessed using the Paced Auditory Serial Addition Task (PASAT; Gronwall, 1977), the California Verbal Learning Test I (CVLT; Delis, 1987), Symbol Digit Modalities Test (SDMT; Smith, 1973) and the Trail Making Test A + B (TMT A + B; Spreen and Strauss, 1991). General cognitive performance was evaluated with the Vocabulary Test and Matrix Reasoning subtests of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). Standardized instructions were followed for all tests.

General procedures

The approximate duration of the complete test battery was 3 h. In order to minimize fatigue effects resulting from long test sessions, the testing for each participant occurred in two separate sessions. All patients completed the tests within 16 days of their injury (session 1, mean 4.23 $\pm$ 1.79 days, range 2–9 days; session 2, mean 6.46 $\pm$ 3.3 days, range 3–16 days). The first session included tests for antisaccades, sequences of memory-guided saccades, self-paced saccades, upper-limb visuomotor testing and neuropsychological assessment (CVLT, PASAT, TMTs and SDMT). The second session included tests for reflexive saccades, oculomotor smooth pursuit and the WASI.

Statistical analysis

Most measures displayed considerable non-normality and skewed distributions. Hence, a non-parametric Wilcoxon matched pairs statistic was used for comparing the CHI group with controls. Differences between groups were considered significant at a two-tailed P value of $\leqslant 0.05$. The analysis comprised 30 matched pairs, with the exception of the smooth pursuit data which included only 28 matched pairs due to technical difficulties during the testing of two participants.

The associations of the IQ difference between the groups (the verbal score in particular) with any detected neuropsychological or motor deficits were explored using linear regression analysis. For this regression analysis, we calculated the IQ difference of all pairs and defined these differences as the independent variable, with the dependent variable being the corresponding pair differences of another cognitive or motor measure. The resulting regression coefficients quantified the strength of the association between the IQ differences and the respective dependent variables.

While this study examined the effect of mild CHI on a number of motor functions and neuropsychological performance, the measures fall into three distinct domains of oculomotor, visuomotor or neuropsychological functional requirements, evaluating different categories of motor performance such as reaction times, motor accuracy, movement velocity and response errors as well as separate neuropsychological aspects of attention, memory and speed of information processing. Each of these categories contains a number of related measures and, therefore, we did not incorporate any correction for multiple comparisons to avoid type I errors.

Table 1 *Saccades: latency, directional errors, accuracy and velocity*

Measure	CHI (n = 30)		Controls (n = 30)		P level
	Mean	SD	Mean	SD	
Latency (ms)					
Reflexive saccades	179	20	177	20	0.550
Antisaccades	279	67	252	42	0.062
Prosaccade errors	203	46	179	29	0.013
Prosaccade correction	162	105	139	82	0.110
Intersaccadic interval of self-paced saccades	560	140	494	110	0.042
No. of self-paced saccades	52.7	12.6	62.4	13.1	0.003
Directional errors (%)					
Antisaccades	27.4	19	19.0	12	0.055
Memory-guided sequences	10.4	11	2.6	5	0.003
Accuracy					
Primary saccade gain (G_p)					
Reflexive saccades	0.97	0.04	0.98	0.05	0.308
Antisaccades	1.56	0.60	1.33	0.41	0.085
Memory-guided sequences	1.11	0.30	0.96	0.18	0.019
Gain final eye position (G_f)					
Reflexive saccades	1.00	0.02	1.01	0.04	0.05
Antisaccades	1.49	0.48	1.21	0.25	0.011
Memory-guided sequences	1.35	0.42	1.13	0.20	0.016
Self-paced saccades	1.02	0.09	1.01	0.06	0.861
Position error (PE, %)					
Reflexive saccades	6.33	2.9	6.37	4.1	0.557
Antisaccades	63.2	44	34.1	20	0.001
Memory-guided sequences	57.1	45	33.0	17	0.006
Self-paced saccades	9.51	8.2	7.57	4.4	0.206
Velocity (°/s)					
Reflexive saccades					
12°	409	39	419	45	0.428
14°	454	57	463	51	0.813
16°	452	58	459	56	0.797
18°	493	60	491	55	0.530
20°	499	65	510	65	0.781
22°	521	55	531	61	0.585
24°	514	59	533	67	0.455
Antisaccades					
5°	321	119	333	86	0.338
15°	322	96	344	107	0.428
Self-paced saccades					
30°	562	88	603	73	0.078

