

REVIEW OF THE LITERATURE



Injury Threshold: Whiplash-Associated Disorders

Charles G. Davis, DC^a

ABSTRACT

Objectives: To review current knowledge and recent concepts of the causes of injuries after minor impact automobile collisions and to acquaint those who treat these types of injuries with possible injury thresholds and mechanisms that may contribute to symptoms.

Data Sources: A review of literature involving mechanisms of injury, tissue tensile threshold, and neurologic considerations was undertaken. A hand-search of relevant engineering, medical/chiropractic, and computer *Index Medicus* sources in disciplines that cover the variety of symptoms was gathered.

Results: Soft-tissue injuries are difficult to diagnose or quantify. There is not one specific injury mechanism or threshold of injury. With physical variations of tissue tensile strength, anatomic differences, and neurophysiologic considerations, such threshold designation is not possible.

Conclusions: To make a competent assessment of injury, it is



important to evaluate each patient individually. The same collision may cause injury to some individuals and leave others unaffected. With the variability of human postures, tensile strength of the ligaments between individuals, body positions in the vehicle, collagen fibers in the same specimen segment, the amount of muscle activation and inhibition of muscles, the size of the spinal canals, and the excitability of the nervous system, one specific threshold is not possible. How individuals react to a stimulus varies widely, and it is evident peripheral stimulation has effects on the central nervous system. It is also clear that the somatosensory system of the neck, in addition to signaling nociception, may influence the control of neck, eyes, limbs, respiratory muscles, and some preganglionic sympathetic nerves. (J Manipulative Physiol Ther 2000;23:420-7)

Key Indexing Terms: Whiplash Injury; Cervical Vertebrae; Spine; Central Nervous System

INTRODUCTION

The method of research of whiplash varies from mail responses, emergency department and doctor records, and crash testing of cadavers, animals, and human volunteers. Whiplash-associated injuries may occur from any angle, with rear-impact collision injuries occurring at a higher rate.¹ Those who develop chronic conditions from whiplash have been reported from none² to 86%.³ It is difficult to extrapolate from the small number of samples of these reports and apply them to the population at large. Whiplash injuries are common, with some symptoms persisting for a number of years in a minority of patients. Vehicular damage may not occur with a delta velocity of less than 13 km/hour,^{4,5} and there does not appear to be a relation between vehicle damage and occupant injury.^{6,9}

Injury has been produced with in vitro ligament tensile tests with force less than the weight of the average head in volunteers with a speed change of 4 km/hour (2.5 mph),¹⁰ torso acceleration of 4.5g,¹¹ and extrapolated from primates, a 50% chance of concussion in a 5-g acceleration.¹²

Experimental studies and postmortem studies have yet to prove the lesion of whiplash, but they have set out possibilities. The lesions are likely tears to muscles, rim lesions of the disks, and occult fractures or injuries to the zygapophyseal joints.¹³

DISCUSSION

Today, vehicles are made with crumple zones. These zones are designed to absorb energy and leave the passenger compartment intact. In a collision with little or no damage to a vehicle, the collision impact would be a sharp impulse with little to no absorption of energy. In whiplash, an inertial type of injury, there is complex buckling with concomitant flexion and extension occurring simultaneously in different regions of the cervical spine. Because of inertia, an S-shaped curvature takes place. This S-shape phase shift has been found in volunteers,¹⁴ cadavers,¹⁵ and animal testing.¹⁶ In a rear-end impact, the neck forms an S-shaped configuration at 50 to 75 ms after accelerating motion. This time period shows cervical spine flexion at the upper levels and greater extension at the lower levels than in the later stages of maximum extension of the head. At 100 to 125 ms, the cervical spine reverts to a C-shape curvature of the entire cervical spine. The physiologic extension limits were exceeded at the lower intervertebral levels. Therefore although there is no gross hyperextension/hyperflexion with ranges of motion in whiplash experiments, segmental levels may exceed physiologic limits

^aPrivate practice of chiropractic, Azusa, Calif. Submit reprint requests to: Charles G. Davis, DC, 474 S Citrus, Azusa, CA 91702.

Paper submitted July 12, 1999; in revised form August 6, 1999.
doi:10.1067/mmt.2000.108140

without overall cervical hyperextension/hyperflexion.¹⁵ This type of injury may involve multiple, noncontiguous mechanisms of injury. There may be injuries to nonadjacent segments, as in damage to the lower cervical segments and at the occipital-atlantal segment. There may be apparently non-musculoskeletal symptoms, such as dizziness, vertigo, mild brain injury, and proprioceptive disorders.

The human spinal column, which is devoid of muscle function, is incapable of carrying the loads imposed on it.¹⁷ Without muscles, the spine buckles under very low loads. The average critical load that causes the osteoligamentous human cervical spine to buckle is 10.5 N, with a standard deviation (SD) of 3.8 N. This is about one fifth to one quarter the weight of the average head.¹⁸ Instability has been defined as loss of stiffness. Mechanical instability is an unstable structure that is not in a state of equilibrium.¹⁹ Mechanical instability has been induced to the cervical spine in vitro, when the acceleration of the T1 vertebrae is subjected to a 4.5-g rear-end acceleration.¹¹ In clinical instability under physiologic loads, there are changes in the patterns of motion that may result in neurologic deficit, excessive deformity, and/or pain, acutely or with time.²⁰ The neutral zone, or lax zone, may be a better parameter in defining the onset and progression of spinal injury than range of motion. The upper boundary of the neutral zone has been described as the displacement at which the ligamentous resistance begins.^{21,22}

Studies have been attempted that evaluate possible injury thresholds based on cadavers and volunteers. One engineering study had their important torque at the occipital condyles. An injury envelope of 47 Nm (35 ft-lb) was proposed as noninjurious, and 57 Nm (42 ft-lb) was proposed to cause injury based on a static torque test. The injury measurement was made from an extrapolation of visible ligamentous damage at 33 Nm (24.6 ft-lb) from a cadaver.²³ The Society of Automotive Engineers indicates a torque of 57 Nm (42 ft-lb) to represent a severe injury.²⁴ The difference in these forces fall within the SD of ligaments tested for failure.^{25,26} Anatomic studies of the tensile strength of spinal ligaments reveal a wide range of variability to failure (Table 1). Variability exists among suboccipital muscles,²⁹ articulations,^{30,31} and in the same specimen at the same level from anterior to posterior.³² In vitro failure testing of whole cadavers and on cadaveric spinal segments has resulted in widely varying patterns of injuries. Even controlled flexion or flexion-compression loading of 2-vertebrae functional spinal units has resulted in a wide variety of injuries, including disruption of the supraspinous and interspinous ligaments, ligamentum flavum, facet capsules, and disks.³³ There are also anatomic differences between the cervical and lumbar spine.³⁴

