

## PROLONGED FUNCTIONAL IMPAIRMENTS AFTER WHIPLASH INJURY

Ylva Hagström, RTP<sup>1</sup> and Jane Carlsson, RTP PhD<sup>2</sup>

From the Departments of <sup>1</sup>Physiotherapy and <sup>2</sup>Clinical Neuroscience, Neurology Section, Sahlgrenska Hospital, Göteborg, Sweden

**ABSTRACT.** Thirty patients with whiplash injuries were examined 1 to 55 months after the accidents. Pain had appeared on the day of the accident in 24 (80%) of the patients and with different delays in the remainder. The mean pain intensity was 43 mm (SD 26) on a visual analogue scale (VAS). All patients had pain in the neck, 17-33% had headache and 6-17% had pain in various regions of the arms. Thirteen patients (43%) suffered from constant pain, while 17 (57%) had pain-free periods. Muscle tenderness was higher at all tested sites compared with controls. The tolerance level to pressure pain in the index finger as well as grip strength and neck mobility was reduced compared with controls. The whiplash patients showed poorer mental well-being compared with a reference group representing the general population and compared with a group of tension-type headache patients.

*Key words:* cervical mobility, grip strength, mental well-being, muscle tenderness, pain, pain tolerance, whiplash injury.

### INTRODUCTION

The term whiplash injury was introduced by Crowe in 1928 (10) to denote soft tissue neck injuries to the occupants of motor vehicles that had sustained rear-end impacts. The cervical spine can be exposed to similar strain from other types of accidents and the diagnosis in clinical practice is not restricted to pure extension-flexion injuries (6, 21). Typically the patient has neck pain and neck stiffness but there are many associated features. The pain can radiate into the shoulders and arms and be felt down in the thoracic spine or as headache. Other symptoms are paraesthesiae and/or weakness in the arms, dysphagia, visual and auditory disturbances, tinnitus and vertigo (6, 21, 40).

Clinical examination may reveal restricted cervical mobility and tense and tender cervical musculature. Some patients present neurological deficits of the

upper limbs (21, 39). The pathological basis of the clinical manifestations is not clear. Many structures of the neck, including muscles, ligaments, discs and joints, can be traumatized (12, 9, 21). A comprehensive review of the clinical features, pathophysiology and treatment of whiplash injury is presented by Barnsley et al (2). Current studies have introduced the term "common whiplash", which excludes fracture and/or dislocations of the cervical spine as well as head trauma or alteration of consciousness (39).

There is so far no consensus on management either in the acute or in the chronic phase (6). Most patients recover within a couple of months but a considerable percentage of patients show protracted disability. Some authors attribute the persistent symptoms to somatic factors (21, 29), whereas others attribute them to underlying psychological factors or the possibility of financial gain (1, 22, 28). In two recent studies, Radanov et al (31, 32) have analysed the importance of somatic and psychological factors influencing the course of recovery. They reported that somatic symptoms suggesting a more severe injury appear to be particularly related to delayed recovery, while psychosocial factors did not prove predictive. Improvement in well-being was mainly associated with recovery from somatic symptoms.

The controversy thus persists and the debate continues. More information is required to clarify the whiplash syndrome. The aim of the present study was to describe and evaluate symptoms and signs in patients with prolonged disability after a whiplash injury.

### METHODS

#### *Subjects*

Thirty patients (22 women, 8 men) diagnosed as whiplash injuries at the Neurological Department, Sahlgrenska Hospital in Göteborg, Sweden, took part in the study. Their mean age was 35 years (women 37 years, men 30 years; range 20-63 years). Further demographic data are given in Table I.