Results

Motor measures

Saccadic reaction times (Table 1)

There was no difference between the groups in latencies for reflexive saccades. Conversely, the CHI group demonstrated prolonged latencies of directional errors (i.e. erroneous prosaccades) in the antisaccade task, longer intersaccadic intervals for self-pacing, corresponding to fewer self-paced saccades within 30 s, and a trend to longer antisaccade latencies. No deficit was detected on the correction times of the directional errors in the antisaccade task.

Saccadic velocity (Table 1)

The CHI group showed normal saccadic velocities of reflexive saccades, antisaccades and self-paced saccades.

Saccadic directional errors (Table 1)

In the antisaccade paradigm, the CHI group tended to show a higher number of directional errors and a clear increase in the number of directional errors in the sequences of memory-guided saccades.

Saccadic motor accuracy (Table 1)

The CHI group exhibited normal spatial accuracy on reflexive saccades. Conversely, the CHI group showed markedly poorer spatial accuracy on the mean absolute position error of final eye position on antisaccades and sequences of memory-guided saccades. The mean gain of antisaccade final eye position was abnormally hypermetric and, on memory-guided sequences, both the primary saccade gain and the final saccade gain showed significant hypermetria.

Table 2 *Oculomotor smooth pursuit*

Measure	CHI (n = 28)		Controls (n = 28)		P level
	Mean	SD	Mean	SD	
20°/s					
Average peak velocity	19.1	1.9	19.6	1.7	0.375
Lag (ms)	–24.3	51.5	–14.8	30.8	0.649
40°/s					
Average peak velocity	38.9	3.0	39.1	3.4	0.187
Lag (ms)	–12.4	23.6	–10.5	19.2	0.967
60°/s					
Average peak velocity	53.8	7.9	53.5	7.3	0.829
Lag (ms)	28.1	29.0	27.0	32.8	0.919
Random					
Average peak velocity	30.0	5.6	31.6	6.5	0.618
Lag (ms)	46.8	21.4	31.1	26.7	0.028

A more detailed analysis of the memory-guided sequences of saccades in our groups has been published elsewhere (Heitger *et al*., 2002). In summary, the results of that analysis showed that the impaired motor accuracy and hypermetria of the CHI group on memory-guided sequences remained following a split into individual sequence steps and independent amplitude tiers, further revealing an inverse relationship between amplitude size and magnitude of position errors. In addition, hypermetria was more pronounced for smaller saccade amplitudes. These deficits in the CHI group were also present in shortened memory-guided sequences. We found no deficits regarding number of saccades required to complete each sequence or temporal accuracy (timing and rhythm).

Oculomotor smooth pursuit (Table 2)

No impairment was seen in the sinusoidal smooth pursuit performance of the CHI group. However, the CHI subjects showed increased lag on the most difficult task, the random smooth pursuit, even though there was no difference in random average peak velocity.

Visuoperception and upper-limb visuomotor performance (Table 3)

All subjects had a visual acuity of ⩾6/9 on the Snellen chart and there was no group difference. The only visuoperceptual difference between groups was on static perception. On basic motor function, the CHI group had a reduced arm movement peak velocity, whereas arm movement reaction time and arm movement steadiness were not found to be impaired.

Consistent deficits were present on the 1D tracking tasks evidenced by larger mean absolute errors on sine, random, sine with preview and step tracking. The CHI group also showed a longer lag on step tracking and sine tracking.

