

# Saccadic eye movement disturbances in whiplash patients with persistent complaints

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## Summary

In order to analyse the possible basis of subjective complaints following whiplash injury, horizontal eye movements were examined in subjects with persistent complaints ('symptomatic group') and subjects who had completely recovered ('recovered group'). The results for the symptomatic and recovered groups were compared with those for age-matched, healthy volunteers (control group). A battery of different saccade paradigms was employed: two were reflexive saccade tasks including a gap and an overlap task, and two were intentional saccade tasks consisting of an antisaccade and a memory-guided saccade task. In addition, the symptomatic and recovered groups also underwent psychiatric evaluation in a structured clinical interview, and all groups were assessed for emotional functioning using the Beck Depression Inventory (BDI). The recovered group did not differ significantly from the control group in saccade performance and emotional functioning. The symptomatic group showed dissociation of their performances of

reflexive and intentional saccade tasks: performance in reflexive saccade tasks was normal, but in intentional saccade tasks the symptomatic group showed significantly impaired inhibition of unwanted reflexive saccades, impaired saccade triggering (i.e. increased latency) and a higher percentage error in amplitude in memory-guided saccades. Based on clinical interviews, no signs of major depression or dysthymia were found in any of the groups. Compared with the other two groups, the symptomatic group had significantly higher overall BDI scores, but these resulted from BDI dimensions that were non-specific to depression, viz. 'physiological manifestations' (e.g. fatigue, sleep disturbance) or 'performance difficulty' (e.g. work inhibition). In summary, in the symptomatic group the pattern of eye movement disturbances together with normal performance in reflexive saccade tasks and impaired performance in the intentional saccade tasks, especially impaired inhibitory function, suggests dysfunction of prefrontal and frontal cortical structures.

**Keywords:** eye movement disturbance; whiplash injury; cortex

**Abbreviations:** BDI = Beck Depression Inventory; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders; F = central fixation point; PEA = percentage error in amplitude; T = amplitude of the visual target

## Introduction

Whiplash injury of the cervical spine generally shows good recovery (Radanov *et al.*, 1995; Spitzer *et al.*, 1995), only about 20% of injured patients suffering from long-lasting complaints such as neck pain, poor concentration and dizziness (Radanov *et al.*, 1995). Some controversy still surrounds the basis on which symptoms following whiplash develop. In particular, there is considerable disagreement with regard to the contribution of psychosocial factors (e.g. personality traits) in determining the outcome (Radanov, 1997). In prospective studies, psychosocial factors did not prove to be of primary relevance to the outcome (Mayou *et al.*, 1993; Radanov *et al.*, 1995), indicating the possibility of selection bias in previous research. In addition, many

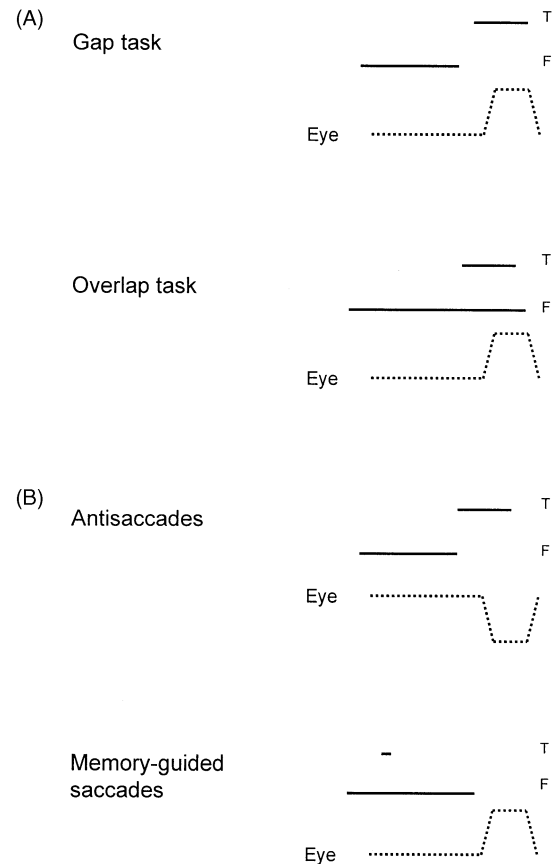
previous studies used a rather broad definition of whiplash, resulting in the inclusion of patients with possible traumatic brain injury (i.e. acceleration–deceleration trauma with loss of consciousness). This may lead to difficulties when comparing factors contributing to the outcome in different studies (Radanov, 1997).

