Cervical neural space narrowing during simulated rear crashes with anti-whiplash systems

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Abstract

Purpose Chronic radicular symptoms have been documented in whiplash patients, potentially caused by cervical neural tissue compression during an automobile rear crash. Our goals were to determine neural space narrowing of the lower cervical spine during simulated rear crashes with whiplash protection system (WHIPS) and active head restraint (AHR) and to compare these data to those obtained with no head restraint (NHR). We extrapolated our results to determine the potential for cord, ganglion, and nerve root compression.

Methods Our model, consisting of a human neck specimen within a BioRID II crash dummy, was subjected to simulated rear crashes in a WHIPS seat ($n = 6$, peak 12.0 g and ΔV 11.4 kph) or AHR seat and subsequently with NHR ($n = 6$, peak 11.0 g and ΔV 10.2 kph with AHR; peak 11.5 g and ΔV 10.7 kph with NHR). Cervical canal and foraminal narrowing were computed and average peak values statistically compared ($P < 0.05$) between WHIPS, AHR, and NHR.

Results Average peak canal and foramen narrowing could not be statistically differentiated between WHIPS, AHR, or NHR. Peak narrowing with WHIPS or AHR was 2.7 mm for canal diameter and 1.6 mm, 2.7 mm, and 5.9 mm² for foraminal width, height and area, respectively.

Conclusions While lower cervical spine cord compression during a rear crash is unlikely in those with normal canal diameters, our results demonstrated foraminal

kinematics sufficient to compress spinal ganglia and nerve roots. Future anti-whiplash systems designed to reduce cervical neural space narrowing may lead to reduced radicular symptoms in whiplash patients.

Keywords Whiplash · Cervical spine · Biomechanics · Spinal cord · Intervertebral foramen

Introduction

Rear automobile crashes may injure cervical neural tissues leading to clinically documented radicular symptoms of muscle weakness and paresthesias of the neck, shoulders, upper back, and arms in whiplash patients [1]. Sensory hypersensitivity and hypoesthesia have been observed in both chronic whiplash and radiculopathy patients, which may indicate similar injury mechanisms [2].

Radiculopathy has been associated with cervical nerve root and ganglion compression injury due to foraminal spondylosis, most commonly caused by uncovertebral osteophytes in the intervertebral foramen [3]. Cervical cord injury without vertebral fracture resulting in the central cord syndrome is most often caused by traumatic neck extension with greater injury severity in those with cervical stenosis [4]. Intervertebral extension and posterior translation cause reduction in the cervical foraminal area and canal diameter and volume, which may exacerbate pain in those with cervical stenosis [5, 6]. These motions may also cause dynamic neural tissue compression during whiplash. An imaging study of cervical specimens demonstrated a 12% decrease in the C5/6 foraminal area with each 1-mm incremental posterior translation of C5 relative to C6 [7]. Another cadaveric study demonstrated that physiologic extension can decrease the cervical foraminal area by as

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much as 20% [8]. Rear crashes simulated using osteoligamentous neck specimens have demonstrated nonphysiologic intervertebral extension and posterior translation of the lower cervical spine [9–11]. These motions may potentially cause transient neural tissue compression leading to injury and chronic radicular symptoms in whiplash patients.

Biomechanical studies have evaluated the potential for transient cervical neural tissue compression during simulated rear crashes with no head restraint or injury prevention system. Abrupt head extension and posterior shear were applied to an *in vivo* porcine model and the observed spinal fluid pressure increases were hypothesized to cause cervical spinal ganglia injury, documented histologically [12]. Other studies have determined neural tissue injury potential due to simulated rear crashes of human head–neck specimens. Using custom transducers to determine spinal canal and foraminal area narrowing, the greatest foraminal area reductions occurred at C5/6 and C6/7, but were of insufficient magnitude to cause injury [13]. Another study found that spinal cord injury during rear impact was unlikely in subjects with normal cervical canal diameters; however, those with severe cervical spinal canal stenosis may be at risk [14]. Nonphysiologic foraminal width narrowing was observed at the lower cervical spine, which indicated potential ganglia compression injury [15].

The goals of this study were to determine neural space narrowing of the lower cervical spine during simulated rear crashes with two distinct anti-whiplash systems, whiplash protection system (WHIPS) and active head restraint (AHR), and to compare these data to those obtained with no head restraint (NHR). We also extrapolated the present results to determine the potential for cord, ganglion, and nerve root compression injuries in individuals with and without cervical stenosis.