In a low-velocity impact, the soft tissue is seldom torn completely. It is most likely stretched beyond its elastic limit, resulting in an incomplete injury.³⁵ A subfailure injury to a ligament significantly alters its mechanical properties,³⁶ and many subfailure injuries have potential injury consequence.³⁷ Microscopic collagen fiber failure begins at 3% to 5% strain. Strain greater than 7% to 8% may result in the ligament undergoing plastic deformation and more exten-

Table 1. The average and SD of force at failure of cervical human spinal ligaments

	ALL	PLL	LF	JC	IS
C2-3	207 (98)	84(81)	86(61)	211 (130)	37(2)
C2-3	66 (37)	150(71)			
C3-4	47(14)	82 (66)	75(8)	224 (60)	33(2)
C3-4	104(99)	111 (49)			
C4-5	47(13)	47(11)	56(17)	170(20)	26 (24)
C4-5	106(61)	102(67)			
C5-6	89 (67)	85 (50)	89 (48)	144 (36)	33(15)
C5-6	104(54)	89 (42)			
C6-7	176(25)	102 (29)	160 (38)	277(147)	31(12)
C6-7	105(44)	95 (65)			

All measurements are in Newtons (N). 1 N » .225 pounds.

ALL, Anterior longitudinal ligament; PLL, posterior longitudinal ligament; LF, ligamentum flavum; JC, joint capsule; IS, interspinous ligament.^{27,28}

sive collagen failure. The load-carrying capacity may be lost, even when the ligaments appear macroscopically continuous.³⁸ The properties of ligaments vary in collagen mRNA levels and cytoskeletal assembly under normal conditions and in response to injury and healing capacities.^{39,40}

Muscles

Because the spine cannot support itself and depending on the sample tested, the amount of force to produce failure of the ligaments can be low. Muscles are needed for protection, and the deep extensor muscles may have a different response than superficial muscles.⁴¹ The deep segmental muscles are under nonvoluntary control. The bigger muscles that cross over multiple segments are under voluntary control. In sled acceleration testing, the average muscle response was first in the levator scapulae (73.2 ms, 15.2 SD) followed by the sternocleidomastoid (73.3 ms, 14.7 SD).⁴² Testing has indicated it takes approximately 200 ms to develop sufficient muscle force to limit motion⁴³; it would appear that the muscle reflex contraction would be too little and too late to restrict motion.⁴⁴ A quick shoulder elevation before sudden accelerations may prevent or reduce injury.⁴⁵ In animal testing, a threshold for stretch-induced muscle injury does exist, with micro-failure occurring at 16.5% strain.⁴⁶

Delayed onset muscle soreness has been shown to occur at submaximal levels.⁴⁷ Disequilibrium from whiplash injury may be caused by hypertonicity of the muscles, auto-nomic reflexes, and over-excitation of proprioceptors that affect the central nervous system.⁴⁸ As a result of disorganized neck proprioceptive activity, a whiplash injury can cause distortion of the posture control system.⁴⁹ Vestibular input and neck motor neurons are closely related,⁵⁰ as the central cervical nucleus relays information between upper cervical muscle afferents and neurons on the contralateral vestibular nuclei.⁵¹ The vestibular contribution in neck reflexes gives an advantage over stretch reflexes alone because the vestibulocollic reflex mediates an earlier response in normal subjects than in labyrinthine-defective subjects.⁵²⁻⁵³ In diagnosing and treating disequilibrium, functional examination of the motion segments of the upper cervical spine⁵⁴ and eye movement are justified.^{49,55,56}

Controlled Testing

In human testing procedures, the normal seated position with the occupant's head in close proximity to the head restraint is coincidentally the optimal position for occupant injury protection in rear-end impact, an intuitive fact demonstrated by the measured increase in injury levels for the few out-of-position tests that have been performed.⁵⁷ Transient symptoms have been produced in test subjects not affiliated with professional associations at 4.0 km/hour (2.5 mph).¹⁰ Minor, temporary symptoms have occurred in controlled testing, but chronic pain has not been reported. The selection process of test subjects in itself may be a discriminating factor in an effort to extrapolate controlled testing findings to the population as a whole.

In an experiment to simulate the trauma to the neck in rear-end impacts, anesthetized pigs were exposed to a swift extension-flexion motion of the cervical spine. This motion also produced an S-shaped phase shift of the cervical spine. Fluid pressure in the spinal canal was monitored, and nerve tissue was microscopically examined with findings of significant injuries in the lower cervical region. Injuries to the spinal ganglia were found, particularly in the lower cervical region.¹⁶ The dorsal root ganglion demonstrates mechanical sensitivity in its normal state, with some discharges occurring spontaneously. This baseline excitability is heightened after peripheral nerve injury, contributing ectopic barrages above and beyond those generated by the region of nerve injury.⁵⁸ With increasing age, there can be sprouting of sympathetic nerves in the dorsal root ganglion, which may be responsible for sympathetic pain generation or maintenance of pain.⁵⁹ The phase shift in the cervical spine, which occurs in low-speed collisions, causes a change in volume of the spinal canal between C2 and C7 motion, an increase in cerebral spinal pressure, and central venous pressure.^{60,61} This phase shift of the spine may also have effects in vertebral artery and somatic and autonomic nerve activity.^{62,63} Human studies have shown increases in the amplitudes of the H-reflex (a monosynaptic segmental reflex used to assess neuropathologic changes in the nerve root and spinal excitability) in all head position movement except flexion.⁶⁴

Symptoms

Anesthetic injections have been used to identify structures that may be the site of peripheral pain generators.⁶⁵ Patients with chronic neck pain had a symptomatic disk and a symptomatic zygapophyseal joint at the same segment in 41% of patients with posttraumatic neck pain. Disks alone were symptomatic in 20%, and zygapophyseal facet joints alone were symptomatic in 20% of the sample.⁶⁶ Studies have indicated that the cervical zygapophyseal (facet) joint may be a source of chronic symptoms after whiplash injuries.^{67,68} In addition to the S-shaped phase shift of the spine that has been found in controlled collision testing in cadavers and in human volunteers, an elevation of the instantaneous axis of rotation takes place in the lower cervical spine. This movement of the cervical spine is different from normal and may cause injury of the facet.¹⁴ Along with changes in the instan-

taneous axis of rotation, axial compression causes a loosening and a decrease in shear stiffness of the cervical ligaments, making it easier for shear type of soft-tissue injury.⁶⁹ The facet joint capsules are not homogenous, with the dorsal part being thin and the ventral part being thick and reinforced with oblique elastic fibers.⁷⁰ Injury to the facet capsular ligament is likely to affect segmental motion⁷¹ and therefore joint sensory input to the neuroaxis.