Several studies showed impaired emotional or cognitive functioning in the long term following whiplash (Radanov *et al.*, 1989, 1993, 1996; Ettlin *et al.*, 1992; Wallis *et al.*, 1997). While CT or MRI studies did not detect brain lesions in symptomatic whiplash patients (Ettlin *et al.*, 1992), previous SPECT (single photon emission computed tomography) and PET studies have found hypoperfusion of

the parieto-occipital regions (Otte *et al.*, 1996, 1997). In a recent study, significant differences in perfusion of the frontal-polar region, the lateral temporal cortex and the putamen were found (Bicik *et al.*, 1998). The functional disturbance in the more rostral brain areas (Bicik *et al.*, 1998) is in accordance with experimental findings after acceleration-deceleration trauma (Ommaya and Gennarelli, 1975), in which lesions were primarily found in these same brain regions.

About 25% of whiplash patients complain of dizziness in the long term (Radanov *et al.*, 1995). This complaint remains difficult to explain (Toglia, 1976; Gimse *et al.*, 1996). Studies concerning eye movement disturbances in whiplash patients have been aimed mainly at uncovering vestibular or brainstem dysfunction (Toglia, 1976; Hildingsson *et al.*, 1989; Oosterveld *et al.*, 1991; Gimse *et al.*, 1996; Tjell and Rosenhall, 1998). However, there is considerable controversy as to whether whiplash trauma results in any relevant or objective otoneurological abnormality. Studies by Toglia and by Oosterveld and colleagues found a high incidence of spontaneous nystagmus, caloric abnormalities and rotational response derangement using electronystagmography (Toglia, 1976; Oosterveld *et al.*, 1991). The reliability of these findings is questionable, as spontaneous nystagmus may be observed during eye closure and positional nystagmus is found in a significant number of normal subjects (Luxon, 1996). In addition, recent studies (Fischer *et al.*, 1995; Gimse *et al.*, 1996) found no nystagmus in whiplash patients.

Considering the observation of possible dysfunction of rostral brain areas after acceleration-deceleration (Bicik *et al.*, 1998), it may be promising to focus on cortical instead of vestibular control of ocular motor performance in whiplash patients. In recent years, substantial advances have been made in knowledge regarding the cortical organization of saccade control. The functional distinction between reflexive and intentional saccades is relevant because different cortical regions are involved in their control (Leigh and Zee, 1999). Reflexive saccades (Fig. 1) are externally triggered by the sudden appearance of a visual target. Intentional or volitional saccades are triggered internally by the subject and are goal-oriented, and can be tested with antisaccade and memory-guided saccade tasks. In the antisaccade task, the subject has to perform a saccade in the direction opposite to that of a suddenly appearing visual target. Correct performance of antisaccades, therefore, requires the inhibition of an unwanted reflexive saccade towards the visual target and the generation of a voluntary saccade to the opposite side (Everling and Fischer, 1998). In the memory-guided saccade task, the subject makes a saccade towards the remembered position of a previously flashed target (Goldman-Rakic, 1987). As summarized earlier (Pierrot-Deseilligny *et al.*, 1995), lesion studies showed differences between frontal and parietal control of saccades: frontal cortical dysfunction, especially that involving prefrontal areas, showed impaired inhibitory function and longer latencies in intentional saccade tasks while the latencies of reflexive saccades were, if anything,



