# Oculomotor problems after cervical spine injury

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Oculomotor function was investigated in 39 patients with a previous soft-tissue injury of the cervical spine. The velocity, the accuracy, and the pattern of the eye movements were disturbed in 20 patients with chronic and disabling symptoms. Oculomotor function in the 19 asymptomatic patients did not differ from a control group.

The oculomotor function seems to be impaired, possibly by brain stem lesions, in patients with chronic symptoms of whiplash injury of the cervical spine.

The whiplash syndrome associated with a acceleration/deceleration in motor car accidents includes headache, neck pain, vertigo, blurring of vision, dysphagia, dysacusis, and fullness of the ear (Macnab 1971, Norris and Watt 1983, Hinoki 1984). Lesions affecting structures in the brain stem and the cerebellum cause eye motor dysfunction (Baloh et al. 1979, Wennmo and Hindfelt 1980, Henriksson et al. 1981, Wennmo et al. 1983).

We have studied the eye motility in patients with disabling symptoms of soft-tissue injury of the cervical spine.

## Patients and methods

Thirty-nine patients with a history of a soft-tissue injury of the cervical spine due to car accident were investigated 6 months or more after the trauma (Table 1). All were noncontact injuries, and the patients with a head injury were excluded. Twenty patients, 7 males and 13 females median age 36 (21–54) years, with chronic and disabling symptoms of more than 1 year, and 19 asymptomatic patients, 10 males and 9 females median age 25 (19–67) years, were evaluated.

The *chronic* cases were 20 consecutive patients admitted to our department because of the severity of symptoms. All had neck ache, neck stiffness, and headache; and all but 1 also had shoulder and arm pain; finally, all but 2 had vertigo or dizziness. Visual symptoms, often blurring of vision, and auditory symptoms, such as tinnitus, dysacusis, and fullness of the ear, were noted in 15 patients. The physical examination revealed no neurologic dysfunction, but all the patients had tenderness over the cervical and shoulder muscles, as well as a reduced range of cervical movement. Radiographs of the cervical spine, including flexion-extension views and myelography, showed no signs of instability; 7 patients had mild degenerative spondylosis.

The *asymptomatic* group consisted of patients exposed to a soft-tissue injury of the cervical spine but without complaints 6 months or more after the trauma.

The *control* group consisted of 25 healthy individuals, students and collaborators, median age 34 (25–40) years, without a history of a soft-tissue injury of the neck or a head injury.

Horizontal eye movements were recorded by bitemporally placed surface electrodes and recorded on a DC-coupled Graz printer. The stimulus consisted of a

Table 1. Type of motor car accident

| | Patients | |
|---|---|---|
| | Chronic | Asymptomatic |
| Front-end | 3 | 4 |
| Rear-end | 8 | 10 |
| Side | 2 | 1 |
| Single and roll over | 7 | 4 |

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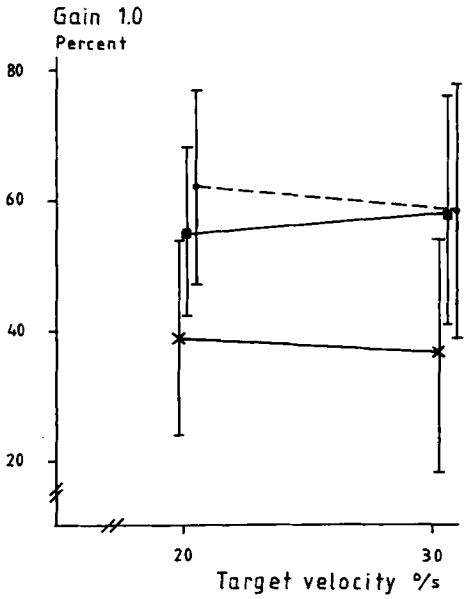


Figure 1. The mean velocity gain, i.e., the ratio between the velocity of the eyes and the velocity of the moving light spot given as group means and standard deviations for velocities 20°/s and 30°/s of the light spot. The percentage of the smooth pursuit with gain = 1.0 is expressed in the figure ( x chronic, ■ controls, --- asymptomatic).

