Geoffrey N. Klein
Anne F. Mannion
Manohar M. Panjabi
Jiri Dvorak

Trapped in the neutral zone: another symptom of whiplash-associated disorder?

Received: 6 April 2000
Revised: 8 December 2000
Accepted: 18 December 2000
Published online: 16 February 2001
© Springer-Verlag 2001

This study was supported by the
Schulthess Klinik Research Fund, Zürich,
Switzerland.

G. N. Klein · A. F. Mannion (✉)
J. Dvorak
Spine Unit, Schulthess Klinik,
Lenggghalde 2, 8008 Zurich, Switzerland
e-mail: afm@kws.ch,
Tel.: +41-1-3857584, Fax: +41-1-3857590

M. M. Panjabi
Biomechanics Laboratory,
Department of Orthopaedics
and Rehabilitation,
Yale University School of Medicine,
New Haven, Connecticut, USA

Abstract Instability of the cervical spine following whiplash trauma has been demonstrated in a number of studies. We hypothesized that, in patients with whiplash-associated disorder, rotation of the head would be accompanied by an earlier onset of neck muscle activity to compensate for intrinsic instability. The aim of the study was to examine the range of motion (RoM) of the cervical spine and the onset and activity of the sternocleidomastoid (SCM) muscles during axial rotation, in healthy control subjects and in patients with chronic whiplash-associated disorder. Forty-eight control subjects (42% male) and 46 patients (33% male) with chronic whiplash-associated disorder (symptoms lasting longer than 3 months) were examined. Cervical axial RoM differed significantly ($P=0.0001$) between the

groups, with the whiplash patients showing lower values ($83^\circ\pm 30^\circ$) than the healthy controls ($137^\circ\pm 19^\circ$). The whiplash patient group showed no evidence of the predicted earlier activation of SCM muscles. Many patients never reached the point in the RoM where SCM muscle activity rises steeply, as it does in the healthy controls (the 'elastic zone'), and their movements remained mostly within the region of low muscle activity (the 'neutral zone'). The whiplash patients appeared either unable or unwilling to drive the cervical spine into this region of high muscle activity, possibly because they were restricted by existing pain or fear of pain.

Keywords Neutral zone · Spinal instability · Surface EMG · Whiplash

Introduction

There is a general consensus in the literature that whiplash injuries and their sequelae are highly prevalent, and affect a significant proportion of the population [8]. A recent study carried out in Germany reported that the proportion of acceleration-related injuries of the cervical spine increased three-fold between 1985 and 1997 [23]. Between 10 and 20% of whiplash patients will develop chronic disabling symptoms following their initial injury [3]. Whiplash-associated disorders include neck pain, headache, shoulder pain, paresthesia, visual disturbance, auditory disturbance, and dizziness [26]. Although the asso-

ciated costs have been estimated and the symptoms are well known, many questions remain as to what exactly whiplash injury comprises.

Some studies have shown that cervical instability occurs in patients with whiplash-associated disorder [1, 11, 12]. A spinal unit is defined as unstable if, under normal loading, the spine can not prevent damage, deformation, or irritation [19]. Functional flexion/extension radiographs of the cervical spine have demonstrated that cervical hypermobility and a shift in the center of vertebral rotation exist in patients with whiplash-associated disorder compared with healthy subjects [7]. Also, studies of the helical access of rotation have shown that whiplash patients rotate at an uncharacteristic level or in an uncharac-

teristic manner [17]. The fact that spinal fusion, an extreme treatment for instability, has also been shown to remove pain in whiplash patients [9] suggests that instability and pain are strongly related.