Not everyone injured has immediate symptoms. Delayed instability may occur,^{37,72} and approximately 22% of those involved in collisions do not have symptoms at the scene but develop them later.¹³ In the joints, there is both a peripheral source of nociceptive input and afferentation arising from the joint mechanoreceptors that decrease the intensity of the nociceptive impulses. In joint dysfunction, nociception that would normally be "gated out," that is, pain usually inhibited by mechanoreceptors (type-A fibers), may lead to deafferentation pain. Pain by this mechanism may occur immediately in one third, within a year in a second third, and after 1 year in the remainder. The pain in deafferentation is described as "burning, raw, or searing" or as a "tingling, numb sensation."⁷³ Activation of nitric oxide signal transduction contributes to the sensitization of wide dynamic range spinothalamic tract neurons in the deep dorsal horn causing simultaneous attenuation in inhibition produced in the periaqueductal gray.⁷⁴ Sensitization of the spinothalamic tract cells is, in part, produced by disinhibition.^{74,75}

The cause of injury, the patient's age, and the presence of head injury or focal neurologic deficit are important predictors of cervical spine fracture. In minor impact collisions, there is a low incidence of fracture.⁷⁶ Head impact increases the risk of fracture by a factor of 3.⁷⁷ The preferred screening modality in trauma patients at high and moderate risk for cervical spine fractures is computer tomography.⁷⁸ Routine use of magnetic resonance imaging is not justified⁷⁹ but may be of use in patients with persistent, radiating pain.⁸⁰ Videofluoroscopy can demonstrate different motion patterns between normal and pathologic spines.⁸¹ Bone scintigraphy may detect occult damage not visible on x-ray.⁸²

Pain

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." Nociceptive pain is the pain that results from activation of nociceptors, nerve fibers, and pathways associated with tissue damage. Pain has subjective elements and does not necessarily require tissue damage. There are technical differences between pain and nociception.⁸³

Activation of subthreshold neural pathways in controlled experimental conditions have been elicited by a puff of air.⁸⁴ After activation of a pathway caused by a stimulation, the nervous system processes this information. Stimulation of the peripheral mechanoreceptors, chemoreceptors, and nociceptors have effects on the central nervous system. Pain can be abolished, inhibited, or facilitated by the brain. Clinically, mechanical pain may be triggered from normal structure.⁸⁵

Pain and changed somatosensory thresholds may occur after relatively minor axonal damage and nerve sheath inflammation when no axonal damage is present.⁸⁶ Nociceptors are primary afferent neurons that respond to noxious or potentially tissue-damaging stimuli, and they can be sensitized. Inflammation increases the sensitivity of the receptors in the periphery and in the central nervous system. All C-fibers of the sensory system have beta-adrenergic receptors. Injury to a muscle or a joint can result in sensitization of peripheral nociceptors⁸⁷ that are associated with unmyelinated and small myelinated peripheral nerve fibers.

Peripheral inflammation results in an enlargement of the receptor fields of many neurons, which may result in pain that is experienced with less intense stimulation than normal and is called allodynia. Injury may also lead to increases in neuronal activity that are reflected in gene expression and prolonged changes in the nervous system. The functional result is hyperalgesia and spontaneous pain associated with tissue injury.⁸⁸

Tissue acidosis appears as a dominant factor in inflammatory pain.⁸⁹ Inflammation increases in the sensitivity in the peripheral terminals of A-delta and C-fibers, which can result in A-beta fibers that normally inhibit nociception to now express substance P.⁹⁰ Substance P in the dorsal horn appears to be controlled by N-methyl-D-aspartate receptors.⁹¹ Inflammation elevates nerve growth factor,⁹² and progressive tactile hyperalgesia induced by peripheral inflammation is nerve growth factor-dependent. Peripheral nerve injury can trigger central sprouting of myelinated afferents⁹³ and central sensitization in the spinal cord.⁹⁴ Cytokines released from an injury may be proinflammatory or antiinflammatory.⁹⁵

Chronic Pain

Chronic neck pain after whiplash injury is a reproducible phenomenon in need of an explanation.⁹⁶ The occurrence of chronic neck pain in benign chronic pain studies in adults varied from 2% to 40% of the population.⁹⁷ A common usage is 13% of the general population.⁹⁸ Chronic problems from whiplash-type injury have been stated to range from 0 in one study² to 86% in another study³ and at least 3%¹³ to 33%⁹⁹ in reviews.

Chronic pain can play a role in locomotor system dysfunction and its perpetuation.^{100,101} Patterns of normal proprioceptive input are profoundly distorted when articular nociception is incurred. This interferes with the precise continuous input necessary for coordinated normal patterns of motion, balance, and equilibrium.¹⁰² Muscle spindle output from the neck muscles is significantly altered when the bradykinin concentration is elevated. This may induce pain through supraspinal projections and at the same time cause disturbances in motor coordination and proprioception by altering the activity of the gamma-muscle spindle system.¹⁰³ Abnormal processing allows transmission of signals along the central nervous system nociceptive pathways, independent of the degree of nociception that is occurring in the periphery. It does not require months for the development of the sensory and affective meaning of persistent pain.¹⁰⁴ Deep pain is different than superficial pain because deep

pain lasts longer than superficial pain¹⁰⁵ and does not follow dermatomal patterns.^{106,107}