Twenty-six patients had sustained car accidents (73% rear, 23% frontal, 4% side collisions) and 4 patients were

Table I. Main social and demographic characteristics of the patient group

Characteristics	Total (n = 30)	Women (n = 22)	Men (n = 8)
Age (years) at examination	Mean value (SD)		
Months after accident at examination	35 (11)	37 (12)	30 (6)
	13 (12)	10 (8)	21 (18)
Education	Frequency number (%)		
Compulsory level	21 (70)		
Higher levels		14 (64)	6 (75)
Source of financial support	19 (30)	8 (36)	2 (25)
Gainful occupation	7 (23)	5 (23)	2 (25)
Sickness benefit	20 (67)	14 (64)	6 (75)
Disability pension	1 (3)	1 (4)	
Unemployed	2 (7)	2 (9)	

exposed to other types of acceleration/deceleration injuries 1 to 55 months before they were referred to the Department of Physiotherapy for treatment and entered the study. One patient had sustained a previous whiplash injury but had no sequelae at the time of the current accident. Three patients suffered from recurrent headache before the accident.

Radiological examination of the cervical spine had revealed no fractures or dislocations. MRT was performed on 24 of the 30 patients. MRT was considered quite normal in 8 patients. Sixteen patients presented degenerative changes of varying degree. Five of these had bulging discs, which in one patient gave a discreet deformation of the cord. Indications of root compression by osteophytes and/or bulging discs were found in 4 patients. No neurophysiological investigations were performed. Twenty-three (80%) patients were involved in litigation at the time of the study.

A control group comprised of 30 healthy individuals (20 women, 10 men) with a mean age of 32 years (range 19–63 years) was used for comparisons of muscle tenderness, pain tolerance level, cervical mobility and grip strength. The controls were recruited from hospital staff and from the authors' relatives and acquaintances. Not all patients performed all measurements. The controls were chosen to match the patients with respect to age and sex as exactly as possible in every measurement. A reference group representing the population of Göteborg (30) served as age- and sex-matched controls in the estimation of mental well-being.

The study was approved by the Ethics Committee at Sahlgrenska University Hospital in Göteborg.

#### Assessment instruments

The examination included: pain intensity, pain characteristics, pain drawing, measurement of muscular tenderness, neck mobility and grip strength and assessment of mental well-being. The pain drawing and assessment of mental well-being were introduced after some time and therefore performed by 18 and 17 patients respectively.

The patients were asked to stop their intake of analgesics and sedatives the day before examination. Twenty-one patients used analgesics in moderate doses and usually irregularly; 10 used salicylates, 8 paracetamol and 3 NSAIDs. Three patients used codeine and 2 patients dextropropoxyphene in combination with paracetamol. Three patients used tricyclic antidepressants.

**Pain intensity.** Pain intensity was assessed by means of a VAS (7). The patients were instructed to rate their pain at the time of examination.

**Pain characteristics.** Different symbols could be selected from a fixed set of symbols describing different pain types, such as aching, burning, throbbing or cutting.

**Pain drawing.** Instructions for the pain drawing were: draw your pain on the figure; include all areas where you feel pain or other sensations; use symbols to indicate the types of pain or other sensations. Eighteen patients performed pain drawings.

**Muscular tenderness.** Two methods were applied: manual palpation and pressure algometry. The measurements were performed at 14 points, which were selected because they corresponded to the location of pain and were easy to identify. The muscle sites chosen for palpation were as follows:

- The origin of the extensor carpi radialis longus muscles at the lateral epicondyle.
- The insertion of the deltoid muscles at the deltoid tuberosity of the humerus.
- The passage of the proximal tendon of the long head of the biceps brachii muscles in the intertubercular sulcus of the humerus.
- The insertion of the levator scapulae muscles at the superior angle of the scapulae.
- The insertion of the sternocleidomastoid muscles at the mastoid process.
- The middle part of the trapezius muscles.
- The origin of the trapezius muscles at the occipital protuberance.

The manual palpation and the algometer test were performed with the patients in relaxed lying and sitting positions. The second and third fingers were used and the palpation was done with small circulating, gently pressing movements. The same procedure was used in the 28 healthy controls. Manual palpation was assessed in accordance with a four-point scale (20). The following levels of pain intensity on palpation were used: 0 = no report of pain and no visible reactions; 1 = report of tenderness but no visible reaction; 2 = report of painful tenderness and visible reaction; 3 = report of severe pain and marked visible reaction, "jump sign" (42).