Materials and methods

Specimen preparation

Twelve fresh-frozen human osteoligamentous whole cervical spine specimens (occiput–T1) were mounted in resin at the occiput and T1 in neutral posture. Six of the specimens were prepared for rear crashes using WHIPS (2 M, 4 F; average age 86 years; range 79–90 years), while the other six were prepared for rear crashes using AHR and subsequently with NHR (4 M, 2 F; average age 83 years, range 79–90 years). Apart from typical age-related degenerative changes, the specimens did not suffer from any disease that could have affected the osteoligamentous structures. Lightweight motion-tracking flags with 9.5-mm diameter markers were rigidly attached to the anterior

aspect of each cervical vertebra. The average weight of each vertebral flag, including marker mass, was 14.5 g.

Simulated rear crashes

The human model of the neck (Fig. 1), described in detail elsewhere [10], consisted of the neck specimen with its T1 mount rigidly connected to the torso of a rear impact dummy, BioRID II (Humanetics Innovative Solutions, Plymouth, MI, USA), and carrying a custom anthropometric surrogate head (4.6 kg mass; 0.0214 kg m² sagittal moment of inertia). The head and neck were stabilized using the compressive muscle force replication system, which provides static postural neck stability and passive resistance to intervertebral motions during rear impact and produces a high-speed kinematic response similar to *in vivo* data [16]. The simulated muscle forces did not model dynamic muscle activation. The muscle force replication was used to maintain the cervical spine posture immediately prior to the crash. The cervical posture with AHR was consistent with *in vivo* neutral neck alignment [17], while with WHIPS it consisted of anterior head translation relative to the *in vivo* neutral posture, thus representing an out-of-position forward posture. The average initial gap between the head and head restraint was 3.5 cm (SD 0.5 cm; range 2.5–3.8 cm) with AHR and 11.9 cm (SD 2.1 cm; range 8.9–15.2 cm) with WHIPS.

Rear crashes were simulated at average maximum measured horizontal sled accelerations of 12.0 g (ΔV 11.4 kph) with WHIPS, 11.0 g (ΔV 10.2 kph) with AHR, and 11.5 g (ΔV 10.7 kph) with NHR. The crash apparatus consisted of an automobile seat mounted on a custom sled in which the model was seated in normal driving posture and secured with a seatbelt (Fig. 1). The WHIPS seat was from a 2005 Volvo XC90 minivan (Volvo Car Corporation, Göteborg, Sweden), while the AHR seat was from a 2006 Kia Sedona minivan (Kia Motors America, Inc, Irvine, CA, USA). The AHR was initially set to midrange gap and height. The crashes with NHR were simulated using the Kia seat with the AHR removed. WHIPS and AHR were activated by torso momentum pressing into the seatback during the crash. WHIPS motions consisted of rearward translation and rotation of the seatback relative to the seat base and plastic deformation of the bilateral WHIPS energy-absorbing components (Fig. 1a). The energy-absorbing components were replaced prior to each subsequent crash (Service Kit, Part #31250443, Volvo Car Corporation, Göteborg, Sweden). The AHR rotated forward via a pivoting mechanism between it and the seatback (Fig. 1b).

A high-speed digital camera recorded the sagittal motions of the flag markers at 500 frames/s (Motion Pro, Redlake, MSAD Inc., San Diego, CA). A contact switch mounted to the back of the head determined the time of

Fig. 1 Photographs of the human model of the neck and schematic drawings of **a** whiplash protection system (WHIPS) and **b** active head restraint (AHR). WHIPS and AHR were activated by torso momentum pressing into the seatback during the crash. WHIPS motions consisted of rearward translation and extension of the seatback relative to the seat base and plastic deformation of bilateral WHIPS

energy-absorbing components. The AHR rotated forward via a pivoting mechanism between it and the seatback. Motion tracking flags were fixed to the cervical vertebrae, while an accelerometer was fixed to the sled. Rotation was defined as positive for flexion and negative for extension

contact of the head with the head restraint. A bi-axial accelerometer (50 g capacity; part no. ADXL250JQC, Analog Devices, Norwood, MA, USA) fixed to the sled was continuously sampled at 1 kHz using an analog-to-digital converter and a personal computer. A braking system consisting of air cylinders in parallel gradually decelerated the model following the crash.

