Eye motility dysfunction after soft-tissue injury of the cervical spine

A controlled, prospective study of 38 patients

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We investigated eye motility prospectively in 40 patients with a soft-tissue injury of the cervical spine. The initial oculomotor test, performed within 3 months, was pathologic in 8 patients. The follow-up test in 38 patients, on average 15 months after the accident, remained pathologic in the 8 patients and 5 additional patients had changed from normal to pathologic test results.

At follow-up, all the 13 patients with oculomotor dysfunction had persisting symptoms, while 5 of the 25 cases with normal test results still were symptomatic.

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Submitted 92-10-03. Accepted 92-11-15

The most frequent complaints after soft-tissue injury of the cervical spine are headache, neck pain, vertigo, blurring of vision, dysphagia, dysacusis and fullness of the ear (Macnab 1971, Norris and Watt 1983, Hinoki 1984). The physical examination of these patients is often normal, despite the severe symptoms (Hohl 1974). The question of involvement of structures more complex than the musculoligamentous and intervertebral disc tissues (Macnab 1971), i.e., subtle injuries of the cervical nerve roots (Tamura 1989) and medulla or even intracranial structures, has been raised (Ommaya 1984).

Recently, we reported oculomotor dysfunction in patients with chronic and disabling symptoms after soft-tissue injury of the cervical spine (Hildingsson et al. 1989). We have now prospectively studied oculomotor function in relation to clinical symptoms in patients with acute soft-tissue injury of the cervical spine.

Patients and methods

A prospective study was carried out in 40 consecutive patients, 16 men and 24 women with a soft-tissue injury of the cervical spine. The median age was 30 (17–56) years. All the patients had noncontact injuries of the cervical spine due to car accidents. Patients with a previous history of neck trauma or a neck-shoulder problem were not included in the study. The impact direction was in 19 cases rear-end, in 11 cases front-end, in 6 cases single and roll-overs, and in 4 cases side collisions. All the patients were initially treated with a cervical collar, and physiotherapy was given when necessary. The first oculomotor test was performed on average 1.7 (0.5–3) months after the accident. 38 of the 40 patients returned for follow-up and a second oculomotor test on average 15 (8–28) months after the accident.

The control group consisted of 25 healthy individuals, students and co-workers, median age 34 (25–40) years, without a history of soft-tissue injury of the neck or head injury. The oculomotor tests were performed by the method described by Hildingsson et al. (1989). The control group's mean ± 2 SD was considered to be the normal value.

Results

Initial examination

The most frequent acute symptoms after injury were aching and stiffness in the neck and headache, followed by dizziness and shoulder pain. The initial symptoms in the patients with a pathologic oculomotor test did not differ from those with a normal test. 8 of the 40 patients had pathologic oculomotor values. The physical examination of these patients did not reveal any abnormalities not seen in the cases with a normal test. Abnormal neurologic signs were found in 2 patients, 1 with sensory impairment in digits 4 and 5 and 1 with reduced motor power in one arm and the musculus pectoralis. These 2 patients had normal oculomotor values.

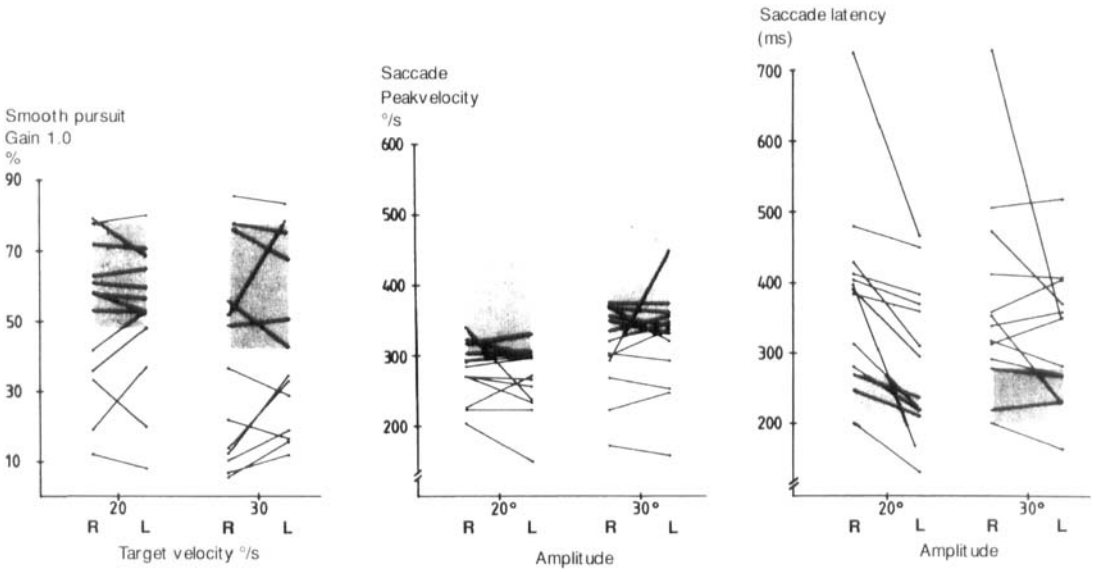

Figure 1. The velocity gain, i.e., the ratio between the eye velocity and the target velocity, the peak velocity and saccade latencies for saccade amplitudes 20° and 30° for the 13 cases with pathologic test results. The shaded area indicates the control group's mean ± 2 SD (R right, L left side).