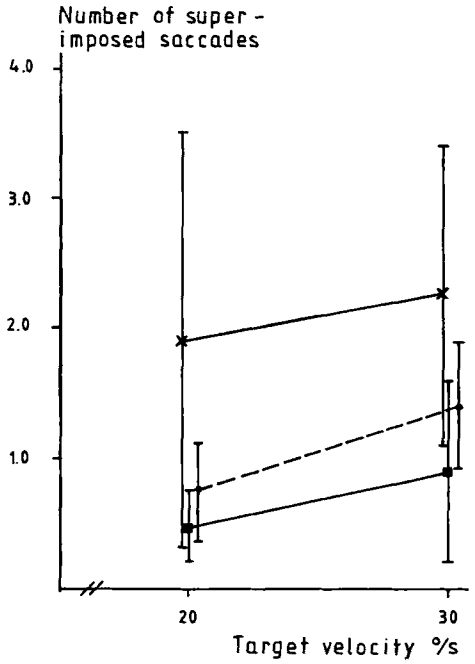


Figure 2. The number of superimposed saccades given as group means and standard deviations for velocities 20°/s and 30°/s of the light spot ( x chronic, ■ controls, --- asymptomatic).

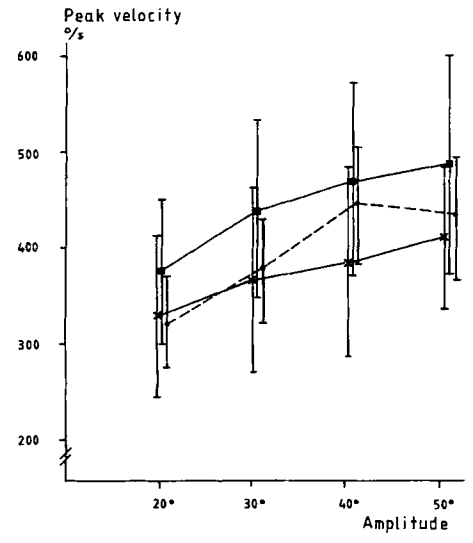


Figure 3. The peak velocity given as group means and standard deviations for angle amplitudes 20°, 30°, 40,°, 50°( x chronic, ■ controls, --- asymptomatic).

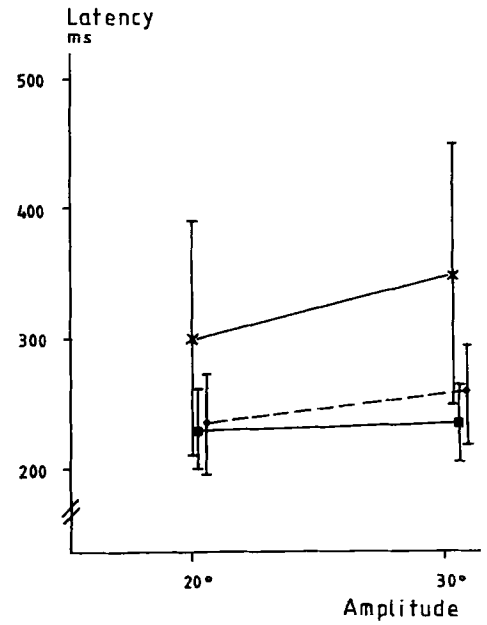


Figure 4. The latencies given as group means and standard deviations for 20° and 30° amplitude ( x chronic, ■ controls, --- asymptomatic).

curve-shaped screen with light-emitting diodes. The diodes and the analysis of the recordings were applied by a computerized automatic procedure (Bergenius 1984). The smooth pursuit test consisted of tracking a pendular light diode moving from a point 30° of midline to a point located 30° to the other side of the midline. The light diode was projected on the curved screen at a constant speed of 20° and 30° per second clockwise and counterclockwise (Bergenius 1984). Each tracking was followed by a refixation saccade back to the starting point. Five smooth pursuits with refixation saccades at each velocity and direction were stored in a computer, and the analysis was presented on a screen and printed. Data obtained by this procedure were the gain (eye velocity/target velocity) of the smooth pursuit, the number of superimposed saccades, and the amplitude of the refixation saccades. The voluntary saccade test was performed by fixation of a light diode on the screen and lighted at angles 20° and 30° on both sides of the midline. Ten saccades of each type were recorded and analyzed by a computer. The accuracy of the saccades as a percentage of the total amplitude, the latency (i.e., the reaction time in milliseconds elapsing from the change of the light spot to the start of the eye movement), and the peak velocity of the saccade were estimated. The test results from each patient were compared with those of the control group. The group mean ± 2 SD was considered to be the normal range. Statistical analysis was performed with the Mann-Whitney *U*-test.