Spinal canal and intervertebral foramen geometry and kinematics

The coordinates of the vertebral flag markers were computed with sub-pixel accuracy in the global coordinate system using custom Matlab programs (Matlab, MathWorks, Natick, MA, USA). A radiograph of the lower cervical spine, C4 through C7, which most closely matched average vertebral dimensions [18, 19] and clearly showed anatomical landmarks, was scaled and aligned accordingly and digitally superimposed on the first frame of the high-speed movie (Adobe Photoshop CS2, Version 9, San Jose, CA). Two spinal canal points (Fig. 2a) and six foraminal

points (Fig. 2b) were mathematically reconstructed for each functional spinal unit, C4/5 through C6/7, using the radiograph and average quantitative cervical anatomy [20]. The canal diameter was defined as the sagittal distance between the posteroinferior corner of the upper vertebral body and the superior end of the spinolaminar line of the lower vertebra. Points at the anterior, middle, and posterior regions of the foraminal medial zone were used to define three foramen parameters of width, height, and area. The width was the distance between the anterior medial zone of the superior vertebra and the posterior medial zone of the inferior vertebra, the height was the distance between the middle medial zone of adjacent vertebrae, and the area was the total area encompassed by the six foraminal points. We established geometrical rigid body relationships between the flag markers and the canal and foraminal points. For each subsequent movie frame, canal and foramen narrowing and widening were computed using as input vertebral rotation data, flag marker translations, and the geometric rigid body relationships. For subsequent evaluation of the potential for nerve root and ganglia compression during the

crashes, the sagittal foraminal width and area narrowing were transformed to determine the width and area narrowing at 45° to the midsagittal plane (Fig. 2b). No transformations of the foraminal height or canal narrowing were performed. The average (SD) system errors, as determined in separate studies, were 0.3 mm (0.2 mm) for canal and foramen narrowing and widening [21] and 0.00° (0.34°) for vertebral rotation [10].

Fig. 2 Schematic drawings of spinal canal and intervertebral foramen points used to determine narrowing and widening during the rear crashes. **a** Sagittal view of the spinal canal. The spinal canal diameter was defined as the distance from the posteroinferior corner of the upper vertebral body to the superior end of the spinolaminar line of the lower vertebra. **b** Superior view of cervical foraminal medial zone, ganglia, and nerve roots. The foraminal width, height, and area were defined by six foraminal points at the anterior, middle, and posterior regions of the medial zone

Data analyses

All data were digitally filtered using a third-order, dual pass, Butterworth low-pass filter with resultant filter order of six and at a cutoff frequency of 30 Hz. We determined average time history responses for the C4/5, C5/6, and C6/7 canal and foramen narrowing and widening. Single factor, non-repeated measures analysis of variance and pairwise Bonferroni post hoc tests were used to determine significant differences ($P < 0.05$) among WHIPS, AHR, and NHR in peak canal and foramen narrowing during the crashes. The peak narrowing was defined for each parameter (canal diameter and foramen width, height, and area) as its maximum change or reduction from its neutral posture value.

Results

Average time history responses for C4/5, C5/6, and C6/7 canal and foramen narrowing, and widening and intervertebral rotations during the crashes (Fig. 3) demonstrated temporal variation among WHIPS, AHR, and NHR. Average peak intervertebral extension ranged from 11.7° to 23.3° with NHR, 4.4°–9.0° with AHR, and 2.3°–7.4° with WHIPS (Table 1; Fig. 3e). Average peak canal and foramen narrowing could not be statistically differentiated between WHIPS, AHR, or NHR (Table 1). With NHR, peak narrowing of the canal was 1.8 mm at C5/6 and of the foramen 1.9 mm in width, 3.9 mm in height, and 6.5 mm² in area at C4/5. These peaks occurred between 181 and 271 ms, subsequent to the intervertebral extension peaks. WHIPS and AHR generally reduced peak narrowing as compared to NHR with few exceptions (WHIPS: C4/5 canal, C6/7 foramen area; AHR: C4/5 foramen area, C6/7 canal and foramen width and area).

With WHIPS, average peak narrowing of the canal reached 1.9 mm and of the foramen reached 1.6 mm in width, 2.7 mm in height, and 5.9 mm² in area, all at C4/5 between 152 and 224 ms (Table 1a). The times of these peaks encompassed the times of peak intervertebral extension, 180 ms, and occurred during or following contact of the head with the head restraint, which occurred between 120 and 216 ms.