**Fig. 1** Schematic drawings of the reflexive (A) and intentional (B) saccade paradigms. In the gap task the central fixation point (F) was switched off 200 ms before the onset of the lateral visual target (T). In the overlap task F remained visible during the presentation of the lateral target. In both tasks the subjects fixated F and performed a saccade to T as soon as T appeared. In the antisaccade task the presentation of F was the same as in the gap task, but the instruction was to look in the opposite direction. In the memory-guided saccade task a lateral target was flashed for 50 ms and was followed by a memorization period while F remained on. The subject had to fixate F and memorize the exact location of T. The extinction of F was the signal to perform a saccade to the location of T. Dotted lines indicate eye position; bold lines indicate target position.

slightly increased (Guitton *et al.*, 1985; Pierrot-Deseilligny *et al.*, 1991; Rivaud *et al.*, 1994). In contrast, parietal dysfunctions do not impair inhibitory function but may increase all latencies (i.e. of reflexive and intentional saccades) independently of the saccade task (Pierrot-Deseilligny *et al.*, 1987, 1991). Therefore a pattern of disturbance of saccadic eye movement control may have value in localizing dysfunction.

The aim of the present study was to analyse possible disturbances of the cortical control of saccades. For this reason, a battery of saccade paradigms, including reflexive saccade and intentional saccade tasks, were employed. If whiplash patients show a particular pattern of disturbance of the cortical control of saccades, the issue of interest may be whether the disturbances have topological value and can be explained by cortical dysfunction.

## Material and methods

### Patients and controls

The inclusion criteria for this study were based on a history of whiplash injury. Whiplash was defined strictly, according to previous research (Hirsch *et al.*, 1988; Radanov *et al.*, 1995; Spitzer *et al.*, 1995), as follows: musculoligamental strain or sprain of the cervical spine due to acceleration–deceleration, without fractures or dislocations of the cervical spine, head contact injury, loss of consciousness or post-traumatic amnesia. In addition, all patients had to discontinue all medication (except contraceptive medication) 24 h before testing in order to avoid a possible psychotropic effect on saccadic performance (e.g. sedation). Table 1 shows a synopsis of the present medications of all patients. In addition, all subjects in the study were required to have German as their native language because all test instructions were in German.

Exclusion criteria were as follows: (i) injuries to other parts of the body; (ii) a history of previous neurological dysfunction; and (iii) mood disorder (e.g. dysthymia or major depression).

Two groups with a history of whiplash injury and a control group were examined. (i) The ‘symptomatic group’ consisted of 11 subjects (eight women and three men; mean age 38.8 years, range 21–54 years) complaining of persistent dizziness of non-specific character, poor concentration, and neck pain or headache. The interval between the accident and the examination in this group averaged 2.8 years (range 1 month to 6 years). (ii) The ‘recovered group’ consisted of 10 subjects (four women and six men; mean age 37 years, range 20–52 years) with a history of whiplash injury and initial complaints, who had recovered completely at the time of examination. The mean interval between the accident and examination averaged 3.0 years (range 1 month to 7 years). (iii) The age-matched control group consisted of 16 healthy volunteers (12 women and four men; mean age, 36.5 years, range 23–64 years).

The Ethics committee of the Faculty of Medicine, University of Berne, approved the study and all subjects gave their informed consent before the investigation.

### Measurements of saccadic eye movements

Eye movements were recorded with an infrared reflection method, described in detail elsewhere (Hess *et al.*, 1986). The subjects were placed in complete darkness during the experiments and the head was fixed to avoid concomitant head movement. The recording system was calibrated before each experiment. The spatial resolution of the system was 0.5°. The instructions were given in the same way for all subjects. To avoid fatigue, the tasks were presented in short blocks of 5–10 saccades, and the subjects were frequently reminded to be attentive. Each subject individually determined the length of the break between two blocks of tasks.