**Algometer.** The Pressure Pain Threshold (PPT) was

measured by means of a pressure algometer (Somedic AB, Farsta, Sweden), which consists of an acrylic handle with a pressure-sensitive strain gauge at the tip connected to an amplifier. The tip of the algometer was provided with a probe 0.5 cm<sup>2</sup> in area, covered with 1 mm thick soft polypropylene material. The pressure is given in KPa (18, 19). The PPT was measured over the same 14 points as were used for manual palpation. The investigator always started with manual palpation. The time interval between the two tests was 10 min. Before the measurements, the patients and the healthy controls were informed about the procedure and told that their pain threshold was to be tested. They were asked to press a button which they held in the hand opposite to the tested side as soon as the sensation changed from pressure to pain. It was stressed that the pain threshold and not the tolerance was to be tested. The tolerance level was measured on the distal phalanx of digit II before the PPT test. The difference between pain threshold and pain tolerance level was carefully explained to each participant.

**Cervical neck mobility.** The neck mobility was measured with an inclinometer (Myrin, LIC, Solna, Sweden) in patients and controls (23). All movements, including flexion, extension, side flexion and rotation, were measured with the participants seated. For each movement, the participants were instructed to move their heads actively as far as they could. Care was taken to make sure that a pure movement of the head took place and movements of the shoulders or the back were minimized. Each movement was repeated twice and the best of the two was recorded. Twenty-six patients performed the test.

**Grip strength.** Grip strength was measured by means of maximal manual compression of a rubber balloon (Vigrometer, Modema AB, Bromma, Sweden). The registered pressures, in Bar, were compared with those of healthy controls. During the test the participants were sitting in a chair with an elbow angle of 90° and with the wrist in a neutral functional position, i.e. with a slight ulnar and dorsal flexion. Three measurements were performed at an interval of 30 s and the mean value was registered (25).

**The Mood Adjective Check List (MACL)** offers a quantitative composite measure of mental well-being (36). The MACL consists of 71 adjectives describing mood and related feelings. The patients indicate their current emotional states by marking a series of 1-to-4 scales; higher scores indicate more positive emotional states. The adjectives are clustered in six bipolar dimensions: Pleasantness/Unpleasantness, Activation/Deactivation, Calmness/Tension, Extroversion/Introversion, Positive-/Negative Social Orientation, Confidence/Lack of Confidence. An overall mood index was calculated as well. Seventeen patients performed the MACL tests. A reference group representing the general population (30) and a group of patients with tension-type headache (8) were used for comparison of the MACL.

#### Statistical methods

For comparison between the two groups Wilcoxon's *p*-test was used. The tests were considered significant at the 5% level. Correlations between variables were calculated by means of Spearman's rank correlation test.

## RESULTS

**Pain appearance.** The neck pain had appeared

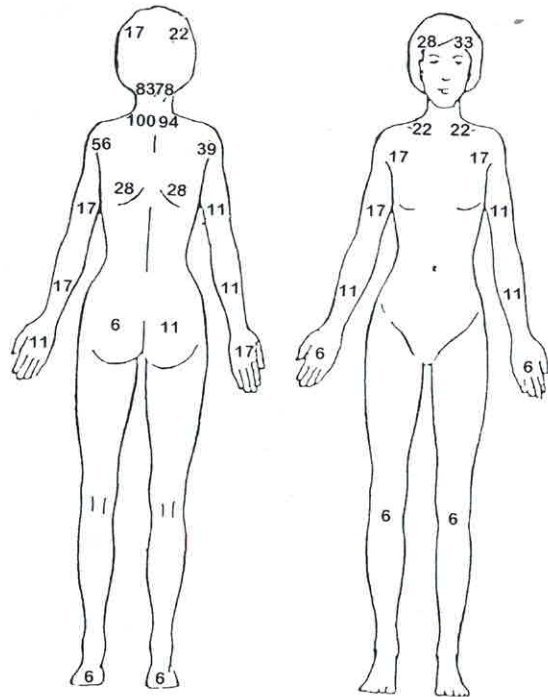


Fig. 1. Pain distribution in percentage of all patients who marked a painful area.

immediately or during the same day as the accident in 24 patients (80%). The other 6 patients reported a pain-free interval from the accident of varying length; in 4 patients the delay exceeded 3 months.