Cadaveric research has demonstrated that spinal instability is associated with an increase in the neutral zone (NZ) of the spine [22]. Total range of motion (RoM) can be divided into two components, the NZ and the elastic zone (EZ). Initially, minimal resistance to motion is encountered as the spine rotates through the NZ, after which continuing movement is accompanied by higher levels of resistance, as the spine rotates within its EZ [20]. Studies subjecting sections of osteoligamentous cadaveric cervical spines to a series of whiplash trauma injuries of gradually increasing impact acceleration have shown that changes in the NZ are better indicators of spinal instability than changes in the RoM [19]. As the force of the simulated trauma increases, the NZ increases first, before significant changes in RoM are observed [20]. The first significant increases in the NZ occur while there are only few observable anatomic lesions [18]. However, after simulated muscle forces are applied to injured cadaveric spine sections, stability increases and the NZ decreases back towards the uninjured state [22, 27].

Two studies have confirmed that the existence of a NZ can also be demonstrated in vivo during movements made in the cardinal planes [15, 16]. In both studies, the resistance of the cervical spine during passive movements was measured. The patients were required to relax their muscles during the measurements in an attempt to ensure that only the spine's passive resistance was measured, rather than muscle forces. The cervical spine showed little resistance to rotation during small angular displacements (the NZ) [15]. To achieve greater rotation, increasingly greater torque was required to rotate the spine (the EZ). In both these studies, only healthy subjects were measured [15, 16], and in one of them the movement pattern in axial rotation was not examined [16].

We hypothesised that, in patients with whiplash-associated disorder, rotation of the head may be accompanied by an earlier onset of neck muscle activity to compensate for 'instability', i.e., that these patients would demonstrate a smaller in vivo NZ.

The aim of the present study was to test the hypothesis that the sternocleidomastoid (SCM) muscle (the main rotator of the head) is activated earlier in the RoM in patients with chronic whiplash disorder than in 'normal' healthy controls.

Materials and methods

Subjects

Forty-eight healthy volunteers (mostly hospital employees; 42% male) and 46 patients with chronic whiplash-associated disorder (33% male) participated in the study. The proportion of men and women in each group was not significantly different ($P=0.49$).

Table 1 Clinical/litigation details of the whiplash patients

Parameter	
Mean symptom duration (mo)	34.1
Headache (%)	53.1
Visual disturbances (%)	26.5
Nausea (%)	12.2
Paresthesia (%)	30.6
Dizziness (%)	32.7
Constant pain (%)	30.6
Motion-induced pain (%)	69.2
Local pain (%)	87.8
Referred pain (%)	69.4
Radicular pain (%)	5.1
Litigation cases (%)	51.0
Claiming disability allowance (%)	59.2

However, the controls were younger (mean age 28.8 ± 6.7 years) than the patients (mean age 36.4 ± 10.1 years) ($P=0.001$), and this was taken into account in the statistical analyses with respect to the group comparisons. Some of the healthy control subjects had experienced neck pain in the past, but none was suffering with any symptoms at the time of the test. The patients had been referred from their general practitioners to the neurology and rheumatology departments of the hospital for further evaluation as a result of continuing neck pain. Chronic whiplash-associated disorder was defined as symptoms (mostly neck pain) lasting more than 3 months attributed to a motor vehicle accident [2]. The mean ($\pm$ SD) duration of symptoms was $33.7 (\pm 26.7)$ months (range 4–120 months). The clinical symptoms of the patients, as determined from the medical history taken during the clinical examination, are shown in Table 1. The severity of whiplash injuries in the patients varied between whiplash grades I and III, most being grade II [26]. No analgesics were taken by the patients prior to testing.

Instrumentation

The Spine Motion Analyzer CA-6000 (CA-600; Orthopedic Systems Inc., Hayward, Calif.) has previously been described in detail [6, 13]. Briefly, it comprises a multibar linkage arm that computes unrestricted six-degree-of-freedom motion using high precision potentiometers. To measure cervical axial rotation, the lower linkage arm of the CA-6000 was attached to the back of a shoulder fixation chair and the upper linkage arm to a metal post on the rear of a helmet (Fig. 1).