**Table 1** Synopsis of present medication of the three groups

Subject	Sex	Age (years)	Medication
Control group			
1	M	23	
2	M	23	
3	M	24	
4	F	26	*
5	F	28	*
6	F	28	*
7	M	29	
8	F	31	
9	F	36	
10	F	36	Paracetamol <sup>†</sup>
11	F	37	
12	F	44	
13	F	48	*
14	F	50	Acetylsalicylic acid <sup>†</sup>
15	F	58	
16	F	64	
Symptomatic group			
1	M	21	Paracetamol*
2	F	29	Paracetamol
3	F	31	Paracetamol
4	F	32	Metamizole sodium <sup>‡</sup>
5	F	32	Ibuprofen <sup>‡</sup>
6	F	34	
7	F	38	
8	F	51	
9	F	52	Oxazepam <sup>‡</sup>
10	M	53	Dihydroergotamine <sup>‡</sup>
11	M	54	
Recovered group			
1	F	20	*
2	M	21	
3	F	36	
4	M	36	
5	M	38	Paracetamol <sup>‡</sup>
6	F	38	
7	M	40	
8	F	42	
9	M	49	
10	M	52	

All medication, apart from contraceptives, was stopped 24 h before examination. F = female; M = male. \*Women taking contraceptive medication; <sup>†</sup>analgesic medication for headache >48 h before the examination; <sup>‡</sup>medication not on a regular basis.

### Tested saccade paradigms (Fig. 1)

**Reflexive saccades.** Two paradigms were tested. (i) In the gap task, the central fixation point (F) was switched off 200 ms (the temporal gap) before the appearance of the lateral visual target (T) 15° to the right or left of F. The direction of T and during the period when F was on were unpredictable. The subject was instructed to fixate F and to look at T as soon as it appeared. (ii) In the overlap task, F remained visible during the presentation of T. The instruction was to look at T immediately after it appeared.

**Intentional or voluntary saccades.** Two paradigms were tested. (i) In the antisaccade task, the presentation of

the visual target was the same as for the gap condition but the instruction was different. The subject was instructed to look not at T but in the opposite direction, away from the lateral visual target. Saccades towards the visual target were considered as errors. The mean percentage of errors was calculated. Furthermore, mean latencies of correct and erroneous saccades were calculated. Forty trials were performed. (ii) In the memory-guided saccade task, the subject was instructed to fixate F until it was switched off, without looking at the briefly flashed target to the left or right of F. While F was on, T was flashed for 50 ms in an unpredictable direction with respect to F. During the memorization period of 2 s, F remained on and the subject had to memorize the exact location of T. The extinction of F was the signal to perform the saccade to the place where T had previously appeared. Unwanted reflexive saccades were considered as saccades towards T in the memorization period.

Mean saccade latency and median percentage error in amplitude (PEA) (i.e. the absolute value of the amplitude of the subjects' first saccade minus the amplitude of the target saccade T divided by  $T \times 100$ ) were calculated by averaging at least 15 saccades in each direction for each subject and for all tasks. The mean percentage of unwanted reflexive saccades in intentional saccade tasks was also calculated. Saccade paradigms were analysed using the Kruskal–Wallis  $\chi^2$  test to compare the three groups, and two-group analysis was performed using the Mann–Whitney *U*-test (two-tailed).