**Pain intensity.** At the time of examination the mean score of the patient group was 43 mm (SD 26), with a range of 0–85 mm.

**Pain characteristics.** Twenty-one patients (70%) described their pain as aching, 7 (23%) as burning and 2 (7%) as throbbing. Thirteen patients (43%) suffered from constant pain while 17 (57%) had pain-free periods.

**Pain location (drawing).** The location of pain is illustrated in Figure 1. All patients had pain over the cervical spine and 28% located their pain to the thoracic spine. Seventeen to 33% complained of headache, 17 to 56% had shoulder pain and 6 to 17% depicted their pain as involving various regions of the arms.

**Muscle tenderness.** The PPTs measured by algometry and tenderness scores measured manually are presented in Table II. It will be seen that PPT is significantly lower at all tested sites compared with

Table II. Mean value (SD) of pressure pain threshold (PPT) in kPa and muscle tenderness (score 0–3) in patients and controls

Muscles	PPT			Muscle tenderness		
	Patients (n = 30)	Controls (n = 28)	p	Patients (n = 30)	Controls (n = 28)	p
M. extensor carpi rad. longus	153 (53)	317 (92)	***	1.15 (0.78)	0.23 (0.37)	***
M. deltoideus	220 (116)	376 (129)	***	0.55 (0.65)	0.05 (0.16)	***
M. biceps brachi	152 (62)	288 (94)	***	1.25 (0.86)	0.20 (0.28)	***
M. levator scapulae	185 (74)	402 (124)	***	1.35 (0.85)	0.05 (0.21)	***
M. sternocleidomastoideus	113 (38)	226 (54)	***	0.78 (0.91)	0.07 (0.22)	***
M. trapezius	106 (44)	242 (79)	***	1.95 (0.79)	0.29 (0.40)	***
M. trapezius (occipital protub)	138(50)	316 (85)	***	1.73 (0.69)	0.25 (0.42)	***

\*\*\*  $p < 0.001$ .

controls. The algometer test and the manual test correlated on a highly significant level ( $r = -0.63$ ,  $p < 0.001$ ) (Table III). There was a significant correlation between the manual test and the pain intensity level measured by VAS ( $r = 0.37$ ,  $p < 0.05$ ) but there was no significant correlation between the algometer test and the pain intensity level ( $r = -0.31$ ).

**Pain tolerance level.** The mean value of the patients' tolerance to pain was 371 kPa (SD 135) and that of the controls 568 kPa (SD 184). The difference is highly significant ( $p < 0.001$ ).

**Neck mobility.** The mobility of all tested movements was significantly lower ( $p < 0.001$ ) in the patient group compared with the controls (Table IV).

**Handgrip strength.** The patient group presented a significantly lower ( $p < 0.001$ ) handgrip strength than did the controls. The mean value of the patients' handgrip strength was 0.76 bar (SD 0.28), compared with 0.99 bar (SD 0.20) for the controls.

Table III. Correlation between manual palpation and pressure algometry

Tenderness of	r	p
m. biceps	-0.55	**
m. deltoideus	-0.61	***
m. extensor carpi rad. longus	-0.38	*
m. levator scapulae	-0.24	n.s
m sternocleidomastoideus	-0.57	***
m. trapezius	-0.47	**
m. trapezius (occipital protub)	-0.67	***
average tenderness	-0.63	***

n.s. = not significant

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

\*\*\*  $p < 0.001$ .

**Mental well-being.** The patient group showed poor mental well-being compared with the reference group (Table V). This was so for the overall MACL ( $p < 0.001$ ) as well as five dimensions ( $p < 0.001$ ). One dimension, "Confidence/Lack of Confidence", did not differ. The patient group also presented a lower score on the overall MACL compared with patients with tension-type headache ( $p < 0.05$ ). There was, however, no difference with respect to the dimensions "Calmness/Tension", "Pos/Neg Social Orientation" and "Confidence/Lack of Confidence", while the other three dimensions showed significantly better mental well-being in the tension-type headache group.