Pairs of disposable surface electrodes (sensitivity 0–1500 μ V; MSB Biotrace-HR Ag/AgCl ECG electrodes; MSB, Wiltshire, UK) were attached bilaterally over the belly of the SCM muscles, 4 cm below the mastoid process, and parallel to the muscle's line of action (i.e., slightly obliquely with respect to the vertical) (Fig. 1). Electrode pairs were also positioned bilaterally over the paraspinalis muscles, halfway between the occipital bump and C7, 1 cm lateral to the midline of the spine (Fig. 1). (Paraspinalis muscle activity was monitored to confirm that the subjects were engaged in pure rotation and were not substantially flexing or extending the head.) At each site, the electrode pair was positioned with a center-to-center distance of approximately 25 mm. A reference electrode was positioned over the ulna styloid. Prior to attachment of the electrodes, the skin was abraded with special skin preparation pads (Bioelect Cardioprep pads; MSB, Wiltshire, UK) and cleaned with alcohol.

The raw electromyographic (EMG) signals were sampled at a frequency of 1200 Hz per channel, band-pass filtered (25–500 Hz

Fig. 1 Instrumentation used to measure range of motion (RoM) and electromyographic (EMG) activity of the sternocleidomastoid (SCM) muscles during axial rotation of the cervical spine

with a 50 Hz notch filter), full-wave rectified and integrated within the EMG hardware, and then transferred to the personal computer.

Axial rotation was measured separately for the left and right sides. Motion was sampled at a frequency of 50 Hz. Before the test, the subject placed their head in a neutral position to zero the device. Then, for right axial rotation, the subject began from a starting position slightly left of center (approx. 15°) and rotated slowly, crossing through the neutral position, as far to the right as possible (this motion lasted about 10 s). Afterwards, left axial rotation was measured in a similar manner. Before each measurement, the investigator demonstrated the motion and the subject practiced until the timing was correct.

Analysis

Text files containing the data were exported from the CA-6000 computer into Microsoft Excel, which was used to plot a graph of axial rotation versus SCM muscle activity. Many attempts were made to quantify the start of the region of higher muscle activity, and thus identify the 'NZ', such that differences between the groups could be computed statistically. Most of these methods came from the available literature. McClure et al. [15] defined the region of low muscle activity, the NZ, as ending when stiffness

equaled 20% of maximum stiffness. In the present study values were computed using McClure's method (using 20% of maximum EMG), but for many patients this did not accurately reflect the start of muscle activity as seen by visual examination of the graphs. In analysing the in vivo NZ of the lumbar spine, Sandler et al. [24] defined the end of the NZ from the biphasic EMG versus displacement curve, using two different methods: the interception of two linear fits to the two phases of the curve, and a 'cut-off' slope (NZ ends when the increase in EMG per degree of rotation exceeds a chosen cut-off point). Using both of Sandler's methods resulted in the same problem, namely that the angle defined as the end of the NZ inconsistently reflected the start of the increased muscle activity as judged by eye. Several other mathematical methods were devised in an attempt to solve this problem, but none was successful. As such, it was decided that the most honest and informative way of presenting the data would be to plot all the individual data from both patients and controls on one graph, and to visually inspect the muscle activity patterns of the two groups. Although only descriptive, this method allowed the questions posed by the study to be answered, whilst avoiding problems caused by misleading mathematical solutions.

Statistical analysis

The dependence of age on RoM was examined using linear regression.

Differences between the mean values of RoM for the two groups were examined using analysis of variance or analysis of covariance, where necessary. Significance was accepted at the 5% level.

Results

Relationship between RoM and age

Increasing age was associated with a decrease in RoM (correlation coefficient = -0.39; $P < 0.001$). As the patient group was significantly older than the control group, this necessitated the use of analysis of covariance for the further investigations of group differences in RoM.

Comparison of mean RoM between the groups

Mean values for right, left, and total RoM were higher in the control group than in the group of whiplash patients (Table 2), and the differences were significant ($P < 0.036$).