Mechanoreceptors and nociceptive nerve endings in the cervical facet capsules and cervical disks prove that these tissues are monitored by the central nervous system and imply that neural input from these structures is important to pain sensation and proprioception in the cervical spine.¹⁰⁸⁻¹⁰⁹ Symptoms may occur with deficient intrinsic pain inhibition mechanisms because a dynamic balance exists between excitatory and inhibitory synaptic input into the spinal dorsal horn that functions to prevent central sensitization. An increase in sensitivity resulting from peripheral tissue injury has both a peripheral and central component. The responses of deep dorsal horn neurons depend on the previous state of the neuron.⁷⁵ The midbrain periaqueductal gray, rostral ventromedial medulla and spinal cord are crucial components of endogenous pain-modulating pathways.^{110,112} Areas of the brain stem descending pathways are not only involved in modulation of spinal nociceptive neurons in response to transient stimuli, they are also active in modulating spinal nociceptive processes in developing persistent pain. These fine-tuning, descending modulating systems underlie the variability of perceived pain and hyperalgesia. The imbalance can be a mechanism in acute and chronic pain conditions.¹¹³ Descending noradrenergic and serotonergic pathways differentially modulate spinoparabrachial neurons in the superficial and deep dorsal horn in inhibiting nociceptive neurons. The descending serotonergic pathway is more effective in suppressing neuronal hyperexcitability in the deep dorsal horn.¹¹⁴ The medullary dorsal reticular nucleus plays a pronociceptive role in both acute and tonic inflammatory pain, leading to amplification of the nociceptive signal,¹¹⁵ and also may underlie the noxious response to the temporomandibular joint.¹¹⁶ Different mechanisms mediate dynamic and static mechanical hyperalgesia.¹¹⁷ In fibromyalgia and chronic whiplash, there is a state of central hyperexcitability in the nociceptive system.^{118,119}

In the subset of patients with persistence symptoms who are resistant to treatment,¹²⁰ central sensitization may have been induced by prior or ongoing nociceptive involvement of the central nervous system.¹²¹ Treatment at the peripheral source may not completely relieve the symptoms because sensitization of the nervous system can cause some excitatory influences that local treatment cannot suppress.¹²² Inhibition of C-fiber synaptic transmission can be achieved by A-delta stimulation¹²³ and with stimulation of afferents in the contralateral deep tissue by activating inhibitory descending projections from higher centers.¹²⁴ With age, there is a greater reduction in the density of myelinated A-delta fibers compared with unmyelinated C-type fibers.¹²⁵ In addition, physical activity can increase the nociceptive reflex threshold.¹²⁶

Brain Imaging

The cerebral cortex is involved in pain activity¹²⁷⁻¹²⁸ and with the side of pain, having a hemispheric effect.^{129,130} Imaging studies of the brain include functional magnetic res-

onance imaging with the signal generated by the differences in magnetic properties of oxygenated versus deoxygenated hemoglobin. The spatial resolution of functional magnetic resonance imaging is higher than positron emission tomography or single photon emission computed tomography. Positron emission tomography or single photon emission computed tomography technologies is used for measurements of molecular targets that bind selective molecules to tissue.¹³¹ In dynamic encoding of pain intensity, functional magnetic resonance imaging has demonstrated a distributed cortical system including the parietal areas and cingulate and frontal regions.^{132,134} Single photon emission computed tomography and positron emission tomography studies indicate brain activation patterns of nociceptive afferent nerves from the upper cervical spine in whiplash patients.^{135,136} Acute skin and muscle pain have similar and different cerebral activation patterns in forebrain structures.¹³⁷

For a person to have suffered a mild traumatic brain injury, a loss of consciousness is not required. An alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused) may be a sign of mild brain injury.¹³⁸ Animal testing has suggested that a person in a car that is accelerated to 5g would have a 50% chance of sustaining a concussion.¹² In crash testing, humans have been exposed to 10g without apparent effects.¹³⁹ In evaluating patients for soft-tissue injury, at least 2 different syndromes have occurred. The cervicoencephalic syndrome is characterized by headache, fatigue, dizziness, poor concentration, disturbed accommodation, and impaired adaptation to light intensity. The second syndrome, the lower cervical spine syndrome, is accompanied by cervical and cervicobrachial pain. Poorer results occur with those with attention deficit.¹⁴⁰ Of course, there may be a combination of these 2 syndromes.

CONCLUSION

An injury or noninjury cannot be deduced from the vehicle alone. Injury has been produced with in vitro ligament tensile tests with the force of less than the weight of the average head in volunteers with a speed change of 4 km/hour (2.5 mph), torso acceleration of 4.5g; a person in 5g acceleration may have a 50% chance of a concussion. With the possibility of 130,027,600 human postures,¹⁴¹ variability of tensile strength of the ligaments between individuals, body positions in the vehicle, the differences of collagen fibers in the same specimen segment, the amount of muscle activation and inhibition of muscles, the size of the spinal canals, and the excitability of the nervous system, one specific threshold is not possible. How individuals react to a stimulus varies widely, and it is clearly evident that peripheral stimulation has effects on the central nervous system. It is also clear that the somatosensory system of the neck, in addition to signaling nociception, may influence the control of neck, eyes, limbs, respiratory muscles, and some preganglionic sympathetic nerves.^{55-56,142,143} Evaluation of the individual patient should include more than a traditional orthopedic examination geared to find ablative lesions. Dysfunction may be apparent in muscles, joints, and motor, sensory, vestibular, and eye

function. The forces applied may be harmless to one person, may cause an injury in another, and may create a chronic condition in some. Individual evaluation is required.