## DISCUSSION

The patients in this study were referred to the neurological clinic because of complaints after whiplash injuries which had occurred 1 to 55 months previously. All patients suffered from neck pain. Compared with healthy controls, the patients were also more tender in the neck, shoulder and arm muscles

Table IV. Neck mobility in patients and controls

Neck mobility	Patients (n = 26)		Controls (n = 30)		p
	Mean	SD	Mean	SD	
Extension	49°	22°	79°	12°	***
Flexion	38°	17°	60°	9°	***
Lateral flexion	31°	13°	45°	6°	***
Rotation	51°	15°	79°	7°	***

\*\*\*  $p < 0.001$ .

Table V. The Mood Adjective Check List (MACL). Range of well-being (score 1–4) in the patient group, reference subjects and patients with tension-type headache

Variables	Reference subjects (n = 112)			Patients with whiplash injury (n = 17)			Patients with tension-type headache (n = 60)	
	Mean	(SD)	<i>p</i>	Mean	(SD)	<i>p</i>	Mean	(SD)
Overall MACL	3.30	(0.39)	***	2.51	(0.50)	*	2.76	(0.39)
Pleasantness/unpleasantness	3.38	(0.50)	***	2.36	(0.67)	*	2.76	(0.58)
Activation/deactivation	3.43	(0.45)	***	2.33	(0.64)	**	2.81	(0.53)
Calmness/tension	3.24	(0.64)	***	2.35	(0.52)	n.s.	2.26	(0.61)
Extroversion/introversion	3.05	(0.49)	***	2.50	(0.53)	*	2.83	(0.42)
Pos/neg social orientation	3.65	(0.34)	***	2.75	(0.65)	n.s.	3.12	(0.45)
Confidence/lack of confidence	3.07	(0.54)	n.s.	2.78	(0.52)	n.s.	2.77	(0.51)

n.s. = not significant.

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

\*\*\*  $p < 0.001$ .

and had reduced neck mobility, lower handgrip strength and poorer mental well-being.

The presence of tender spots over the patients' neck, shoulder and arm muscles was demonstrated by manual palpation and algometry, with a high level of correlation between the two methods. It is especially notable that the whiplash patients presented a lower tolerance to pain measured over digit II than the controls. This suggests a central disturbance of pain modulation in line with what has been suggested in tension-type headache. Schoenen et al. (34) reported that pain pressure thresholds were significantly lower in chronic tension-type headache not only at pericranial sites but also over the Achilles tendon. In whiplash injuries, a similar central dysmodulation of nociceptive impulses does not necessarily imply a central lesion caused by the accident. It might be induced by a long-lasting peripheral noxious input with sensitization of central synapses (43).

On the other hand, it is well documented in both animal experiments and clinical studies that brain damage can occur from whiplash injury without loss of consciousness (15, 26). Torres & Shapiro (41) compared patients with whiplash injuries with and without head injuries and patients with closed head trauma. They found that both groups presented a similar clinical picture consistent with the postconcussion syndrome (17). The symptoms presented by the patients in the present study, including those obtained from the MACL, are similar to the symptoms associated with the postconcussion syndrome, including head and neck pain, dizziness, anxiety, irritability,

difficulty in concentrating, insomnia and depressed mood. In this context, it is of interest to note that patients who have sustained severe cervical injuries with dislocation and fracture of cervical vertebrae resulting in permanent tetrapareses seldom or never complain of headache and neck pain. Similarly, it is considered that the postconcussion syndrome more often appears after light head injuries than after severe head injuries (17). This inverse relationship between the severity of the acute cervical or brain injury and the development of a post-traumatic syndrome is notable but has not so far provided any useful clue to the understanding of the syndrome.

There was a weak correlation ( $p < 0.05$ ) between muscle tenderness registered by manual palpation and the VAS registration of the intensity of spontaneous pain but no significant correlation between the VAS registration and muscle tenderness measured by means of algometry. This might be interpreted in various ways. Tenderness and spontaneous pain may have a common origin but develop independently of each other. There might be differentiated susceptibility levels of the nociceptors with respect to pressure and spontaneous pain: at one end a level with low tenderness but with spontaneous pain and at the other extreme high tenderness but no spontaneous pain. It is also possible that the occurrence of spontaneous pain requires the sum of nociceptive inputs from a number of tender spots, each of which possibly with low tenderness. There is sometimes, as in the present study, a delay in the occurrence of spontaneous pain. Studies of patients in this pain-free interval

could possibly answer the question concerning the temporal relationship between the occurrence of tender areas and the occurrence of spontaneous pain.