Table 2 Mean (SD) range of motion (RoM) in rotation, in controls and whiplash patients

Direction of movement	RoM (degrees)		
	Controls	Whiplash patients	<i>P</i> (between the groups) ^a
Left	69.4 (9.8)	43.1 (16.4)	0.036
Right	67.8 (10.8)	41.7 (16.9)	0.017
Total	137.2 (18.5)	84.8 (31.0)	0.016

^aSignificance determined from analysis of covariance, where age acts as covariant

Table 3 Mean (SD) RoM in subgroups of whiplash patients with and without specific clinical symptoms/compensation (N/A analysis not possible, as <20% of the group suffered with the symptom)

Symptom (proportion of patients affected)	RoM in patients with symptom (degrees)	RoM in patients without symptom (degrees)	<i>P</i>
Headache (67%)	83.4 (30.3)	87.5 (33.2)	0.68
Visual disturbances (26%)	77.9 (32.4)	87.2 (30.6)	0.38
Nausea (11%)	N/A	N/A	N/A
Paresthesia (26%)	83.9 (31.2)	85.1 (31.4)	0.91
Dizziness (30%)	70.9 (19.5)	90.8 (33.3)	0.04*
Constant pain (30%)	65.0 (18.1)	93.4 (31.7)	0.003*
Local pain (80%)	85.1 (28.6)	83.5 (41.3)	0.90
Referred pain (65%)	84.8 (29.7)	87.8 (35.9)	0.99
Radicular pain (2%)	N/A	N/A	N/A
Litigation cases (48%)	83.9 (28.8)	85.6 (33.5)	0.85
Disability allowance (59%)	74.4 (29.3)	99.5 (27.8)	0.006*

* $P < 0.05$; difference in RoM between patients with and those without the symptom

even when age was covaried for. Within each group, the mean values of right and left RoM did not differ significantly ($P > 0.05$).

Effect of symptoms/compensation claims on mean range of motion

The data were analyzed to examine whether there were differences in the RoM between patients with and without specific symptoms (Table 3). The analysis was not done when one group had too few patients with the specific symptom (under 20%). For each of the symptoms examined, there was no significant difference in either mean age or gender distribution between the group that did and

the group that did not declare suffering the particular symptom ($P > 0.15$ for each symptom). Thus no covariance for age or gender was required for these analyses.

Mean RoM was significantly lower in the subgroup of patients who declared being in constant pain and suffering dizziness than it was in the subgroup who did not experience these symptoms. In addition, patients who were receiving a disability allowance demonstrated a significantly lower mean RoM than those who were not in receipt of such an allowance. Involvement in litigation had no influence on the recorded RoM.

For the other symptoms, the mean RoM was generally lower in patients with the given symptom than in those without that symptom, but the differences were not significant.

Muscle activity

Figure 2 shows the relationship between RoM and SCM EMG (as agonist and antagonist) for one healthy subject. There is a clearly identifiable region of low SCM muscle activity during the initial phase of motion (0° – 30°), after which activity increases somewhat (between 30° and 40°) and then rises steeply during movement to the end of the RoM (75°).

The individual muscle activity patterns for all the healthy subjects and all the patients with chronic whiplash are shown in Fig. 3A. It can be seen that there are no obvious differences between the patients and the controls in the pattern of change in muscle activity with increasing rotation. However, what clearly differs between the patient groups and the normal controls is the point at which the movement ends. To assist in visualising the angle at which the subjects stop rotating, every 10° along the angle axes of Fig. 3A the percentages of patients and healthy controls who could rotate to that point are indicated; this

Fig. 2 Relationship between rotation angle and SCM surface EMG activity for one healthy subject

Fig.3 **A** Relationship between rotation angle and SCM surface EMG activity for patients and controls. **B** Proportion of patients and controls reaching defined rotation angles during rotation to the right (similar data were obtained for rotation to the left)

is also demonstrated graphically in Fig. 3B. Only approximately 50% of the whiplash patients were able to rotate beyond 40°, the point around which the large increase in muscle activity occurs.