REFERENCES

1. States JD, Balcerak JC, Williams, et al. Injury frequency and head restraint effectiveness in rear-end impact accidents. Presented at the 11th STAPP Car Crash Conference. Warrendale (PA): Society of Automotive Engineers; 1972.
2. Obelieniene D, Schrader H, Bovim G, Misevic I, Sand T. Pain after whiplash: a prospective controlled inception cohort study. *JNeuro Neurosurg Psychiatry* 1999;66:279-83.
3. Robinson DD, Cassar-Pullicini VN. Acute neck sprain after road traffic accident: a long term clinical and radiological review. *Injury* 1993;24:79-82.
4. Bailey MN, Wong BC, Lawrence JM. Data and methods for estimating the severity of minor impacts. Detroit (MI): Society of Automotive Engineers; 1995. 950352.
5. Navin FPD, Romilly DP. An investigation into vehicle and occupant response subjected to low-speed rear impacts. Proceedings of the Multidisciplinary Road Safety Conference. Fredericton, New Brunswick: National Science and Engineering Council; 1989.
6. Farmer H, Raymakers R. Neck injuries from rear impact road traffic accidents: prognosis in persons seeking compensation. *Injury* 1993;24:75-8.
7. Robbins MC. Lack of relationship between vehicle damage and occupant injury. Detroit (MI): Society of Automotive Engineers; 1997.
8. Hirsch SA, Hirsch PJ, Hiramoto H, Weiss A. Whiplash syndrome: fact or fiction? *Ortho Clin North Am* 1988; 19:791-5.
9. Sturzenegger M, DiStefano G, Radanov B, Schmidrig A. Presenting symptoms and signs after whiplash injury. *Neurology* 1994;44:688-93.
10. Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical response of human subjects to rear-end automobile collisions. *Arch Phys Med Rehabil* 1998;79:72-80.
11. Panjabi MM, Nibu K, Cholewicki J. Whiplash injuries and the potential for mechanical instability. *Eur Spine J* 1998;7:484-92.
12. Ommaya AK, Hirsch AE. Tolerances for cerebral concussion from head injury: impact and whiplash in primates. *J Biomechanics* 1971;4:13-21.
13. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, et al. Scientific monograph of the Quebec Task Force on whiplash-associated-disorders: redefining "whiplash" and its management. *Spine* 1995;20(Suppl):1S-73S.
14. Kaneoka K, Ono K, Inami S, Hayashi K. Motion analysis of cervical vertebrae during whiplash loading. *Spine* 1999;24:763-70.
15. Grauer JN, Panjabi MM, Cholewicki J, Nibu K, Dvorak J. Whiplash produces S-shaped curvatures of the neck with hyperextension at lower levels. *Spine* 1997;22:2489-94.
16. Ortengren T, Hansson HA, Lovsund P, Svensson MY, Suneson A, Saljo A. Membrane leakage in spinal ganglion nerve cells induced by experimental whiplash extension motion: a study in pigs. *JNeurotrauma* 1996;13:171-9.
17. Panjabi MM, Abumi K, Duranceau J, Oxland T. Spinal stability and intersegmental muscle forces: a biomechanical model. *Spine* 1989;14:194-200.
18. Panjabi MM, Cholewicki J, Nibu K, Grauer J, Babat L, Dvorak J. Critical load of the human cervical spine: an in vitro experimental study. *Clin Biomech* 1998;13:11-7.
19. Pope MH, Panjabi M. Biomechanical definitions of spinal instability. *Spine* 1984;9:255-6.
20. Panjabi MM, Lydon C, Vasavada A, Grob D, Crisco II, Dvorak J. On the understanding of clinical instability. *Spine* 1994; 19:264-50.
21. Oxland TR, Panjabi MM. The onset and progression of spinal injury: a demonstration of neutral zone sensitivity. *J Biomech* 1992;25:1165-72.