Neuropsychological measures (Table 4)

On the PASAT, the CHI group performed worse on the fastest (1.2 s) pacing while the impairments on the slower paces were, at best, marginal. The CHI group also performed less well on the SDMT and had longer time to completion scores on the TMT B. No difference was found on the time to completion scores of TMT A. The CHI group made fewer errors on the TMT A, whereas there was no significant difference in error rate on the TMT B. The CHI group also achieved lower performance on several measures of the CVLT. The CHI group had a lower full-scale WASI IQ, which was primarily due to a lower Vocabulary Test score.

Associations between (verbal) IQ and other measures

While the performance on tests for both verbal and performance IQ may be impaired following mild head trauma, the literature on this topic indicates that performance IQ usually shows the adverse effects of head trauma to a larger extent than verbal IQ (e.g. Crosson *et al*., 1990; Reitan and Wolfson, 1997; Richardson, 2000). Finding the opposite (preserved performance IQ and impaired verbal IQ) in our study suggests that the observed IQ difference may have been due to an unexpected selection bias, with the control group having a higher IQ even compared with the pre-morbid IQ of the CHI group. We therefore used linear regression analysis to explore further whether the motor deficits and the poorer neuropsychological performance were associated with the IQ difference between our groups.

Our analysis confirmed that the difference in full WASI IQ was caused by the differences on the WASI Vocabulary Test. Consequently, we concentrated on the Vocabulary T score as a measure of verbal IQ. Although the difference in years of education was marginally significant (12.8 versus 13.2, $P = 0.054$), this variable accounted for <6% of the variation in verbal IQ ($R^2 = 0.055$), and the group difference in verbal IQ remained after controlling for years of education ($P = 0.007$).

Table 3 *Visuoperceptual and upper-limb visuomotor measures*

Measure	CHI (n = 30)		Controls (n = 30)		P level
	Mean	SD	Mean	SD	
Visuoperceptual tests					
Visual acuity (Snellen denominator)	5.56	1.4	5.30	1.1	0.422
Visual resolution (mm)	0.42	0.1	0.42	0.1	1
Arrow perception (no. correct)	12.97	0.2	13	0.0	0.32
Static perception (mm)	1.30	0.8	1.01	0.1	0.01
Dynamic perception (mm)	1.78	0.6	1.61	0.6	0.3
Basic motor function					
Reaction time (ms)	294	32.9	280	31.3	0.132
Movement peak velocity (mm/s)	821	246	949	225	0.033
Steadiness (mm/s)	2.98	1.12	2.63	1.00	0.153
Tracking mean absolute error (mm)					
Sine tracking	6.96	1.7	5.78	1.5	0.003
Random tracking	6.96	1.8	6.18	1.5	0.017
Sine tracking with preview	8.45	2.6	7.25	2.3	0.021
Random tracking with preview	6.2	2.3	5.37	1.6	0.11
Step tracking	11.34	1.5	10.37	1.1	0.002
Tracking mean lag (ms)					
Sine tracking	68.6	31.9	57.8	28.8	0.051
Random tracking	115.4	39.3	106.3	35.5	0.161
Sine tracking with preview	63.4	68.4	62.4	47.6	0.942
Random tracking with preview	89.6	61.9	82.4	63.4	0.942
Step tracking	642.0	102.9	588.8	57.9	0.013