Effect of symptoms/compensation claims on mean RoM

Within the group of whiplash patients, those with and without specific symptoms were compared to see whether there were any differences in the onset of muscle activity.

All the symptoms listed in Table 3 were analyzed. This analysis was done graphically in a similar manner to that described above. There was no evidence of different patterns or onsets of muscle activity between the subgroups of patients with and without the different symptoms.

Antagonist muscle activity

Antagonist SCM EMG activity (i.e., activity of the right SCM, whilst turning to the right) was analyzed to see

whether the onset or size of co-contractions differed between patients and controls. This analysis was done by overlaying the data for antagonist and agonist muscles on one graph, for each individual. For both groups there was no antagonist muscle activity in the initial stage (0° – 30°) of the RoM during which there was low agonist muscle activity. All subjects who were able to rotate into the EZ showed small co-contractions when there was large agonist muscle activity (Fig. 2). In the patient group, there was no marked increase in antagonist muscle activity at the end of the RoM that might otherwise have indicated protective co-contraction.

Discussion

The CA-6000 spine motion analyzer has been used in numerous studies concerned with establishing normative cervical range of motion data for individuals of differing age and gender [5, 6, 13] and for identifying deficits in mobility associated with certain disorders of the cervical spine [10]. It has been established as a reliable and popular tool for mobility measurements [6, 13, 14], although the capacity of the instrument to simultaneously record muscle activity during measurement of the RoM has not been well exploited.

The previously reported relationship between age and mobility [6] was confirmed in the present study, and the results also support the conclusion of Hagen et al. [10], that cervical RoM testing is a useful tool to assess impairment. The mean RoM was significantly lower in the group of whiplash patients than in the healthy controls and, within the patient group, symptomatic patients had for the most part lower average RoMs than asymptomatic patients. These differences were statistically significant for the symptoms of 'constant pain' and 'dizziness', indicating that dizziness should perhaps be studied as a potential predictor of patients whose problems will ultimately be associated with more severe limitations in mobility. Whiplash patients collecting disability allowance had a significantly smaller RoM than those receiving no such benefit. However, there was no association between RoM and litigation status, suggesting that the phenomenon of malingering and secondary gain did not account for the patients' 'underperformance'.

The SCM muscle activity pattern observed for healthy subjects appears to be an effective technique to visualise the NZ: the general pattern of activity during rotation was in agreement with both *in vivo* measurements of resistance to cervical rotation [15] and *in vitro* measurements of load-displacement in cadaveric spine sections [18, 19, 20, 21, 22]. It is logical that the EMG patterns should accord with the previous assessments of stiffness, because the rotator muscles (measured in this study) must generate the torque (measured in previous studies) required to overcome the inherent stiffness of the spine in effecting

rotational displacement (measured in both studies). Serious difficulties arose in the numerical quantification of the *in vivo* NZ: many mathematical models were investigated in an attempt to define the inflection point of the load-displacement curve, none of which, when uniformly applied to all individuals' data, gave an honest representation of the point that would have been chosen by visual assessment. This may reflect the difficulties of using 'active' indications of resistance to movement (i.e., EMG), particularly in groups of patients whose movements are not always made in the smoothest or most energetically efficient manner. Alternatively, the inability to consistently describe the data using one unique mathematical function may simply have resulted from the fact that few patients actually moved into the second phase (EZ).

The whiplash patients did not show the hypothesised earlier muscle activation when compared with the healthy subjects. Instead, the muscle activity patterns of the controls and the patients proceeded in a similar fashion throughout the (albeit shorter) excursions that the patients undertook. Thus, the movement pattern of the whiplash patients can be considered to be a 'truncated' version of that of the controls – whereby the patients simply do not reach the same end-point of large movement amplitudes and high EMGs – rather than a 'scaled down' (to fit into their smaller RoM) version.