We have tried to ensure that the manual palpation did not influence the algometer test by allowing a sufficient delay between the two tests. In consecutive algometer measurements, the succeeding measurement might be affected. On the one hand, Kosek et al. (19) found that the PPT of the second immediate algometer measurement was significantly lower than the first and that a third measurement 20–30 minutes later was significantly higher than the first and second determinations. Brennum et al. (5), on the other hand, found no significant differences in PPTs during consecutive measurements with 10-second intervals. This is in line with studies of others, who found no difference in PPTs when remeasuring with varying time intervals (27, 33).

It is known that medical intervention can change the pain threshold (18). In the present study none of the patients or controls had taken any drugs during the 24 h prior to the investigation that might have influenced the pain threshold. It is rational to assume that the pain threshold becomes lower after cessation of analgesic intake compared with the pain threshold during analgesic intake. To the best of our knowledge, however, there are no studies which have evaluated the effect on pain threshold of the withdrawal of analgesics. As few of the patients were on continuous medication with analgesics, any transient withdrawal effects influencing the results of the whole patient group would in all likelihood have been minimal.

It is notable that four patients diagnosed as whiplash injuries presented their first symptoms 3 months after the injury. It cannot be excluded for certain that their symptoms have another aetiology than a whiplash injury, e.g. cervical spondylosis. However, some authors in the past have strongly emphasized that symptoms from the whiplash injury may be delayed for weeks or even months (4, 16, 35).

Pain after a whiplash injury may originate from numerous sources, e.g. periosteal, ligamentous, muscular fascial and tendinous tears, disc injury, instability and subluxation (2). The location of the pain may be remote from its source. Several authors have considered this pain to be referred from deep skeletal and soft tissue structures (13). The pain from the injured tissue might spread by means of spinal reflexes which cause muscle spasms and pain in a segmentally related area, which in turn further perpetuates the

firing of the anterior horn cells, which increases the muscle spasm, and so on (3). This implies that the patients' shoulder/arm pain is not necessarily caused by nerve root damage but might just as well be due to such self-propagating muscular pain analogous to the proposed development of myofascial pain (14, 42).

The weakness of the handgrip, as compared with controls, may be due to nerve root involvement, but it might also be a consequence of reflex inhibition of the involved muscles by pain. Such reflex inhibition of the quadriceps muscle has been noted in patients with knee joint and muscle pain (37). Furthermore, reduction of pain has been shown to reduce the muscle inhibition (38).

The reduced cervical mobility can be considered to be an expected consequence of the injury. The cervical joint apparatus is complex and many structures can be injured in the traumatized tissue. The healing process creates scar tissue which is less elastic than the original tissue and mobility is reduced. If only one or a few segments are involved, the reduced mobility will be compensated for by hypermobility at adjacent, uninjured levels, which in turn may result in degenerative disc disease and spondylosis. The relationship between cervical spondylosis and trauma has been pointed out by several authors (24, 44). Ehni (11) has coined the term "traumatic arthritism" for this condition.

Whether there is a relationship between the reduced cervical mobility and the neck pain is still an unsolved question. It has implications for the early treatment, however. Should the cervical spine be mobilized or immobilized? Both methods have been recommended (2). In the light of the natural history of the whiplash injury, the real value of any early treatment is unclear since 75% of the patients will spontaneously improve in the first few months after the injury (2). But it would be of exceptional value to find an intervention that prevented the development of chronic symptoms. So far no treatment has demonstrated this capacity (2). The treatment methods used in chronic whiplash patients are also unsatisfactory. A recent review by Teasell et al. (40) outlines a variety of treatments but none has been evaluated in controlled trials.

The patients in the present study showed an overall poorer mental well-being than healthy controls. This was so for five of the six dimensions and implies that the patients are, for example, more worried, tense, nervous, unconcentrated, irritated and insecure. Pain may be an aetiological and exacerbating factor for

many of these complaints and may account for a range of other seemingly diverse symptoms.