22. Crawford NR, Peles JD, Dickman CA. The spinal lax zone and neutral zone: measurement techniques and parameter comparisons. *J Spine Disord* 1998;1:1416-29.
23. Mertz HJ, Patrick LM. Strength and response of the human neck. 15th Stapp Car Crash Conference. San Diego (CA): Society of Automotive Engineers; 1971.
24. SAE Vehicle Occupant Restraint Systems and Components Standards Manual. 1993, Society of Automotive Engineers, SAEHS-13.
25. Heller JG, Amrani J, Button WC. Transverse ligament failure: a biomechanical study. *J Spinal Disord* 1993;6:162-5.
26. Spence KF, Decker S, Sell KW. Bursting atlantal fracture associated with rupture of the transverse ligament. *J Bone Joint Surg Am* 1970;52:543-9.
27. Mykelbust IB, Pintar F, Yoganandan N, Cusick JF, Maiman D, Myers TJ, et al. Tensile strength of spinal ligaments. *Spine* 1988;13:526-31.
28. Przybylski GJ, Carlin GJ, Patel PR, Woo SLY. Human anterior and posterior cervical longitudinal ligaments possess similar tensile properties. *J Orthop Res* 1996;14:1005-8.
29. Richmond FRJ, Singh K, Corniel BD. Marked non-uniformity of fiber-type composition in the primate suboccipital muscle obliquus capitis inferior. *Exp Brain Res* 1999;125:14-8.
30. Ross JK, Bereznic DE, McGill SM. Atlas-axis facet asymmetry: implications in manual palpation. *Spine* 1999;24:1203-9.
31. Abumi K, Fujiya M, Saita M, Kaneda K. Occipitoatlantal instability associated with articular tropism. *Eur Spine J* 1998;7:76-9.
32. Mercer S, Bogduk N. The ligaments and anulus fibrosus of the human adult intervertebral discs. *Spine* 1999;24:619-28.
33. Shea M, Wittenberg RH, Edwards WT, White AA, Hayes WC. In vitro hyperextension injuries in the human cadaveric cervical spine. *J Orthop Res* 1992; 10:911-6.
34. Bland JH. New anatomy and physiology with clinical and historical implications. In: Bland JH, editor. *Disorders of the cervical spine: diagnosis and medical management*. 2nd ed. Philadelphia: W.B. Saunders; 1994. p. 72-3.
35. Panjabi MM, Cholewicki J, Nibu K, Babat LB, Dvorak J. Simulation of whiplash trauma using whole cervical spine specimens. *Spine* 1998;23:17-24.
36. Panjabi MM, Yoldas E, Oxland TR, Crisco JJ. Subfailure injury of the rabbit ACL. *J Orthop Res* 1996; 14:216-22.
37. Herkowitz HN, Rothman RH. Subacute instability of the cervical spine. *Spine* 1984;9:348-57.
38. Noyes FR, Keller CS, Grood ES, Butler DL. Advances in the understanding of knee ligament injury, repair, and rehabilitation. *Med Sci Sports Exer* 1984;16:427-43.
39. Wiig ME, Amiel D, Ivarsson M, Nagineni CN, Wallace CD, Arfors KE. Type I procollagen gene expression in normal and early healing of the medial collateral and anterior cruciate ligaments in rabbits: an in situ hybridization study. *J Orthop Res* 1991;9:374-82.
40. Sung KLP, Yang L, Whittemore DE, Shi Y, Jin Y, Heich AH, et al. The differential adhesion forces of anterior cruciate and medial collateral ligament fibroblasts: effects of tropomodulin, talin, vinculin, and α -actinin. *Proc Natl Acad Sci U S A* 1998;93:9182-7.
41. Kunita K, Fujiwara K. Relationship between reaction time of eye movement and activity of the neck extensors. *Eur J Appl Physiol* 1996;74:553-7.
42. Magnusson ML, Pope MH, Hasselquist L, Bolte KM, Ross M, Goel VK, et al. Cervical electromyographic activity during low-speed rear impact. *Eur Spine J* 1999;8:118-25.
43. Tennyson SA, Mital NK, King AL. Electromyographic signals of the spinal musculature during +Gz impact acceleration. *Orthop Clin North Am* 1977;8:97-119.
44. Foust DR, Chaffin DB, Snyder RG, Baum JK. Cervical range of motion and dynamic response and strength of cervical muscles. 17th Stapp car crash conference. Oklahoma City (OK): Society of Automotive Engineers; 1973.
45. Pope MH, Aleksiev A, Hasselquist L, Magnusson ML, Spratt K, Szpalski M. Neurophysical mechanisms of low-velocity non-head-contact cervical acceleration. In: Gunzburg R, Szpalski M, editors. *Whiplash injuries: current concepts in prevention, diagnosis, and treatment of the cervical whiplash syndrome*. Philadelphia (PA): Lippincott-Raven; 1998. p. 89-93.
46. Sun JS, Hang YS, Tsuang YH, Cheng CK, Tsao KY, Hsu SH. Morphological changes of the triceps surae muscle-tendon unit during passive extension: an in vivo rabbit model. *Clin Biomech* 1998;13:634-40.
47. Evans OFF, Haller RG, Wyrick PS, Parkey RW, Fleckenstein JL. Submaximal delayed-onset muscle soreness: correlations between MR imaging findings and clinical measures. *Radiographics* 1998;208:815-20.
48. Hinoki M. Vertigo due to whiplash injury: a neurotological approach. *Acta Otolaryngol (Stockh)* 1985;419:9-29.
49. Gimse R, Tjell C, Bjorgen IA, Saunte C. Disturbed eye movement after whiplash due to injuries the posture control system. *J Clin Exp Neuropsychol* 1996; 18:178-86.
50. Shinoda Y, Sugiuchi Y, Futami T, Ando N, Yagi J. Input patterns and pathways from six semicircular canals to motoneurons of neck muscles. II The longissimus and semispinalis muscle groups. *J Physiol* 1997;77:1234-53.
51. Sato H, Ohkawa T, Uchino Y, Wilson VJ. Excitatory connections between neurons of the central cervical nucleus and vestibular neurons in the cat. *Exp Brain Res* 1997;115:381-6.
52. Ito Y, Corna S, von Brevern M, Bronstein A, Gresty M. The functional effectiveness of neck reflexes for head-righting in response to sudden fall. *Exp Brain Res* 1997;117:266-72.
53. Ito Y, Corna S, von Breven M, Bronstein A, Rothwell J, Gresty M. Neck muscle responses to abrupt free fall of the head: comparison of normal with labyrinthine-defective human subjects. *J Physiol (Lond)* 1995;489:911-6.
54. Galm R, Rittmeister M, Schmitt E. Vertigo in patients with cervical spine dysfunction. *Eur Spine J* 1998;7:55-8.
55. Fischer A JEM, Huygen PLM, Folgering HT, Verhagen WIM, Theunissen EJJM. Hyperactive VOR and hyperventilation after whiplash injury. *Acta Otolaryngol (Stockh)* 1995;520(Suppl): 49-52.
56. Heikkila HV, Wenngren BI. Cervicocephalic kinesthetic sensibility, active range of cervical motion, and oculomotor function in patients with whiplash injury. *Arch Phys Med Rehabil* 1998;79:1089-94.
57. Benson BR, Smith GC, Kent RW, Monson CR. Effectiveness of seat stiffness in out-of-position occupant response in rear-end collisions. Warrendale (PA): Society of Automotive Engineers; 1996.
58. LaRocca H. A taxonomy of chronic pain syndromes. *Spine* 1992;17:S344-55.
59. Ramer MS, Bisby MA. Normal and injury-induced sympathetic innervation of rat dorsal root ganglia increases with age. *J Comp Neurol* 1998;394:38-47.
60. Holmes A, Han ZH, Dang GT, Chen ZQ, Wang ZG, Fang J. Changes in cervical canal spinal volume during in vitro flexion-extension. *Spine* 1996;21:1313-9.
61. Dinsmore J, Bacon RC, Hollway TE. The effect of increasing degrees of spinal flexion on cerebrospinal fluid pressure. *Anesthesia* 1998;53:431-4.
62. Nibu K, Cholewicki J, Panjabi MM, Babat LB, Grauer JN, Kothe R, et al. Dynamic elongation of the vertebral artery during an in vitro whiplash simulation. *Eur Spine J* 1997;6:286-9.
63. Bogduk N, Windsor M, Inglis A. The innervation of the cervical intervertebral discs. *Spine* 1988;13:2-8.
64. Sabbahi M, Abdulwahab S. Cervical root compression monitoring by flexor carpi radialis H-reflex in healthy subjects. *Spine* 1999;24:137-41.
65. Bamsley L, Bogduk N. Medial branch blocks are specific for the diagnosis of cervical zygapophyseal joint pain. *Reg Anesth Pain Med* 1993;18:343-50.