With AHR, average peak narrowing of the canal reached 2.7 mm and of the foramen reached 1.6 mm in width, 1.9 mm in height, and 4.6 mm² in area between 148 and 230 ms (Table 1b, c). The narrowing peaks were observed at C6/7 for the canal and foramen width and height and at C5/6 for the foramen area. The times of the narrowing peaks occurred during intervertebral extension and during or following contact of the head with the AHR, which occurred between 66 and 148 ms.

Fig. 3 Average time history responses including: narrowing and widening of **a** spinal canal diameter, **b** foramen width, **c** foramen height, **d** foramen area, and **e** intervertebral rotation. Narrowing is represented as negative, while widening is positive. Average onset

and end of contact of the head with the head restraint is indicated for the crashes with the WHIPS: open circles and AHR: filled circles. For clarity, shaded regions representing the average response ± 1 SD are shown only for the crashes with no head restraint (NHR)

Discussion

Chronic radicular symptoms of muscle weakness and paresthesias of the neck, shoulders, upper back, and arms have been documented in whiplash patients [2]. Epidemiological studies indicate that modern anti-whiplash systems reduce the risk of neck injury during rear crashes [22], though their effects on cervical neural space kinematics are not fully understood. The present study evaluated the effects of WHIPS and AHR on cervical canal and foraminal narrowing of the lower cervical spine during simulated rear crashes of a human model of the neck, consisting of a neck specimen mounted to the torso of BioRID II and carrying a surrogate head and stabilized with muscle force replication (Fig. 1). Our present model is a logical evolution of our previous whole cervical spine model with muscle force replication [16], which provides a kinematic response similar to in vivo data [23] for simulated rear crashes below the human injury threshold. In the present study, contact of

the head with the head restraint occurred earlier with AHR as compared to WHIPS (Fig. 3) due to forward rotation of the AHR during the crash and differences in neutral posture neck alignment between WHIPS and AHR (Fig. 1). The neutral cervical posture with AHR was consistent with the in vivo in-position neutral posture [17], while with WHIPS it consisted of an out-of-position forward posture. This posture is consistent with field data [24, 25], which indicate that in the majority of rear crashes, the struck vehicle was slowing or stopping at the time of the crash implying that the occupants would be moving forward in their seats, in a forward out-of-position posture. The WHIPS and AHR mechanisms were activated during all the present crashes. Up to 1 cm of plastic deformation of the energy-absorbing elements was observed due to the crashes with WHIPS [26]. Average peak forward rotation of the AHR relative to the seatback was 5.3° [10].

Average peak canal and foramen narrowing could not be statistically differentiated between WHIPS, AHR, or NHR

Table 1 Average peak spinal canal and foramen narrowing (mm or mm²), intervertebral rotations (deg), and occurrence times (ms) of the peaks at (a) C4/5, (b) C5/6, and (c) C6/7 during the rear crashes with

the whiplash protection system (WHIPS), active head restraint (AHR), and with no head restraint (NHR)

	Narrowing				Intervertebral rotation	
	Canal diameter	Foramen width	Foramen height	Foramen area	Flexion	Extension
(a) C4/5						
WHIPS	1.9 (1.4); 175	1.6 (1.1); 204	2.7 (1.2); 224	5.9 (2.1); 152	1.2 (1.0); 118	2.3 (1.5); 222
AHR	0.7 (0.6); 115	0.8 (0.3); 144	1.2 (0.8); 179	3.4 (2.3); 179	1.2 (1.8); 264	4.4 (4.1); 164
NHR	1.4 (1.3); 104	1.9 (1.3); 181	3.9 (2.5); 220	6.5 (1.5); 193	0.8 (0.5); 74	23.3 (6.2); 180
(b) C5/6						
WHIPS	0.6 (0.5); 117	0.5 (0.4); 130	1.3 (1.0); 125	3.5 (2.1); 125	1.1 (1.2); 80	2.8 (2.7); 176
AHR	1.3 (0.5); 217	1.0 (0.7); 205	1.5 (1.1); 155	4.6 (2.4); 148	4.1 (1.8); 300	5.2 (2.6); 164
NHR	1.8 (1.0); 271	1.2 (0.9); 150	1.6 (1.1); 130	4.4 (2.3); 122	1.1 (0.6); 70	11.7 (6.6); 178
(c) C6/7						
WHIPS	0.4 (0.2); 101	0.2 (0.2); 115	1.0 (0.4); 188	2.4 (0.9); 188	1.6 (0.9); 94	7.4 (1.6); 150
AHR	2.7 (2.9); 152	1.6 (1.0); 118	1.9 (1.2); 230	3.3 (1.2); 165	1.7 (0.9); 68	9.0 (2.5); 144
NHR	1.7 (1.1); 176	1.3 (0.6); 181	2.0 (2.8); 131	1.9 (1.4); 101	1.3 (0.5); 64	15.0 (4.7); 192