Follow-up

25 of the 38 patients had normal test values; 20 of these had recovered with no or only minor discomfort, not interfering with daily activities or work. The remaining 5 patients with normal test results had persisting symptoms interfering with their working ability.

The 8 patients with initial oculomotor dysfunction remained pathologic. An additional 5 patients had changed from almost normal to pathologic test results. There was no intraindividual variability between the initial and the second test in any of the remaining cases. All the 13 patients with oculomotor dysfunction had disabling symptoms. In this abnormal group, 2 patients showed a slight improvement while 5 had more pronounced pathology. The others remained unchanged.

In 6 patients the smooth pursuit eye movements were abnormal with reduced velocity gain and with an abnormal number of superimposed saccades compared with the normal values. In the whole pathologic group, the saccades showed low peak velocity and prolonged latencies (Figure 1).

3 patients showed pronounced abnormalities affecting both smooth pursuits and saccades, while 4 patients had abnormalities of the same type, but to a lesser degree. 1 case showed mainly an increased number of superimposed saccades while 5 had mainly saccade latency prolongations compared with the control group (Table 1). The impact direction in patients with normal and pathologic tests was the same with 50 percent rear-end collisions.

Discussion

An accurate estimation of impact is difficult (Norris and Watt 1983) and thus it was not possible to differentiate the patients in our series as to the energy of impact. The 45 percent residual symptoms in our series confirms previous reports (Macnab 1971, Hohl 1974).

Lesions at different locations in the central nervous system may cause oculomotor dysfunction. The maximum saccadic velocity is reduced and the smooth pursuit velocity gain decreased in brain stem disorders (Henriksson et al. 1981, Schalén et al. 1982, Wennmo et al. 1983). Cerebellar disorders cause reduced smooth pursuit velocity gain (Schalén et al. 1982, Wennmo et al. 1983). A combination of brain stem and cerebellar symptoms, due to posterior fossa lesions, result in eye motility dysfunction (Wennmo et al. 1980).

Recently, we reported on oculomotor dysfunction in 18 of 20 cases with chronic symptoms after soft-tissue injury of the cervical spine (Hildingsson et al. 1989). 13 of the 18 cases with persisting symptoms in the

Table 1. Specification of the oculomotor test results

			Smooth pursuit				Saccade			
Case	Sex	Age	G1	G2	S1	S2	P1	P2	L1	L2
1	M	56	– – –	– – –	+++	+++	– –	–(–)	+++	+++
2	F	23	– –	–(–)	+	+	–	–	++u	++
3	F	32	– –	– – –	+++	++	–u	–u	++	++u
4	F	31	N	–	++	+	–	– –	N	+++
5	F	44	N	– –	+++	+++	–	–	N	+
6	F	17	–	N	+	++	–	–	+	+++
7	F	27	N	N	++	++u	N	N	++	+++
8	F	22	–	N	+++	+++	– –	–	N	N
9	F	26	– – –u	– – –	+++	+++	–	– –	N	+++
10	M	34	N	N	+	N	N	–(–)	+++	+++
11	F	25	N	N	N	N	N	–u	N	+++
12	F	26	N	N	(++)	(+)	N	–	++u	++u
13	F	41	N	(–)	(++)	N	–	(– –)	+++	+++

G1 Gain at initial test, G2 Gain at follow-up test
S1 Superimposed saccades at initial test, S2 Superimposed saccades at follow-up test
P1 Peak-velocity at initial test, P2 Peak-velocity at follow-up test
L1 Latency at initial test, L2 Latency at follow-up test
N Normal test result, () Part of test, u Unilateral
– decreased ≥ 1 SD, – – decreased ≥ 2 SD, – – – decreased ≥ 3 SD
+ increased ≥ 1 SD, ++ increased ≥ 2 SD, +++ increased ≥ 3 SD

present study had pathologic oculomotor test results. 3 cases had severe abnormalities comparable with the most pronounced cases in the previous report. An explanation of these pronounced abnormalities besides pathologic proprioceptive signals could be brain stem dysfunction.

Animal experiments have shown that noncontact injury of the cervical spine might result in pathologic alterations in the central nervous system, especially in the brain stem region (Domer et el. 1979, Unterharnscheidt 1982, Boismare et al. 1985). However, the anatomic and physiologic bases as well as the force of trauma are different from the conditions in humans, and any attempt to explain our clinical findings as a result of brain stem or medullary lesions is thus a hypothesis.

The neck-muscle proprioceptive system can influence the oculomotor and vestibular system (Hikosaka and Maeda 1973, Barlow and Freedman 1980, Kobayashi et al. 1986). Recently, oculomotor disturbance was reported in patients with chronic primary fibromyalgia with dysesthesia (Rosenhall et al. 1987) and tension headache syndrome (Carlsson and Rosenhall 1988). The oculomotor abnormalities, classified as moderate and seen in most of our cases, were more severe than the disturbances in the patients with tension headache. They proposed that the abnormalities in their cases were a result of pathologic proprioception from the neck. It is reasonable to believe that the soft-tissue injury of the cervical spine in our patients also might result in a dysfunction of the proprioceptive system in the cervicocranial area.

Acknowledgements

Financial support was provided by the Trygg-Hansa Insurance Company, the National Societies for Aiding Traffic Accident Victims and Polio Victims, and the University of Umeå Medical Faculty.

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