66. Bogduk N, April C. On the nature of neck pain, discography and cervical zygapophysial joint blocks. *Pain* 1993;54:213-7.
67. Barnsley L, Lord SM, Wallis BJ, Bogduk N. The prevalence of chronic cervical zygapophysial joint pain after whiplash. *Spine* 1995;20:20-6.
68. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo controlled prevalence study. *Spine* 1996;12:1737-45.
69. Yang KH, Bergeman PC, Muser M, Niederer P, Walz F. On the role of cervical facet joints in rear end impact neck injury mechanisms. Detroit (MI): Society of Automotive Engineers; 1997.970497.
70. Tonetti J, Peoc'h M, Merloz P, Pasquier B, Chirossel JP. Elastic reinforcement and thickness of the joint capsules of the lower cervical spine. *Surg Radiol Anat* 1999;21:35-9.
71. Goel VK, Clark CR, McGowan D, Goyal S. An in-vitro study of the kinematics of the normal, injured and stabilized cervical spine. *J Biomech* 1984;17:363-76.
72. Delfini R, Dorizzi A, Facchinetti G, Faccioli F, Galzio R, Vangelista T. Delayed post-traumatic cervical instability. *Surg Neurol* 1999;51:588-95.
73. Tasker RR, Dostrovsky JO. Deafferentation and central pain. In: Wall PD, Melzack R, editors. *Textbook of pain*. Edinburgh: Churchill Livingstone; 1989. p. 154-80.
74. Lin Q, Peng YB, Wu J, Willis WD. Involvement of cGMP in nociceptive processing by and sensitization of spinothalamic neurons in primates. *J Neurosci* 1997;17:3293-302.
75. Traub RJ. Spinal modulation of the induction of central sensitization. *Brain Res* 1997;778:34-42.
76. Blackmore CC, Emerson SS, Mann FA, Koepsell TD. Cervical spine imaging in patients with trauma: determination of fracture risk to optimize use. *Radiology* 1999;211:759-65.
77. Foret-Bruno JY, Tariere C, Le Coz JY, Got C, Guillon F. Risk of cervical lesions in real-world and simulated collisions. 34th Annual Proceedings of the Association for the Advancement of Automobile Medicine; 1990 October 1-3; Scottsdale, AZ.
78. Blackmore CC, Ramsey SD, Mann FA, Deyo RA. Cervical spine screening with CT in trauma patients: a cost-effectiveness analysis. *Radiology* 1999;212:117-25.
79. Voyvodic F, Dolinis J, Moore VM. MRI of car occupants with whiplash injury. *Neuroradiology* 1997;39:35-40.
80. Jonsson H, Cesarini K, Sahlstedt B, Rauschnig W. Findings and outcomes in whiplash-type neck distortions. *Spine* 1994;19:2733-43.
81. Hino H, Abumi K, Kanayama M, Kaneda K. Dynamic motion analysis of normal and unstable cervical spines using cineradiography. *Spine* 1999;24:163-8.
82. Versijpt J, Dierckx RA, De Bondt P, Dierckx I, Lambrecht L, De Sadeleer C. The contribution of bone scintigraphy in occupational health or medical insurance claims: a retrospective study. *Eur J Nucl Med* 1999;26:804-11.
83. Merskey H, Bogduk N, editors. *Classification of chronic pain*. 2nd ed. Seattle: IASP Press; 1994.
84. Swadlow HA, Hicks TP. Subthreshold receptive fields and baseline excitability of "silent" SI callosal neurons in awake rabbits: contributions of AMPA/kinate and NMDA receptors. *Exp Brain Res* 1997;115:403-9.
85. Shacklock MO. Central pain mechanisms: a new horizon in manual therapy. *Austr J Physiother* 1999;45:83-92.
86. Greening J, Lynn B. Minor peripheral nerve injuries: an underestimated source of pain? *Man Ther* 1998;3:187-94.
87. Sluka KA. Pain mechanisms involved in musculoskeletal disorders. *J Orthop Sports Phys Ther* 1996;24:240-54.
88. Dubner R, Ruda MA. Activity-dependent neuronal plasticity following tissue injury and inflammation. *TINS* 1992; 15:96-103.
89. Steen KH, Steen AE, Kreysel HW, Reeh PW. Inflammatory mediators potentiate pain induced by experimental tissue acidosis. *Pain* 1996;66:163-70.
90. Neumann S, Doubell TP, Leslie T, Woolf CJ. Inflammatory pain hypersensitivity mediated by phenotypic switch in myelinated primary sensory neurons. *Nature* 1996;384:360-4.
91. Marvizon JCG, Martinez V, Grady EF, Bunnett K.W, Mayer EA. Neurokinin 1 receptor interbalization in spinal cord slices induced by dorsal root stimulation is mediated by NMDA receptors. *J Neurosci* 1997; 12:8129-36.
92. Ma AP, Woolf CJ. The progressive tactile hyperalgesia induced by peripheral inflammation is nerve growth factor dependent. *Neuroreport* 1997;8:807-10.
93. Woolf CJ, Shortland P, Coggeshall RE. Peripheral nerve injury triggers central sprouting of myelinated afferents. *Nature* 1992;355:75-7.
94. Wu J, Lin Q, McAdoo DJ, Willis WD. Nitric oxide contributes to central sensitization following intradermal injection of capsaicin. *Neuroreport* 1998;9:589-92.
95. Cunha FQ, Poole S, Lorenzetti BB, Veiga FH, Ferreira SH. Cytokine-mediated inflammatory hyperalgesia limited by interleukin-4. *Br J Pharmacol* 1999;126:45-50.
96. Wallis BJ, Lord SM, Bogduk N. Pain and psychological symptoms of Australian patients with whiplash [letter]. *Spine* 1997;21:114-5.
97. Verhaak PFM, Kerssens JJ, Dekker J, Sorbi MJ, Sensing JM. Prevalence of chronic benign pain disorder among adults: a review of the literature. *Pain* 1998;77:231-9.
98. Bovin G, Schrader H, Sand T. Neck pain in the general population. *Spine* 1994; 19:1307-9.
99. Freeman MD, Croft AC, Rossignol AM, Weaver DS, Reiser M. A review and methodologic critique of the literature refuting whiplash syndrome. *Spine* 1999;24:86-98.
100. Matre DA, Sinkaer T, Svensson P, Arendt-Nielsen L. Experimental muscle pain increases the human stretch reflex. *Pain* 1998;75:331-39.
101. Sheather-Reid RB, Cohen ML. Psychophysical evidence for a neuropathic component of chronic neck pain. *Pain* 1998; 75:341-7.
102. Wyke BD. Articular neurology and manipulative therapy. In: Glasgow EF, Twomey LT, Sell ER, Idezak RM, editors. *Aspects of manipulative therapy*. Melbourne: Churchill Livingstone; 1985. p. 72-7.
103. Wenngren BI, Pedersen J, Sjolander P, Bergenheim M, Johansson H. Bradykinin and muscle stretch alter contralateral cat neck muscle spindle output. *Neuroscience Res* 1998;32:119-29.
104. Stohler CS, Kowalski CJ. Spatial and temporal summation of sensory and affective dimensions of deep somatic pain. *Pain* 1999;79:165-73.
105. Woolf CJ, Wall PD. Relative effectiveness of C primary afferent fibers of different origins in evoking a prolonged facilitation of the flexor reflex in the rat. *J Neurosci* 1986; 6:1433-42.
106. Kellgren JH. On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. *Clin Sci(Colch)* 1939;4:35-46.
107. Feinstein B, Langton JNK, Jameson RM, Schiller F. Experiments on pain referred from deep somatic tissues. *J Bone Joint Surg Am* 1954;36:981-97.
108. McLain RF. Mechanoreceptor endings in the human cervical facet joints. *Spine* 1994; 19:495-501.
109. Mendel T, Wink CS, Zimny ML. Neural elements in human cervical intervertebral discs. *Spine* 1992; 17:132-5.
110. Gutstein HB, Mansour A, Watson SJ, Akil H, Fields H. Mu and kappa opioid receptors in periaqueductal gray and rostral ventromedial medulla. *Neuroreport* 1998;9:1777-81.
111. Hammond DL, Wang H, Nakashima N, Basbaum AI. Differential effects of intrathecally administered delta and mu opioid receptor agonists on formalin-evoked nociception and on the expression of Fos-like immunoreactivity in the spinal cord of the rat. *J Pharmacol Exp Ther* 1998;284:378-87.