It may be concluded that patients with prolonged disability after a whiplash injury present a complex clinical picture, with both somatic and mental symptoms, most of which are hard to explain. This should be seen as reflecting the inadequacy of our diagnostic methods and not as a reason for classifying the whiplash injury syndrome as a non-organic disorder arising from neurosis and the desire for compensation.

#### ACKNOWLEDGEMENTS

This work was supported by grants from the Stiftelsen Lars Hiertas Minne and the Renee Eanders Hjälpfond.

#### REFERENCES

1. Awerbuch, M. S.: Whiplash in Australia: illness or injury. *Med J Aust* 157: 193–196, 1992.
2. Barnsley, L., Lord, S. & Bogduk, N.: Whiplash injury. *Pain* 58: 283–307, 1994.
3. Bogduk, N.: Innervation and pain patterns in the cervical spine. *Clin Phys Ther* 17: 1–13, 1988.
4. Braaff, M. M. & Rosner, S.: Symptomatology and treatment of injuries of the neck. *NY State J Med* 55: 237–242, 1955.
5. Brennum, J., Kjeldsen, M., Jensen, K. & Jensen, T. S.: Measurement of human pressure-pain threshold on fingers and toes. *Pain* 38: 211–217, 1989.
6. Carette, S.: Whiplash injury and chronic neck pain. *New Engl J Med* 15: 1083–1084, 1994.
7. Carlsson, A. M.: Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain* 16: 87–101, 1983.
8. Carlsson, J., Augustinsson, L.-E., Blomstrand, C. & Sullivan, M.: Health status in patients with tension headache treated with acupuncture or physiotherapy. *Headache* 30: 593–599, 1990.
9. Croft, A. C.: Soft tissue injury: long and short-term effects. In *Whiplash injuries* (ed. S. M. Foreman & A. C. Croft), pp. 271–327. Williams and Wilkins, London, 1988.
10. Crowe, H. E.: Injuries to the cervical spine. Paper presented at the meeting of the Western Orthopaedic Association. San Francisco, 1928.
11. Ehni, G.: Degenerative motion segment encroachments. In *Cervical arthrosis: diseases of the cervical motion segments*, 54 pp. Year Book, Chicago, 1984.
12. Evans, R. W., Evans, I. & Sharp, M. J.: The physician survey on the post-concussion and whiplash syndromes. *Headache* 43: 268–274, 1994.
13. Feinstein, B., Langton, J. N. K., Jameson, R. M. & Schiller, F.: Experiments of pain referred from deep somatic tissues. *J Bone Joint Surg* 36A: 981–997, 1954.
14. Friction, J. R.: Myofascial pain and whiplash. *Spine: State of the Art Reviews* 7: 403–422, 1993.
15. Gibbs, F. A.: Objective evidence of brain disorder in cases of whiplash injury. *Clin Electroencephalogr* 2: 107–110, 1971.
16. Gotten, N.: Survey of one hundred cases of whiplash injury after settlement and litigation. *JAMA* 162: 865–867, 1956.
17. Gurdjian, E. S. & Webster, J. E.: Head injuries. Mechanisms, diagnosis and management. Brown and Co, Boston, 1958.
18. Jensen, K., Orback Andersen, H., Olesen, J. & Lindblom, U.: Pressure pain threshold in human temporal region. Evaluation of a new pressure algometer. *Pain* 25: 313–323, 1986.
19. Kosek, E., Ekholm, J. & Nordemar, R.: A comparison of pressure pain threshold in different tissues and body regions. *Scand J Rehab Med* 25: 117–124, 1993.
20. Langemark, M. & Olesen, J.: Pericranial tenderness in tension headache. A blind controlled study. *Cephalalgia* 7: 249–255, 1987.
21. Macnab, I.: The whiplash syndrome. *Orthop Clin North Am* 2: 389–403, 1971.
22. Mills, H. & Horne, G.: Whiplash-manmade disease. *NZ Med J* 99: 373–374, 1986.
23. Moffett, J. A. K., Hughes, I. & Griffith, P.: Measurement of cervical spine movements using a simple inclinometer. *Physiotherapy* 75: 309–312, 1989.
24. Norris, S. H. & Watt, I.: The prognosis of neck injuries resulting from rear-end vehicle collisions. *J Bone Joint Surg Br* 65: 608–611, 1983.
25. Öberg, T., Öberg, U. & Karsznia, A.: Handgrip and fingerpinch strength. *Phys Theory Practice* 10: 27–34, 1994.
26. Ommaya, A. K., Faas, F. & Yarnell, P.: Whiplash injury and brain damage: an experimental study. *JAMA* 204: 285–289, 1968.
27. Ohrbach, R. & Gale, E.: Pressure pain thresholds, clinical assessment, and differential diagnosis; reliability and validity in patients with myogenic pain. *Pain* 39: 157–169, 1989.
28. Pearce, J. M. S.: Whiplash injury: a reappraisal. *J Neurol Neurosurg Psychiatry* 52: 1329–1333, 1989.
29. Pennie, B. & Agambar, L.: Patterns of injury and recovery in whiplash. *Injury* 22: 57–59, 1991.
30. Persson, L.: Unpublished data. Gothenburg College of Health Sciences, Department of Nursing, Göteborg, Sweden.
31. Radanov, B. P., Stefano, G. D., Schnidrig, A. & Sturzenegger, M.: Common whiplash: psychosomatic or somatopsychic? *J Neurol Neurosurg Psychiatry* 57: 486–490, 1994.
32. Radanov, B. P., Sturzenegger, M., Stefano, G. D. & Schnidrig, A.: Relationship between early somatic, radiological, cognitive and psychosocial findings and outcome during a one-year follow-up in 117 patients suffering from common whiplash. *Br J Rheumatol* 33: 442–448, 1994.
33. Reeves, J., Jaeger, B. & Graff-Radford, S.: Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity. *Pain* 24: 313–321, 1986.
34. Schoenen, J., Bottin D., Hardy, F. & Gerard, P.: Cephalic and extracephalic pressure pain threshold in chronic tension-type headache. *Pain* 47: 145–149, 1991.
35. Schutt, C. H. & Dohan, F. C.: Neck injury to women in auto accidents. *JAMA* 206: 2689–2692, 1968.
36. Sjöberg, L., Svensson, E. & Persson, L.: The measurement of mood. *Scand J Psychol* 20: 1–18, 1979.
37. Spencer, J. D., Hayes, K. C. & Alexander, I. J.: Knee joint effusion and quadriceps reflex inhibition in man. *Arch Phys Med Rehabil* 65: 171–177, 1984.
38. Stokes, M. & Young, A.: A contribution of reflex inhibition to arthrogenous muscle weakness. *Clin Sci* 67: 7–14, 1984.