### ***Emotional assessment***

To satisfy exclusion criteria, before saccadic eye movement assessment the symptomatic and recovered groups underwent an in-depth clinical interview by an experienced psychiatrist (BPR) with particular focus on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria of depression or dysthymic disorder (American Psychiatric Association, 1994). In addition, all subjects were evaluated using the revised German version of the Beck Depression Inventory (BDI) (Beck and Steer, 1987; Hautzinger *et al.*, 1995), which is self-rating and consists of 21 groups of items. The score ranged from 0 to 3 for each of the 21 groups of items, and the overall BDI score ranged from 0 to 63. Since several BDI items have a somatic content (e.g. fatigue, sleep disturbance), such items are scored by both depressed and non-depressed subjects with somatic complaints, and the overall BDI score may therefore misleadingly indicate the existence of depression in a population with somatic illness (Williams and Richardson, 1993; Chibnall and Tait, 1994; Novy *et al.*, 1995). In order to analyse the BDI results, the items were arranged into the following three dimensions, according to previous research (Novy *et al.*, 1995): (dimension 1) 'negative attitude and suicide', including items with depressive cognition (e.g. sadness, pessimism); (dimension 2) 'performance difficulty', consisting of items such as work inhibition and indecisiveness; (dimension 3) 'physiological manifestations', including items with somatic

content (e.g. insomnia, fatigue, weight loss). Statistical analysis of BDI data was performed in three steps. (i) The three groups of subjects were compared with respect to their overall BDI score using the Kruskal–Wallis test. (ii) The mean score of the three BDI dimensions and their 95% confidence intervals were calculated for each group. (iii) To investigate which of the three BDI dimensions may have contributed to group differences, the following procedure was applied. The average scores for each of the three dimensions of the control group were considered as zero points. The individual scores of the three dimensions of symptomatic and recovered patients were subtracted from the means of the corresponding dimensions of the control group. The difference between the established zero point and the score of individuals from symptomatic and recovered groups thus reflects an absolute score difference from the mean of the control group. On this basis, using the Friedman test the differences between dimensions 1, 2 and 3 were calculated for the symptomatic and recovered groups. To test the differences with respect to dimensions between pairs within the groups, the Wilcoxon signed rank test was used. The size of the pair differences was considered to be a measure of the difference from the control group.

## **Results**

### ***Analysis of saccadic eye movement measurements***

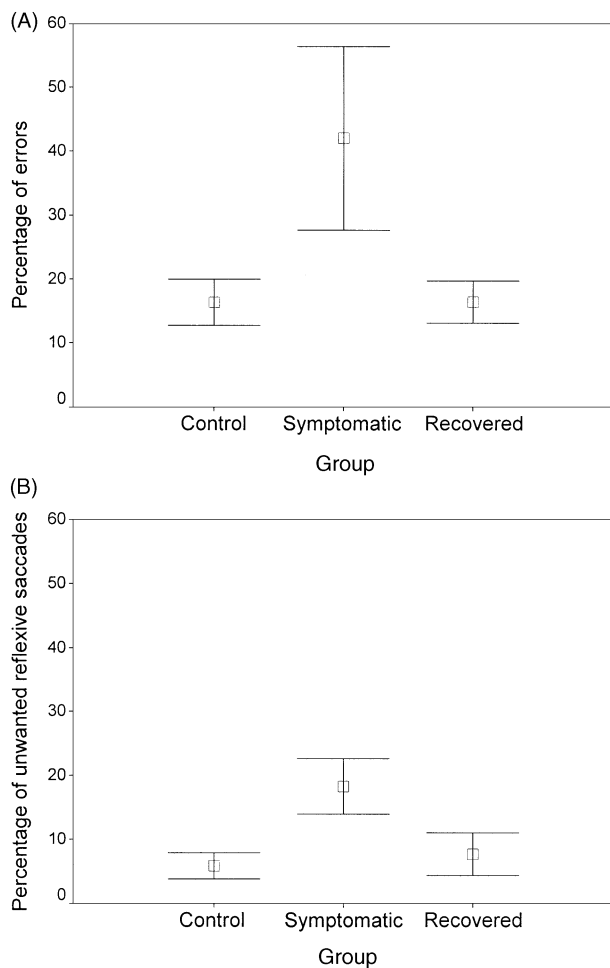
No significant left–right side differences were found after analysis of all saccade tasks. Therefore, data from each subject for leftward and rightward saccades were pooled for further analysis. Saccade latency and the median PEA of the reflexive and intentional saccade tasks are presented in Table 2.