Table 4 *Neuropsychological measures*

Measure	CHI (n = 30)		Controls (n = 30)		P level
	Mean	SD	Mean	SD	
PASAT (Z–score)					
2.4 s pacing	–0.74	1.11	–0.35	1.19	0.063
2.0 s pacing	–0.64	0.89	–0.27	0.95	0.139
1.6 s pacing	–0.53	0.83	–0.16	0.81	0.091
1.2 s pacing	–0.57	0.75	–0.21	0.81	0.039
SDMT (Z-score)	0.11	1.04	0.73	0.97	0.020
Trail making					
Time test A (s)	24.67	18.16	22.53	5.85	0.133
Time test B (s)	62.60	18.16	52.47	17.42	0.020
Errors test A	0.13	0.35	0.53	0.82	0.016
Errors test B	0.38	0.68	0.33	0.61	0.875
WASI					
WASI IQ	106.87	13.27	114.83	11.91	0.008
Vocabulary T score	52.70	9.37	59.17	7.43	0.002
Matrix T score	54.80	7.74	57.53	7.65	0.073
CVLT (Z-score)					
Total standard score	42.40	12.66	53.83	11.10	0.003
Trial 1	–0.37	1.16	0.03	0.89	0.210
Trial 5	–1.40	1.61	–0.10	1.35	0.008
List B	–0.30	1.15	0.07	1.23	0.226
Short delay free recall	–1.33	1.15	0.10	1.09	0.001
Short delay cued recall	–1.27	1.26	–0.27	1.20	0.010
Long delay free recall	–1.10	1.37	–0.33	1.03	0.028
Long delay cued recall	–1.47	1.22	–0.50	1.11	0.007
Semantic cluster	–0.93	0.98	–0.23	1.19	0.019
Serial cluster	0.30	1.09	–0.07	1.14	0.183
Recall consistency	–0.37	1.03	0.10	0.84	0.070
Recognition hits	–0.97	1.56	–0.37	0.85	0.111
Recognition discriminability	–0.27	0.52	–0.03	0.18	0.028

Table 5 *Associations between WASI vocabulary T score and neuropsychological measures*

Neuropsychological measures	Regression β coefficient	*P* level
PASAT (*Z*-score)		
2.4 s pacing	0.44	0.02
2.0 s pacing	0.36	0.05
1.6 s pacing	0.51	0.004
1.2 s pacing*	0.47	0.01
SDMT (*Z*-score)	0.56	0.001
Trail making		
Time test A	–0.07	0.69
Time test B*	0.32	0.09
CVLT (*Z*-score)*		
Total standard*	0.49	0.01
Trial 1	0.30	0.10
Trial 5*	0.47	0.01
List B	0.33	0.07
Short delay free*	0.31	0.10
Short delay cued*	0.30	0.11
Long delay free*	0.34	0.07
Long delay cued*	0.31	0.09
Recall consistency	0.43	0.02
Semantic cluster*	0.07	0.70
Serial cluster	0.09	0.64
Recognition hits	–0.16	0.41
Recognition discriminability*	0.07	0.72

*Impaired in CHI group on Wilcoxon matched pairs analysis.

The linear regression analysis detected no significant associations between verbal or full WASI IQ and any of the oculomotor or visuomotor deficits (all *P* levels >0.2, with the exception of visuomotor sine tracking with $P = 0.08$). Conversely, we found significant associations between the IQ performance and most of the neuropsychological measures (Table 5), including the PASAT, SDMT and some measures of the CVLT, showing that IQ impacted significantly on the results of other neuropsychological tests. No significant association was found between verbal IQ and the TMTs A or B. Further analysis of the measures associated with IQ (which comprised neuropsychological measures only) showed that, in all cases, any significant differences between the groups disappeared after controlling for verbal IQ difference and no new significant effects emerged (Table 6).

Discussion

The results from this study indicate that mild CHI causes deficits in saccades and impaired upper-limb visuomotor function, despite there being no oculomotor or visuomotor deficits on standard clinical examination. Whilst the patient group also scored lower on several neuropsychological tests, most of these differences could be accounted for by the sampling-dependent IQ difference between the groups, which adversely affected the neuropsychological test results while having no significant effects on the motor performance. This indicates that the extent of head trauma-related cognitive deficits was marginal and that impairment of oculomotor and arm visuomotor function can occur independently of neuropsychological deficits following mild CHI.