The point at which the patients tended to stop rotating occurred before, or during, the period in which most healthy subjects required a marked increase in muscle activity to effect further movement. This finding may play a central role in explaining the lower RoM in the patient group. The SCM spans many vertebrae, and when activated exerts both rotational and compressive forces along the axis of the spine. According to a biomechanical computer model of the cervical spine, axial rotation of less than 35° does not need great muscle forces, but rotation greater than 35° causes muscle and joint reaction forces to increase rapidly [25]. Applying an elevated force to an already damaged spine may cause pain, which in turn would cause the patients to cease moving. As movement into extreme positions of rotation would also result in increased tension of the passive tissues of the spine, it is understandable that patients may attempt to avoid such movements. Whether this reluctance or inability to move into the region of high muscle activity is a characteristic specific to whiplash patients, or is simply a manifestation of any painful neck disorder, remains to be established.

The fact that many patients appeared unable to rotate into the region of higher muscle activity suggests that this may represent a useful test to evaluate the severity of the disorder and the effectiveness of therapy, as an adjunct to RoM testing. Individual values of RoM can be difficult to interpret: one can compare with 'normal values', but, without a knowledge of the individual's previous capacity, it is difficult to quantify the effects of injury with any certainty. If, however, it could be shown that a patient exhib-

ited a reduced RoM, but with the 'complete' pattern of muscle activity i.e., including motion into the EZ, then the reduced mobility would likely be an intrinsic characteristic of the individual, governed by anatomical constraints, rather than the result of injury. If, on the other hand, a reduced RoM were coupled with a failure to enter the EZ, then this would suggest that expression of the true physical (or anatomical) capacity had somehow been inhibited, either voluntarily or reflexly.

In explaining the lack of any early muscle activity in the patients, a number of factors must be considered. It is possible that instability is less important in these patients than originally supposed; there are certainly a number of other explanations for the symptoms of whiplash injury [4]. It is also possible that our methods of measurement were not sensitive enough to detect changes associated with spinal instability. Conceivably the deeper, short neck muscles are more important for spinal stability than is the SCM. In stabilising the spine, the deeper muscles have several advantages over the prime movers: they are smaller and closer to the spine and can provide finer control with less muscle length change. Nonetheless, increased deep muscle activation would be expected to

cause a greater internal resistance to spinal movement. This in turn would result in the need for an increased muscle force to rotate the spine, which would be reflected in higher SCM EMG values. That higher EMG values were not recorded suggests that the whiplash patients did not display increased deep muscle activity. Future studies employing needle EMG examinations of the deeper muscles would, however, be required to confirm this.

Conclusions

This study confirmed that RoM remains a useful tool to measure cervical spine impairment. There was no evidence to support the hypothesis that patients with chronic whiplash-associated disorder activate the SCM earlier during controlled rotation of the cervical spine. Rather, it appears that these patients are unable or unwilling to rotate into the region where higher muscle activity is required to effect further movement. The use of EMG in conjunction with RoM testing may provide a useful means for explaining individual deficits in RoM.