In the gap and overlap tasks, no significant differences were found among the three groups with regard to latencies and PEA. Significant differences among the three groups were found for latencies of correct antisaccades, of memory-guided saccades and for the PEA of memory-guided saccades. Regarding the latencies of erroneous antisaccades, which are reflexive, no significant differences were found among the three groups. Compared with the control group, the symptomatic group showed significantly increased latencies for correct antisaccades ( $P < 0.05$ ) and memory-guided saccades ( $P < 0.01$ ). In contrast, no significant differences were found between the latencies of the recovered and control groups. A significant difference in PEA was found between the symptomatic and control groups ( $P < 0.05$ ), whereas the recovered group did not differ significantly from the control group. Inhibitory function in the intentional saccade tasks is shown in Fig. 2.

The percentage of errors in the antisaccade task ( $P < 0.01$ ) and the percentage of unwanted reflexive saccades in the memory-guided saccade task ( $P < 0.001$ ) showed significant differences among the three groups. The

**Table 2** Mean latency and accuracy (median PEA) of reflexive and intentional saccades

	Control group ( $\pm$ SD or range)	Symptomatic group ( $\pm$ SD or range)	Recovered group ( $\pm$ SD or range)	Statistics (Kruskal–Wallis test)
<b>Reflexive saccades</b>				
<b>Gap task</b>				
Mean latency (ms)	170 ( $\pm$ 22)	188 ( $\pm$ 39)	170 ( $\pm$ 37)	n.s.
Median PEA (%)	4 (0–9)	5 (0–15)	3 (0–13)	n.s.
<b>Overlap task</b>				
Mean latency (ms)	302 ( $\pm$ 42)	318 ( $\pm$ 43)	301 ( $\pm$ 45)	n.s.
Median PEA (%)	2 (0–8)	3 (3–19)	4 (0–5)	n.s.
<b>Intentional saccades</b>				
<b>Antisaccade task</b>				
Mean latency of correct antisaccades (ms)	274 ( $\pm$ 58)	355 ( $\pm$ 109)	262 ( $\pm$ 24)	$P < 0.05$
Mean latency of error antisaccades (ms)	171 ( $\pm$ 58)	201 ( $\pm$ 78)	137 ( $\pm$ 28)	n.s.
<b>Memory-guided saccade task</b>				
Mean latency (ms)	382 ( $\pm$ 100)	477 ( $\pm$ 145)	372 ( $\pm$ 64)	$P < 0.01$
Median PEA (%)	20 (6–39)	29 (10–69)	20 (8–37)	$P < 0.05$



**Fig. 2** Impaired inhibitory function in intentional saccade tasks. Mean percentage of error in the antisaccade task (A) and mean percentage of unwanted reflexive saccades in the memory-guided saccade task (B), with 95% confidence intervals. Note that the confidence interval for the symptomatic group does not overlap with the corresponding intervals for the other two groups.

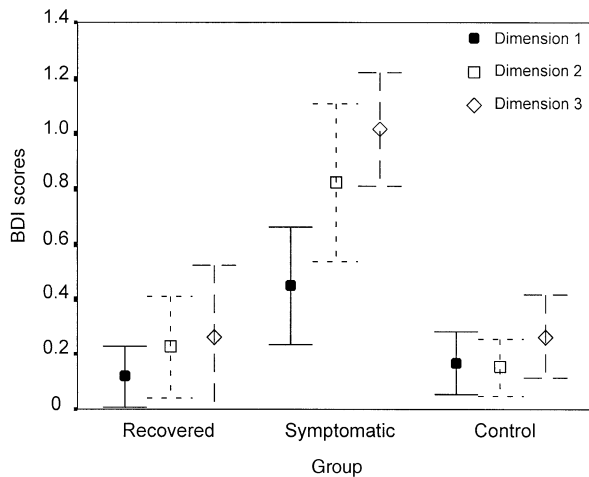
symptomatic group showed a significantly increased percentage of errors in the antisaccade task when compared with the control group ( $P < 0.01$ ). In the memory-guided saccade task, the symptomatic group had a significantly increased percentage of unwanted reflexive saccades when compared with the control group ( $P < 0.001$ ). For the same task, no significant difference was found between the recovered and the control groups.