The finding that our groups did not differ regarding visual acuity and accuracy of normal reflexive saccades suggests that the observed motor deficits were not due to a fault in the sensory system delivering visual information to the cerebral motor areas. This implies that the observed motor deficits were likely to be due to the impaired transformation of sensory input to motor output in key components of the cerebral motor systems.

Oculomotor deficits

There have been few previous studies on eye movement function following CHI. Mulhall *et al*. (1999) undertook bedside examinations of antisaccades, single memory-guided saccades and self-paced saccades in a group of 19 cases of severe head trauma, and the only significant difference was a lower number of self-paced saccades in the head-injured group. They compared their findings with results from infrared oculographic tests of saccades and concluded that bedside tests of saccades have only limited value in patients with head trauma. Williams *et al*. (1997) found saccade deficits in 16 patients with severe traumatic brain injury (mean PTA of 43.7 days). Their findings included prolonged latencies of reflexive saccades, antisaccades and simple memory-guided saccades, smaller numbers of self-paced saccades, hypometria of reflexive saccades and increased response errors on antisaccades and simple memory-guided saccades. While we detected saccadic deficits similar to Williams *et al*., the impairments of our CHI group were smaller in degree, compatible with the much less severe injury status of our patient group. Crevits *et al*. (2000) investigated latencies and response errors in single remembered saccades and antisaccades following mild CHI, but detected no saccadic deficits. Their selection criteria were similar to our own (GCS 13–15, PTA <24 h, impaired consciousness) with 25 non-intoxicated mild CHI patients and a separately analysed subgroup of six intoxicated cases. However, all cases showed the maximal GCS score of 15, only 15 had lost consciousness, none exceeded a PTA of 1 h and seven patients experienced no PTA at all. Thus, the likely reason why Crevits *et al*. failed to detect oculomotor deficits is that their final CHI group was milder than our own. Furthermore, their tasks were different from our paradigms, comprising long stimulus presentation times (3–5 s) and constant amplitudes, and thereby considerably easing response pressure for the subjects. These factors, we believe, are likely to have contributed to the negative results of Crevits *et al*. (2000).

Imaging studies and assessment of motor performance in patients with focal brain lesions or neurological disorders have led to a good understanding of the functional neuroanatomy of eye movements. The decreased accuracy and increased response errors in memory-guided sequences and antisaccade task are consistent with deficits originating in the

Table 6 *Corrected group differences for all measures sharing significant associations with IQ*

Measure	Corrected difference	*P* level	Group differences significantly different from zero after controlling for (verbal) IQ?
PASAT (Z-score)			
2.4 s pacing	–0.04	0.89	No
2.0 s pacing	0.04	0.88	No
1.6 s pacing	–0.02	0.92	No
1.2 s pacing	0.04	0.85	No
SDMT (*Z*-score)	0.09	0.73	No
CVLT (*Z*-score)			
Total standard	5.79	0.10	No
Trial 5	0.59	0.20	No
Recall consistency	0.08	0.78	No

posterior parietal cortex (PPC) (Gnadt and Andersen, 1988; Muri *et al*., 1996; Zhang and Barash, 2000; Heide *et al*., 2001), supplementary motor area (SMA) (Pierrot-Deseilligny *et al*., 1991*a*; Schlag-Rey *et al*., 1997), frontal eye field (FEF) (Guitton *et al*., 1985; Fukushima *et al*., 1994; Rivaud *et al*., 1994; Pierrot-Deseilligny *et al*., 1995; Ploner *et al*., 1999) and dorsolateral prefrontal cortex (DLPFC) (Pierrot-Deseilligny *et al*., 1991*a*; Brandt *et al*., 1998; Gaymard *et al*., 1998). The impaired CHI performance on sequences of memory-guided saccades emphasizes impaired function of the SMA (Gaymard *et al*., 1990; Muri *et al*., 1994, 1995). Interruption of input from the FEF and the DLPFC into the superior colliculus pathway has been suggested as a possible reason for significantly decreased rates of self-paced saccades following head trauma (Williams *et al*., 1997). The significantly lower number of self-paced saccades in our patient group indicates impaired function of frontal brain regions, particularly the FEF. Lesions in the FEF impair the ability to generate saccades but do not impair the ability to suppress erroneous prosaccades in the antisaccade task (Gaymard *et al*., 1999). This is consistent with the combination of fewer self-paced saccades but only marginally increased rates of erroneous prosaccades in our CHI group.