39. Sturzenegger, M., Distefano, G., Radanov, B. P. & Schnidrig, A.: Presenting symptoms and signs after whiplash injury: the influence of accident mechanisms. *Neurology* 44: 688-693, 1994.
40. Teasell, R. W., Shapira, A. P. & Mailis, A.: Medical management of whiplash injuries: an overview. *Spine: State of the Art Reviews* 7: 481-499, 1993.
41. Torres, F. & Shapiro, S. K.: Electroencephalograms in whiplash injury. *Acta Neurol* 5: 28-35, 1961.
42. Travell, J. G. & Simons, D. G.: Myofascial pain and dysfunction. The trigger point manual. Williams and Wilkins, Baltimore, 1983.
43. Wall, P. D.: Changes in adult spinal cord induced by changes in the periphery. *In* Development and plasticity of the mammalian spinal cord (ed. M. E. Goldberger, A. Gorio & M. Murray), pp. 101-113. Padova, Liviana Press, 1986.
44. Watkinson, A., Gargan, M. F. & Bannister, G. C.: Prognostic factors in soft tissue injuries of the cervical spine. *Injury* 22: 307-309, 1991.

*Accepted March 21, 1996*

*Address for offprints:*

Ylva Hagström  
Department of Physiotherapy  
Sahlgrenska Hospital  
S-413 45 Göteborg  
Sweden