Only one patient in the symptomatic group was taking a sedative with a short half-life (oxazepam); this was discontinued 24 h before the examination. In this patient, separate data analysis showed that the results of the reflexive and intentional saccades were within the 95% confidence interval of the symptomatic group for all parameters investigated (data not shown).

### Analysis of emotional assessment

None of the subjects of the whiplash groups fulfilled DSM-IV criteria for mood disorder (e.g. major depression or dysthymic disorder). The mean overall BDI scores were 13.8 (range 5–24) in the symptomatic group, 3.7 (range 0–16) in the recovered group and 4.0 (range 0–11) in the control group, on which basis a significant difference between the groups was found ( $P < 0.001$ ). The group results for the three dimensions (i.e. negative attitude/suicide, performance difficulty and physiological manifestations) and 95% confidence intervals are shown in Fig. 3.

The 95% confidence intervals of dimensions 2 and 3 of the symptomatic group did not overlap with the same dimensions of the recovered and control groups. In contrast, the 95% confidence interval of dimension 1 of the symptomatic group did overlap with the intervals of the recovered and control groups. Statistical analysis of the transformed data demonstrated a significant difference between the three BDI dimensions within the symptomatic group ( $P < 0.01$ ). However, a significant difference between



**Fig. 3** Comparison of the three BDI dimensions among the three groups. Dimension 1 = negative attitude and suicide; dimension 2 = performance difficulty; dimension 3 = physiological manifestation. The graph shows mean scores and 95% confidence intervals for the three BDI dimensions.

the three BDI dimensions was not found in the recovered or control groups. The Wilcoxon signed rank test showed significant paired differences between dimensions 2 and 1 ( $P < 0.01$ ) and dimensions 3 and 1 ( $P < 0.01$ ), but there was no significant difference between dimensions 2 and 3.

## Discussion

The results of the present study suggest that subjects with persistent complaints after whiplash injury may have a particular pattern of eye movement disturbance. This disturbance pattern is characterized by a dissociation in performance of reflexive and intentional saccade paradigms and is significantly different from the performance of the recovered group and the control group. There were no significant differences between the last two groups with respect to any of the eye movement assessment paradigms used. The performance of the symptomatic group was normal with respect to reflexive saccade tasks and there were no significant differences among the three groups with respect to the mean latency of erroneous antisaccades, which are also reflexive saccades. However, the symptomatic group showed significant differences in the performance of intentional saccades (antisaccades and memory-guided saccades), with impaired inhibitory function as indicated by increased errors in the antisaccades and an increased percentage of unwanted reflexive saccades towards the flashed target in the memory-guided saccade task. Moreover, in this group the latencies of intentional saccades (i.e. latencies of correct antisaccades and memory-guided saccades) and the PEA of memory-guided saccades were increased.

The pattern of eye movement disturbances observed in the symptomatic group is suggestive of frontal rather than parietal dysfunction. Although they are not directly comparable with the patients taking part in the present study, patients with

unilateral lesions of the parietal cortex have been found to show increased latency in reflexive visually guided saccade tasks (Pierrot-Deseilligny *et al.*, 1987, 1991, 1995). In contrast, subjects with frontal lesions show normal or slightly increased latencies in reflexive visually guided tasks (Pierrot-Deseilligny *et al.*, 1991) but increased latencies in intentional saccade tasks (Guitton *et al.*, 1985; Pierrot-Deseilligny *et al.*, 1991; Rivaud *et al.*, 1994). Frontal dysfunction, especially when prefrontal structures are involved, results in impaired inhibition of intentional saccades (Pierrot-Deseilligny *et al.*, 1991). Parietal dysfunction, however, does not result in an increased percentage of errors in the antisaccade task (Pierrot-Deseilligny *et al.*, 1991).