## Results

### *Smooth pursuit eye movements*

There was no difference between the control group and the asymptomatic patients (Figure 1). The patients with chronic symptoms had reduced velocity gain ($P < 0.01$) compared with the asymptomatic patients.The smooth pursuit eye movements were abnormal, with reduced velocity gain in 14 of the patients with chronic symptoms, and in 12 of these the velocity gain was asymmetrically reduced.

The number of superimposed saccades was increased ($P < 0.001$) in the chronic group, but did not differ from normal in the asymptomatic patients (Figure 2). The smooth pursuit showed superimposed saccades in 16 of the chronic patients, and in 13 of these it was unilateral.

The amplitude of the refixation saccades was decreased ($P < 0.01$) in the patients with chronic symptoms, and this was especially pronounced in target velocity 30°/s. The refixation saccade showed a two-step appearance in 11 of the chronic patients—in both directions in 5 and in one direction in 6.

### *Saccades*

In 9 patients with chronic symptoms, the accuracy of voluntary saccades was affected towards hypometric, but the whole chronic group did not differ from normal. Six patients with chronic symptoms had lower accuracy and maximal velocity of the saccades, and 5 of these patients also showed latency prolongation.

The peak velocity was reduced in the chronic group ($P < 0.05$; Figure 3). The patients with chronic symptoms had prolonged latency ($P < 0.001$; Figure 4).

Eighteen of the twenty patients with chronic and disabling symptoms had dysfunctions of more than ± 2 SD of either one or both of the eye motor tests used,the smooth pursuit and/or the saccade test. Only two had normal oculomotor test functions. Four patients showed pronounced abnormalities of the smooth pursuit in combination with saccadic dysfunction. None of the patients with saccadic dysfunction had normal pursuit.

## Discussion

It was not possible to evaluate the force of impact in each case of whiplash injury (Norris and Watt 1983), but we have no information that the asymptomatic patients would have experienced a less severe type of injury. The clinical examination revealed no abnormalities despite the severe symptoms, which is a regular finding in these patients (Hohl 1974, Balla 1980).

Oculomotor dysfunction was present in 18 of 20 patients with persisting symptoms after a soft-tissue injury of the cervical spine. Four patients had pronounced abnormalities, and 14 had moderate oculomotor dysfunction.

In the 14 patients with moderate oculomotor dysfunction, i.e., the smooth pursuit abnormalities, the disturbances may be explained by affection of the proprioceptive system in the cervicocranial area (Hinoki 1984, Rosenhall et al. 1987). The smooth pursuit and the saccade are eye motility functions with important relay stations in the brain stem and cerebellum. Lesions affecting structures in the brain stem and the cerebellum often cause eye motor dysfunction, i.e., reduction of saccadic velocity and smooth pursuit velocity gain (Baloh and Honrubia 1979, Wennmo and Hindfelt 1980, Henriksson et al. 1981, Wennmo et al. 1983).

Four of our patients had pronounced abnormalities of the smooth pursuit in combination with saccadic dysfunction consistent with lesions in the brain stem. None of the patients with saccadic dysfunction in our series had normal pursuit test, thus excluding frontal lobe lesions.

Recently, pathologic oculomotor dysfunction was reported in patients with chronic primary fibromyalgia with dysesthesia (Rosenhall et al. 1987). Most of their patients had moderate oculomotor disturbances, which were probably due to a proprioceptive dysfunction of the neck. However, they also reported supratentorial affection in several cases.

Although our patients had a traumatic etiology, it is interesting to note that they often showed moderate oculomotor disturbances, probably secondary to impaired proprioception of the cervical spine (Rosenhall et al. 1987). Four of them had oculomotor test results similar to the findings in patients with brain stem lesions. It seems that patients with chronic symptoms after soft-tissue injury have a localized lesion of the brain stem or an afferent proprioceptive dysfunction of the cervical spine.

Our results show different degrees of oculomotor dysfunction in patients with chronic symptoms after soft-tissue injury of the cervical spine. Possibly the pronounced oculomotor dysfunction in some whiplash cases can be caused by medullary lesions.

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