Prolonged saccadic latencies suggest frontal lobe damage (Pierrot-Deseilligny *et al*., 1991*b*; Fukushima *et al*., 1994; Rivaud *et al*., 1994; Terao *et al*., 1998; Gaymard *et al*., 1999; Connolly *et al*., 2002) although dysfunction of the PPC may also contribute to increased saccade latencies (Braun *et al*., 1992; Elkington *et al*., 1992; Terao *et al*., 1998). The discrepancy between normal latencies of simple reflexive saccades and prolonged latencies of erroneous prosaccades in the antisaccade task appears unusual, as these prosaccades are essentially also reflexive saccades by nature. While there are indications that erroneous prosaccades in the antisaccade task may have longer latencies than simple reflexive saccades (personal communication relating to Terao *et al*., 1998), there are no previous examples in the literature for the observed contrast between latencies of reflexive saccades and erroneous prosaccades in the antisaccade task. Thus, we can only speculate on the underlying cause of the prolonged prosaccade latencies in our CHI group. The execution of such prosaccades is significantly influenced by the context and increased attentional complexity of the antisaccade task, which comprises diversion of attention away from the stimulus as well as activated response inhibition, and by the concurrent top-down process of antisaccade preparation, which involves different activation patterns in frontal and parietal brain regions compared with intentional prosaccades (Sweeney *et al*., 1996; Doricchi *et al*., 1997; Schlag-Rey *et al*., 1997; Everling *et al*., 1998; Connolly *et al*., 2002). We suggest that CHI impaired the synchronization of the simultaneous processes of antisaccade preparation and prosaccade inhibition, whereby the antisaccade preparation is initiated and in progress when prosaccade inhibition fails, triggering the termination of the antisaccade preparation and the initiation of a delayed prosaccade. This effect may become even stronger if response inhibition is only marginally impaired, as observed in our CHI group, causing the prosaccade inhibition to fail mostly in the late stages of antisaccade preparation.

While the FEF and the parietal eye field (PEF) act complementarily in the triggering of saccades, the initiation of reflexive visually guided saccades appears to be mediated predominantly by the PEF (Pierrot-Deseilligny *et al*., 2002). Consequently, the normal latency of reflexive saccades in the CHI group may suggest preservation of PEF function. However, the posterior parietal cortex also represents the cortical substrate for the visuospatial transformation and integration of primary visuosensory information from the striate cortex, this being essential for accurate eye and arm movement and involving multiple areas within the PPC (Pierrot-Deseilligny *et al*., 2003*b*). Zhang and Barash (2000) showed that the lateral intraparietal area contributes to sensorimotor transformations for antisaccades, and transcranial magnetic stimulation (TMS) over the PPC has been found to prolong antisaccade latencies if applied within 80–100 ms of stimulus onset (Terao *et al*., 1998). Recordings of cerebral event-related potentials have shown that the parietal