On the basis of these data, some additional factors that may have influenced our findings will be discussed. (i) Based on research with patients who had suffered mild traumatic brain injury (i.e. loss of consciousness) (Schoenhuber and Gentilini, 1988), depression has been discussed as a possible basis of the long-term symptoms of whiplash patients (Alexander, 1998). Traumatic brain injury was excluded from this study, and a strict definition of whiplash was applied. In addition, retrospective (Radanov *et al.*, 1989) and prospective (Radanov *et al.*, 1996) research with whiplash patients, selected according to a strict definition of injury, failed to confirm the occurrence of mood disorders (i.e. depression or dysthymia). Furthermore, depression was an exclusion criterion in this study. Nevertheless, the mean overall BDI score of the symptomatic group was significantly higher than the scores of the recovered and control groups. However, it has been shown previously (Williams and Richardson, 1993; Chibnall and Tait, 1994; Novy *et al.*, 1995) that the overall BDI score may falsely indicate the occurrence of depression in patients with somatic complaints. This tendency results from the higher scoring of BDI items with somatic content (e.g. fatigue), which are not specific to depression. Furthermore, patients with somatic complaints often have difficulty in performance (e.g. inability to do physical work) and may consequently have higher scores on the respective items. Detailed analysis of the three BDI dimensions in the present study showed that the elevated overall BDI scores in the symptomatic group came from the dimensions of physiological manifestations and performance difficulty. Furthermore, eye movement studies with depressed patients have shown a pattern of saccadic disturbances (Fukushima *et al.*, 1990; Sweeney *et al.*, 1998) that is different from the results found here. Fukushima and colleagues found no increase in the percentage of errors in the antisaccade task, and latencies of intentional or reflexive saccades were not increased. In the study by Sweeney and colleagues, depressed patients showed more errors in the antisaccade task and had less accuracy in the memory-guided saccade task. However, these patients had normal latencies in intentional saccades and showed cerebellar signs such as dysmetric visually guided saccades and an increased rate of saccadic intrusions (Sweeney *et al.*, 1998). On these grounds, depression is a highly unlikely explanation for the oculographic findings in

this study. (ii) Because previous research has shown a rather diffusely impaired psychological profile in whiplash patients (Radanov *et al.*, 1989, 1996; Wallis *et al.*, 1997), the related symptoms may theoretically constitute a basis for the oculographic findings in these patients. However, a recent study with patients complaining of dizziness found evidence of minor psychological problems but could not confirm any specific oculographic disturbance pattern (Yardley *et al.*, 1998). (iii) The tendency of whiplash patients to malingering, particularly in the context of litigation, has been discussed frequently (Schmand *et al.*, 1998). However, the Swiss accident insurance scheme does not provide compensation for non-economic loss such as pain or suffering, and it is considered that the scheme does not primarily promote malingering (Radanov *et al.*, 1995). In addition, the particular pattern of eye movement disturbance in the symptomatic group of the present study cannot be explained by malingering, since faking performance in the respective paradigms would require considerable knowledge of how to perform and the ability to do so. (iv) An influence of medication on oculographic findings is possible. For this reason, patients in the present study stopped medication 24 h before the examination of eye movements. Therefore, the pattern of saccadic eye movement disturbance found here cannot be explained by medication. (v) An influence of pain on oculographic findings (e.g. by distracting the subject) may be assumed, but a way of interpreting the disturbance pattern found in the present study on this basis appears rather unlikely.

In conclusion, dissociation of the performance of reflexive and intentional saccades, with normal performance of reflexive saccades and impaired performance of intentional saccades (i.e. impaired inhibitory function, increased latency and increased PEA), was found in the symptomatic group. This pattern may be explained by a dysfunction of frontal, especially prefrontal, cortical structures, which remains open to further examination and aetiological consideration.

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