References

1. Algers G, Pettersson K, Hildingsson C, Toolanen G (1993) Surgery for chronic symptoms after whiplash injury. Follow-up of 20 cases. *Acta Orthop Scand* 64:654–656
2. Barnsley LS, Lord SM, Bogduk N (1994) Whiplash injury – a clinical review. *Pain* 58:283–307
3. Bogduk N (1999) The neck. *Bailliere's Clin Rheumatol* 13:261–285
4. Bogduk N, Lord SM (1998) Cervical spine disorders. *Curr Opin Rheumatol* 10:110–115
5. Chen J, Solinger AB, Poncet JF, Lantz CA (1999) Meta-analysis of normative cervical motion. *Spine* 25:1571–1578
6. Dvorak J, Antinnes J, Panjabi M, Loustalot D, Bonomo M (1992) Age and gender related normal motion of the cervical spine. *Spine* 17:393–398
7. Dvorak J, Panjabi MM, Grob D, Novotny JE, Antinnes JA (1993) Clinical validation of functional flexion/extension radiographs of the cervical spine. *Spine* 18:120–127
8. Freeman MD, Croft AC, Rossignol AM, Weaver DS, Reiser M (1999) A review and methodologic critique of the literature refuting whiplash syndrome. *Spine* 24:86–96
9. Grob D (1998) Posterior surgery. In: Gunzburg R, Szpalski M (eds) *Whiplash injuries: current concepts in prevention, diagnosis, and treatment of the cervical whiplash syndrome*. Lipincott-Raven, Philadelphia, pp 241–246
10. Hagen KB, Harms-Ringdahl K, Enger NO, Hedenstad R, Morten H (1997) Relationship between subjective neck disorders and cervical spine mobility and motion-related pain in male machine operators. *Spine* 22:1501–1507
11. Hamer AJ, Gargan MF, Bannister GC, Nelson RJ (1993) Whiplash injury and surgically treated cervical disc disease. *Injury* 24:549–550
12. Jonsson H Jr, Cesarini K, Sahlstedt B, Rauschnig W (1994) Findings and outcome in whiplash-type neck distortions. *Spine* 19:2733–2743
13. Lantz CA, Chen J, Buch D (1999) Clinical validity and stability of active and passive cervical range of motion with regard to total and unilateral uniplanar motion. *Spine* 24:1082–1089
14. Mannion AF, Klein GN, Lantz C, Dvorak J (2000) Range of global motion of the cervical spine: intraindividual reliability and the influence of measurement device. *Eur Spine J* 9:379–385
15. McClure P, Siegler S, Nobile R (1998) Three-dimensional flexibility characteristics of the human cervical spine in vivo. *Spine* 2:216–223
16. McGill S, Jones K, Bennett G, Bishop PJ (1994) Passive stiffness of the human neck in flexion, extension, and lateral bending. *Clin Biomech* 3:194–198
17. Osterbauer PJ, Long K, Ribaud TA, Petermann EA, Fuhr AW, Bigos SJ, Yamaguchi GT (1996) Three-dimensional head kinematics and cervical range of motion in the diagnosis of patients with neck trauma. *J Manip Physiol Ther* 4: 231–237
18. Oxland TR, Panjabi MM (1992) The onset and progression of spinal injury: a demonstration of neutral zone sensitivity. *J Biomech* 10:1165–1172
19. Panjabi MM (1992a) The stabilizing system of the spine. I. Function, dysfunction, adaptation, and enhancement. *Spine* 4:383–389
20. Panjabi MM (1992b) The stabilizing system of the spine. 2. Neutral zone and instability hypothesis. *Spine* 4: 390–397
21. Panjabi M, Dvorak J, Crisco J, Oda T, Hilibrand A, Grob D (1991) Flexion, extension, and lateral bending of the upper cervical spine in response to alar ligament transections. *J Spinal Disord* 2:157–167
22. Panjabi MM, Nibu K, Cholewicki J (1998) Whiplash injuries and the potential for mechanical instability. *Eur Spine J* 7:484–492

-
23. Richter M, Otte D, Pohlemann T, Krettek C, Blauth M (2000) Whiplash-type neck distortion in restrained car drivers: frequency, causes and long-term results. *Eur Spine J* 9:109–117
24. Sandler AJ, Kendall N, Macias ME, Dvorak J, Panjabi MM (1996) Normal in vivo values of the neutral zone in lumbar axial rotation. *Proceedings of the European Spine Society Annual Meeting, Zürich, November 1996*, pp 34, 35
25. Snijders CJ, Hoek van Dijke GA, Roosch ER (1991) A biomechanical model for the analysis of the cervical spine in static postures. *J Biomech* 24:738–792
26. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, Zeiss E (1995) Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining whiplash and its management. *Spine* 20 [Suppl]: 1S–73S
27. Wilke HJ, Wolf S, Claes LE, Arand M, Wiesend A (1995) Stability increase of the lumbar spine with different muscle groups: a biomechanical in vitro study. *Spine* 2:192–198