Data are provided in the format: magnitude (SD); occurrence time

The occurrence times of the peaks are relative to the onset of the sled horizontal acceleration

(Table 1), most likely due to small sample sizes, large standard deviations, adjustment of *p* values for multiple post hoc comparisons, and use of non-repeated measures statistical analyses. Continued experimental research with greater sample sizes would be needed to conclusively determine whether or not significant differences exist in cervical neural space kinematics between WHIPS, AHR, and NHR.

Peak cervical canal and foramen narrowing of the present study with NHR (Table 1) are contrasted by previously reported data from simulated rear crashes of a human head–neck model [14, 15]. Present peak foraminal width narrowing of 1.9 mm at C4/5 compares favorably with 1.7 mm, previously reported at C5/6. Present peak canal narrowing was smaller (1.8 vs. 3.5 mm) and peak foraminal height and area narrowing were larger (3.9 vs. 1.8 mm; 6.5 vs. 4.5 mm²) than the previously reported data, likely due to differences in neck loads. The head–neck model, with its T1 vertebra fixed to a mini-sled which accelerated horizontally, experienced large shear and tensile neck loads [11] likely increasing canal narrowing. These loads are contrasted by large compressive neck loads of the present model due to straightening of the kyphotic thoracic curvature and upward torso ramping [9], which likely increased foraminal height and area narrowing. Average upward translation of the T1 vertebra reached 4.8 cm [9]. The compressive neck loads with WHIPS, as determined in a separate study using inverse dynamics [9], reached 617.7 N with peak compression occurring prior to contact of the head with the head restraint. In contrast, the neural space narrowing and rotation peaks occurred during

or following contact of the head with the head restraint during intervertebral extension (Fig. 3; Table 1).

As with all in vitro studies, the limitations and assumptions of our model should be considered when interpreting our results. The present specimens, with an average age of 86 years, were older than younger adults who are more often involved in motor vehicle crashes. However, older occupants have a higher risk of injury, sustain greater injury severity, and experience longer recovery times for a given crash as compared to younger occupants [27, 28]. The canal and foraminal points used to calculate neural space narrowing were mathematically reconstructed based upon previously reported in vitro measurements of cervical quantitative anatomy. As such, the present neural space narrowing data represent approximations of the actual narrowing based on the mathematical representations of the canal and foraminal points. While we studied two of the most common anti-whiplash systems, WHIPS and AHR, we did not investigate the response of traditional, non-active, non-yielding seats. Various AHR designs exist and are specific to automobile manufacturer and model. We studied a mechanically based AHR, which uses the displacement of the torso between the side members of the seatback to rotate the head restraint forward in response to torso load applied to the seatback (Fig. 1b). We did not study other AHR designs such as electronically based AHR, which likely offers protective performance that varies from the present results. While we compared neural space narrowing between WHIPS, AHR, and NHR, we did not compare these data with those obtained during physiologic flexion–extension motions. The comparisons

between WHIPS and AHR may be confounded by differences in neutral posture neck alignment and the initial gap between the head and head restraint among the test conditions (Fig. 1). The dynamic responses of WHIPS and AHR and consequently the canal and foraminal narrowing are dependent upon occupant size, weight, and crash severity, though we did not vary these parameters. While limited by these factors, our study provides insight into the effects of WHIPS and AHR on cervical neural space narrowing and neural tissue injury potential.