112. Bellgowan PSF, Helmstetter FJ. The role of mu and kappa opioid receptors within the periaqueductal gray in the expression of conditional hypoalgesia. *Brain Res* 1998;791:83-9.
113. Wei F, Dubner R, Ren K. Nucleus reticularis gigantocellularis and nucleus raphe magnus in the brain stem exert opposite effects on behavioral hyperalgesia and spinal Fos protein expression after peripheral inflammation. *Pain* 1999;80:127-41.
114. Wei F, Dubner R, Ren K. Laminar-selective noradrenergic and serotonergic modulation includes spinoparabrachial cells after inflammation. *Neuroreport* 1999;10:1757-61.
115. Almeida A, Storkson R, Lima D, Hole K, Tjolsen A. The medullary dorsal reticular nucleus facilitates pain behaviour induced by formalin in the rat. *Eur J Neurosci* 1999; 11:110-22.
116. Tsai C. The caudal subnucleus caudalis (medullary dorsal horn) acts as interneuronal relay site in craniofacial nociceptive reflex activity. *Brain Res* 1999;826:293-7.
117. Gottrup H, Nielsen J, Arendt-Nielsen L, Jensen TS. The relationship between sensory thresholds and mechanical hyperalgesia in nerve injury. *Pain* 1998;75:321-9.
118. Sorensen J, Graven-Nielsen T, Henriksson KG, Bengtsson M, Arendt-Nielsen L. Hyperexcitability in fibromyalgia. *J Rheumatol* 1998;25:152-5.
119. Koelbaek Johansen M, Graven-Nielsen T, Schou Olesen A, Arendt-Nielsen L. Generalised muscular hyperalgesia in chronic whiplash syndrome. *Pain* 1999;83:229-34.
120. Khan S, Cook J, Gargan M, Bannister G. A symptomatic classification of whiplash injury and the implications for treatment. *J Orthop Med* 1999;21:22-5.
121. Kramis RC, Roberts WJ, Gillette RG. Non-nociceptive aspects of persistent musculoskeletal pain. *J Orthop Sports PhysTher* 1996;24:255-67.
122. Kayser V, Idanpaan-Heikkila JJ, Guilbaud G. Sensitization of the nervous system, induced by two successive hindpaw inflammations is suppressed by a local anesthetic. *Brain Res* 1998;794:19-27.
123. Liu XG, Morton CR, Azkue JJ, Zimmermann M, Sandkuhler J. Long-term depression of C-fibre-evoked spinal field potentials by stimulation of primary afferent A fibres in the adult rat. *Eur J Neurosci* 1998;10:3069-75.
124. Gjerstad J, Tjolsen A, Svendsen F, Hole K. Inhibition of evoked C-fibre responses in the dorsal horn after contralateral injection of capsaicin involves activation of descending pathways. *Pain* 1999;80:413-8.
125. Chakour MC, Gibson SJ, Bradbeer M, Hlme RD. The effect of age on A delta- and C-fibre thermal pain perception. *Pain* 1996;64:143-52.
126. Guieu R, Blin O, Pouget J, Serratrice G. Nociceptive threshold and physical activity. *Can J Neurol Sci* 1992; 19:69-71.
127. Bushnell MC, Duncan GH, Hofbauer RK, Ha B, Chen JJ, Carrier B. Pain perception: is there a role for primary somatosensory cortex? *Proc Natl Acad Sci U S A* 1999;96:7705-9.
128. Ploghaus A, Tracey I, Gati JS, Clare S, Menon RS, Matthews PM, et al. Dissociating pain from its anticipation in the human brain. *Science* 1999;284:1979-81.
129. Pauli P, Wiedemann G, Nickola M. Pain sensitivity, cerebral laterality, and negative affect. *Pain* 1999;80:359-64.
130. Gagliese L, Schiff BB, Taylor A. Differential consequences of left- and right-sided chronic pain. *Clin JPain* 1995;11:201-7.
131. Volkow ND, Rosen B, Farde L. Imaging the living human brain: magnetic resonance imaging and positron emission tomography. *Proc Natl Acad Sci U S A* 1997;94:2787-8.
132. Davis KD, Taylor SJ, Crawley AP, Wood ML, Mikulis DJ. Functional MRI of pain- and attention-related activations in the human cingulate cortex. *J Neurophysiol* 1997;77:3370-80.
133. Porro CA, Cettolo V, Francescato MP, Baraldi P. Temporal and intensity coding of pain in human cortex. *J Neurophysiol* 1998;80:3312-20.
134. Rainville P, Duncan GH, Price DD, Carrier B, Bushnell C. Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science* 1997;277:968-71.
135. Otte A, Ettl TM, Nitzsche EU, Wachter K, Hoegerle S, Simon GH, et al. PET and SPECT in whiplash syndrome: a new approach to a forgotten brain? *J Neurol Neurosurg Psychiatry* 1997;63:368-72.
136. Radanov BP, Bicik I, Dvorak J, Antinnes J, von Schulthess GK, Buck A. Relation between neuropsychological and neuroimaging findings in patients with late whiplash syndrome. *J Neurol Neurosurg Psychiatry* 1999;66:485-9.
137. Svensson P, Minoshima S, Beydoun A, Morrow TJ, Casey KL. Cerebral processing of acute skin and muscle pain in humans. *J Neurophysiol* 1997;78:450-60.
138. Mild Traumatic Brain Injury Committee of the Head Injury Special Interest Group of the American Congress of Rehabilitation Medicine. Definition of mild traumatic brain injury. *J Head Trauma Rehabil* 1993;8:86-7.
139. Szabo TJ, Welcher JB. Human subject kinematics and electromyographic activity during low speed rear impacts. Warren-dale (PA): Society of Automotive Engineers; 1996.
140. Radanov BP, Dvorak J, Valach L. Cognitive deficits in patient after soft tissue injury of the cervical spine. *Spine* 1992; 17:127-31.
141. Harrison DD. Abnormal human posture: permutations of the rotations and translations of the skull, thorax, and pelvis in three dimensions. In: Harrison DD, editor. *Spinal biomechanics: a chiropractic perspective*. Harvest (AL): Chiropractic Biophysics; 1992. p. 43-59.
142. Bolton PS. The somatosensory system of the neck and its effects on the central nervous system. *J Manipulative Physiol Ther* 1998;21:553-63.
143. Verhagen AP, Lanser K, de Bie RA, de Vet HC. Whiplash: assessing the validity of diagnostic test in a cervical sensory disturbance. *J Manipulative Physiol Ther* 1996;19:508-12.