cortex participates in generating a neural representation of the antisaccade stimulus in the hemifield ipsilateral to the stimulus before saccade generation (Everling *et al*., 1998). However, visuospatial transformation and positional coding in the PPC appear to be most crucial for the execution and accuracy of memory-guided sequences of saccades, with functional MRI showing activation of multiple foci within the PPC during triple-step memory-guided sequences as used in our study (Heide *et al*., 2001). TMS over the PPC causes inaccuracy of memory-guided saccades (Oyachi and Ohtsuka, 1995), and Heide *et al*. (1995, 1998) showed that lesions affecting the PPC cause saccadic dysmetria in memory-guided sequences. The induction of 'artificial lesions' via application of TMS over the PPC during sequenced saccades similarly produces saccadic inaccuracy (van Donkelaar and Muri, 2002) and further impairs visuospatial transformation in the case of arm movements (van Donkelaar *et al*., 2002). Both Heide *et al*. (2001) and van Donkelaar *et al*. (2002) proposed that the PPC is crucial for spatial constancy across saccades and is a key substrate for the necessary visuospatial transformations. These spatial computations include the use of extraretinal information (efference copy) on saccadic eye displacement for updating the spatial representation of consecutive targets within a sequence. Based on the results of a recent study on memory-guided saccade sequences in 10 patients with PPC lesions, Heide *et al*. (2003) concluded that the observed spatial inaccuracy of saccades was due to a deficit in computing the efference copy signal for a current saccade, resulting in faulty spatial referencing and inaccurate updating of the retinal location for the next saccade target. In the light of these findings, the significant saccadic inaccuracy of memory-guided sequences and antisaccades, in combination with significant upper-limb inaccuracy, in our CHI group suggests impaired function of the PPC.

In summary, the results of the saccade tasks indicate that motor functions originating in frontal regions such as the FEF and SMA were likely to be impaired in our patient group either by direct injury or by deafferentation due to diffuse axonal injury. Conversely, the marginal deficit of the CHI group on directional errors in the antisaccade task suggests only limited damage to the DLPFC, as lesions to prefrontal areas are likely to cause significant impairments on the directional error rate in the antisaccade task (Walker *et al*., 1998; Pierrot-Deseilligny *et al*., 2003*a*). The poorer motor accuracy of antisaccades and, in particular, sequences of memory-guided saccades further suggests that the function of parietal areas such as the PPC was also adversely affected.

The near normal oculomotor smooth pursuit performance suggests that deeper brain regions, temporal areas such as the middle temporal and medial superior temporal area, and the cerebellum are largely spared from damage in mild CHI (Tusa and Ungerleider, 1988; Thier *et al*., 1991; Pierrot-Deseilligny and Gaymard, 1992; Morrow and Sharpe, 1993). Further, the cerebellar vermis mediates the subconscious adaptation of reflexive saccades (Desmurget *et al*., 2000), which has also been demonstrated to be unaffected in mild CHI (Heitger *et al*., 2001).

Saccadic peak velocity has been applied previously as a measure for function of the reticular brainstem formation (Bittencourt *et al*., 1981). The finding of preserved saccadic velocities in our CHI group suggests minimal damage to deeper subcortical structures and the brainstem. Thus, our results support the notion of a centripetal gradient of impact forces causing neural damage in CHI (Wilson, 1990), consistent with research on the biomechanics of CHI (Wilson, 1990; Ommaya, 1995).

Upper-limb visuomotor deficits

There is only limited previous evidence of impaired upper-limb visuomotor function following CHI despite substantial evidence of impairment of upper-limb visuomotor performance in patients with focal lesions or neurological disorders (e.g. Oepen *et al*., 1985; Jones *et al*., 1989, 1996; Hocherman and Giladi, 1998; Schwartz *et al*., 1999). Jones and Donaldson (1981) showed the adverse effects of severe traumatic brain injury on upper-limb motor function using early versions of the visuomotor tests used in this study. Chistyakov *et al*. (1998) used TMS to demonstrate that minor head injury can alter motor cortex excitability, which is likely to adversely affect visuomotor performance, although they did not demonstrate this quantitatively. Ingersoll (1993), looking at posture control following sport-related CHI, suggested that somatosensory input appears to be improperly processed following CHI.