To evaluate the potential for cervical neural tissue compression injury during the rear crashes and to determine the effects of WHIPS and AHR, we computed the minimum soft tissue canal diameter and minimum bony foraminal width, height, and area with and without cervical stenosis, and contrasted these data with average neural tissue dimensions (Fig. 4). These minima were computed by subtracting the peak narrowing of the present study from the corresponding nonstenotic and stenotic dimensions as reported in previous studies and used for similar comparisons in earlier work [14, 15, 29]. The previously reported data of quantitative foraminal anatomy were measured at 45° to the midsagittal plane. These extrapolations assume that canal and foramen narrowing during rear crashes are similar in individuals with and without cervical stenosis.

The present minimum canal and foramen dimensions for individuals without cervical stenosis during the rear crashes with NHR (Fig. 4a) compare favorably with previously reported results [14, 15, 29]. These cumulative data indicate no risk of cord compression due to bony impingement in those with normal canal diameters [14]; however, ganglia compression injury may occur due to foraminal width and height narrowing [15, 29]. Foraminal area narrowing was unable to predict neural tissue injury in those without cervical stenosis, again consistent with previous studies [13]. Our data with NHR demonstrated greater potential ganglion and nerve root compression in those with cervical stenosis due to nonphysiologic foraminal width, height, and area narrowing (Fig. 4b).

The present data demonstrated minimal effects of WHIPS and AHR in reducing potential ganglia and nerve root compression injuries. In those without cervical stenosis, potential ganglion injury exists at C6/7 due to foraminal width narrowing even with the AHR (Fig. 4a). For those with cervical stenosis, the injury risk greatly increases and spreads to include the ganglia at C4/5 through C6/7 with WHIPS or AHR in addition to the C6/7 nerve root with AHR (Fig. 4b). These potential injuries occur due to intervertebral extension during or following contact of the head with the head restraint.

While both WHIPS and AHR have been shown in epidemiological studies to reduce the risk of whiplash injuries

Fig. 4 The minimum canal diameter and foraminal width, height, and area for **a** nonstenotic and **b** stenotic canal and foramen during the rear crashes (whiplash protection system, WHIPS: open circles; active head restraint, AHR: filled circles; no head restraint, NHR: dashed line). The minimum values were obtained by subtracting the peak narrowing of the present study from previously reported average neutral posture dimensions of the soft tissue canal diameter and bony foraminal width, height, and area. To evaluate the potential for neural tissue compression, previously reported average cord, ganglia, and nerve root dimensions are also shown. The cord diameter and nerve root dimensions are darkly shaded, while the ganglia dimensions are lightly shaded

[22], previous biomechanical studies have demonstrated non-physiologic lower cervical spine motions during simulated rear crashes even with these modern anti-whiplash systems. While rear crashes may injure multiple cervical

tissues, our present biomechanical study of simulated whiplash demonstrated neural space kinematics of sufficient magnitude to injure the spinal ganglia and nerve roots of the lower cervical spine. Ultimately, anti-whiplash systems may be improved to reduce foraminal narrowing, which may lead to reduced radicular symptoms and sensory hypersensitivity and hypoesthesia in whiplash patients.

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Conflict of interest None.

References

- Barnsley L, Lord S, Bogduk N (1994) Whiplash injury. *Pain* 58:283–307
- Chien A, Eliav E, Sterling M (2008) Whiplash (grade II) and cervical radiculopathy share a similar sensory presentation: an investigation using quantitative sensory testing. *Clin J Pain* 24:595–603
- Humphreys SC, Hodges SD, Patwardhan A, Eck JC, Covington LA, Sartori M (1998) The natural history of the cervical foramen in symptomatic and asymptomatic individuals aged 20–60 years as measured by magnetic resonance imaging. A descriptive approach. *Spine (Phila Pa 1976)* 23:2180–2184
- Hardy AG (1977) Cervical spinal cord injury without bony injury. *Paraplegia* 14:296–305
- Chen IH, Vasavada A, Panjabi MM (1994) Kinematics of the cervical spine canal: changes with sagittal plane loads. *J Spinal Disord* 7:93–101
- Holmes A, Han ZH, Dang GT, Chen ZQ, Wang ZG, Fang J (1996) Changes in cervical canal spinal volume during in vitro flexion–extension. *Spine (Phila Pa 1976)* 21:1313–1319
- Ebraheim NA, Liu J, Shafiq Q, Lu J, Pataparla S, Yeasting RA, Woldenberg L (2006) Quantitative analysis of changes in cervical intervertebral foramen size with vertebral translation. *Spine (Phila Pa 1976)* 31:E62–E65
- Nuckley DJ, Konodi MA, Raynak GC, Ching RP, Mirza SK (2002) Neural space integrity of the lower cervical spine: effect of normal range of motion. *Spine* 27:587–595
- Ivancic PC, Xiao M (2011) Understanding whiplash injury and prevention mechanisms using a human model of the neck. *Accid Anal Prev* 43:1392–1399
- Ivancic PC, Sha D, Panjabi MM (2009) Whiplash injury prevention with active head restraint. *Clin Biomech (Bristol, Avon)* 24:699–707
- Ivancic PC, Panjabi MM, Ito S (2006) Cervical spine loads and intervertebral motions during whiplash. *Traffic Inj Prev* 7:389–399
- Ortengren T, Hansson HA, Lovsund P, Svensson MY, Suneson A, Saljo A (1996) Membrane leakage in spinal ganglion nerve cells induced by experimental whiplash extension motion: a study in pigs. *J Neurotrauma* 13:171–180
- Nuckley DJ, Van Nausdile JA, Raynak GC, Eck MP, Harrington RM, Ching RP (2003) Examining the relationship between whiplash kinematics and a direct neurologic injury mechanism. *Int J Vehicle Des* 31:68–82
- Ito S, Panjabi MM, Ivancic PC, Pearson AM (2004) Spinal canal narrowing during simulated whiplash. *Spine* 29:1330–1339
- Panjabi MM, Maak TG, Ivancic PC, Ito S (2006) Dynamic intervertebral foramen narrowing during simulated rear impact. *Spine* 31:E128–E134
- Ivancic PC, Panjabi MM, Ito S, Crompton PA, Wang JL (2005) Biofidelic whole cervical spine model with muscle force replication for whiplash simulation. *Eur Spine J* 14:346–355
- Descarreaux M, Blouin JS, Teasdale N (2003) A non-invasive technique for measurement of cervical vertebral angle: report of a preliminary study. *Eur Spine J* 12:314–319
- Panjabi MM, Duranceau J, Goel V, Oxland T, Takata K (1991) Cervical human vertebrae. Quantitative three-dimensional anatomy of the middle and lower regions. *Spine* 16:861–869
- Panjabi MM, Oxland TR, Parks EH (1991) Quantitative anatomy of cervical spine ligaments. Part II. Middle and lower cervical spine. *J Spinal Disord* 4:277–285
- Ebraheim NA, An HS, Xu R, Ahmad M, Yeasting RA (1996) The quantitative anatomy of the cervical nerve root groove and the intervertebral foramen. *Spine* 21:1619–1623
- Pearson AM, Ivancic PC, Ito S, Panjabi MM (2004) Facet joint kinematics and injury mechanisms during simulated whiplash. *Spine* 29:390–397
- Farmer CM, Wells JK, Lund AK (2003) Effects of head restraint and seat redesign on neck injury risk in rear-end crashes. *Traffic Inj Prev* 4:83–90
- Kaneoka K, Ono K, Inami S, Yokoi N, Hayashi K (1997) Human cervical spine kinematics during whiplash loading. In: International conference on new frontiers in biomechanical engineering. Tokyo, Japan, pp. 265–268
- Viano DC (2002) Role of seat in car crash safety. Society of Automotive Engineers, Inc., Warrendale, PA, USA
- NHTSA (2012) National Highway Traffic Safety Administration. National Automotive Sampling System–Crashworthiness Data System. US Department of Transportation, Washington. <http://www.nhtsa.gov/NASS>; <ftp://ftp.nhtsa.dot.gov/NASS/>. Accessed Jan 2012
- Xiao M, Ivancic PC (2010) WHIPS seat and occupant motions during simulated rear crashes. *Traffic Inj Prev* 11:514–521
- Kent R, Henary B, Matsuoka F (2005) On the fatal crash experience of older drivers. *Annu Proc Assoc Adv Automot Med* 49:371–391
- Morris A, Welsh R, Hassan A (2003) Requirements for the crash protection of older vehicle passengers. *Annu Proc Assoc Adv Automot Med* 47:165–180
- Tominaga Y, Maak TG, Ivancic PC, Panjabi MM, Cunningham BW (2006) Head-turned rear impact causing dynamic cervical intervertebral foramen narrowing: implications for ganglion and nerve root injury. *J Neurosurg Spine* 4:380–387