There is a widespread neural network for limb motor processing involving multiple corticocortical connections. Visuomotor control simultaneously engages functionally related frontal and parietal areas linked by corticocortical connections (Caminiti *et al*., 1998). Several studies have emphasized the importance of parietal areas for motor control (Andersen, 1995; DeSouza *et al*., 2000; Ferraina *et al*., 2001). Primate- and human-based research has shown that other areas such as the SMA (Ohara *et al*., 2000), pre-SMA (Picard and Strick, 1996) and the dorsal and ventral premotor cortex (Kurata, 1994) are essential for movement-related visuomotor coordination, with specialization of the ventral premotor cortex for motor execution under visual guidance. The cerebellum also projects to the primary motor cortex via the thalamus, and thereby shares a function in motor programming of limb movements. Purkinje cell populations in the cerebellar cortex contribute substantially to the encoding of (non-ballistic) arm movement velocity (Coltz *et al*., 1999). Consequently, the finding of intact arm movement steadiness in the CHI group further supports the notion of preserved cerebellar function.

We propose that deficits in upper-limb visuomotor performance are likely to have their origin in impaired sensory input transformation in the PPC or premotor cortex, impaired function of the premotor areas or damage to corticocortical connections between frontal and parietal motor areas. This

interpretation would be consistent with the oculomotor abnormalities suggesting functional damage mainly to frontal and parietal brain regions. While visual resolution was preserved and perception remained unaffected in two out of three perceptual tasks in the visuomotor battery, the CHI group demonstrated a deficit on the static perception of the visuomotor target reference. This observation suggests that perceptual transformations of the visuomotor target display in the PPC were adversely affected, given that the normal reflexive saccade accuracy implies undamaged visuosensory input systems. However, it is unclear, especially as the more complex dynamic perception was preserved in our CHI group, whether the apparent deficit in static perception impacted significantly on the visuomotor 1D tracking tasks, which are also of dynamic nature.

Neuropsychological factors

The initial Wilcoxon matched pairs analysis revealed significantly lower scores of the CHI group on the PASAT, SDMT, TMT B, CVLT and the WASI, consistent with previous evidence of neuropsychological deficits following mild CHI (Bassett and Slater, 1990; Bohnen *et al*., 1992; Levin *et al*., 1992; Arcia and Gualtieri, 1993, 1994; Macciocchi *et al*., 1996; Parker and Rosenblum, 1996; Tremont *et al*., 1997; Tiller and Persinger, 1998; Wallesch *et al*., 2001). It was evident, however, that these specific differences were due to an unexpected difference in verbal IQ between the groups. Our findings were therefore similar to those of other reports, such as Taylor *et al*. (1996), who examined whiplash patients and found no differences on neuropsychological tests such as the PASAT once IQ has been controlled for.

These findings indicate that the apparent CHI deficits on PASAT, SDMT and several measures of the CVLT were not triggered primarily by the impact of mild CHI and that the levels of head injury-related neuropsychological impairment in the CHI group were marginal and much smaller than evident from the primary Wilcoxon matched pairs analysis. These results generally indicate good levels of cognitive functioning, attention, speed of information processing and short-term/working memory in our CHI group and support the notion that any cognitive deficits had, at most, a minor impact on motor performance. The oculomotor and upper-limb visuomotor deficits were, as evident from the linear regression analysis, unrelated to the levels of neuropsychological performance including IQ.

Conclusions

This study shows that mild CHI impairs multiple motor systems. The nature of the observed motor deficits indicates that mild CHI impairs motor functions originating predominantly in frontal and dorso-parietal brain regions, and that deeper subcortical areas, the occipital lobe and the cerebellum, are largely spared from damage.

The current findings indicate that abnormalities of saccades and upper-limb visuomotor function following CHI may provide sensitive markers of cerebral dysfunction, independent of psychometric status. We believe that sensitive computerized motor testing may have the potential to supplement established methods for CHI patient assessment and that further studies are warranted to determine whether such motor testing might help to predict and to track recovery after mild CHI.

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