Paul C. Ivancic
Manohar M. Panjabi
Yasuhiro Tominaga
Adam M. Pearson
S. Elena Gimenez
Travis G. Maak

Spinal canal narrowing during simulated frontal impact

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P. C. Ivancic · M. M. Panjabi (✉)
Y. Tominaga · S. Elena Gimenez
T. G. Maak
Department of Orthopaedics and
Rehabilitation, Biomechanics Research
Laboratory, Yale University School of
Medicine, 333 Cedar St.,
P.O. Box 208071, New Haven,
CT 06520-8071, USA
E-mail: manohar.panjabi@yale.edu
Tel.: +1-203-7852812
Fax: +1-203-7857069

A. M. Pearson
Department of Orthopaedic Surgery,
Dartmouth-Hitchcock Medical Center,
Lebanon, NH, USA

Abstract Between 23 and 70% of occupants involved in frontal impacts sustain cervical spine injuries, many with neurological involvement. It has been hypothesized that cervical spinal cord compression and injury may explain the variable neurological profile described by frontal impact victims. The goals of the present study, using a biofidelic whole cervical spine model with muscle force replication, were to quantify canal pinch diameter (CPD) narrowing during frontal impact and to evaluate the potential for cord compression. The biofidelic model and a sled apparatus were used to simulate frontal impacts at 4, 6, 8, and 10 g horizontal accelerations of the T1 vertebra. The CPD was measured in the intact specimen in the neutral posture (neutral posture CPD), under static sagittal pure moments of 1.5 Nm (pre-impact

CPD), during dynamic frontal impact (dynamic impact CPD), and again under static pure moments following each impact (post-impact CPD). Frontal impact caused significant ($P < 0.05$) dynamic CPD narrowing at C0-dens, C2-C3, and C6-C7. The narrowest dynamic CPD was observed at C0-dens during the 10 g impact and was 25.9% narrower than the corresponding neutral posture CPD. Interpretation of the present results indicate that the neurological symptomatology reported by frontal impact victims is most likely not due to cervical spinal cord compression. Cord compression due to residual spinal instability is also not likely.

Keywords Frontal impact · Injury mechanism · Spinal cord injury · Cervical spine · Spinal cord narrowing

Introduction

Whiplash-type trauma can result in soft tissue injuries of the cervical spine that may remain undetected using current imaging modalities [14, 28]. Most previous research has investigated the injury mechanisms due to rear-end collisions [5, 9, 26, 33, 51], with relatively few frontal impact investigations [8, 12, 13, 46, 49]. Between 23 and 70% of occupants involved in frontal impacts sustain cervical spine injuries, many with neurological involvement [1, 3, 25, 43, 50]. The resulting symptoms

include neck pain, headache, weakness, and paresthesias [19]. Although less common, central nervous injury symptoms such as visual disturbances, dizziness, trigeminal sensory impairment and confusion have also been reported [3, 25, 43]. While most neurological symptoms are resolved with conservative management, a minority of patients experience chronic sequelae [14].

Both transient and permanent neurological symptoms, without vertebral fracture or dislocation, have been reported due to frontal impact or hyperflexion loading of the cervical spine [17, 41, 44]. Patients with

narrow cervical spinal canals are more likely to develop chronic sequelae than those with normal canal diameters [40]. Further, patients with cervical spondylosis exhibited an increase in cervical stenosis due to physiological flexion causing narrowing of the anterior subarachnoid space and anterior cervical cord compression [30].

In addition to clinical evidence of neurological injury due to whiplash-type trauma, biomechanical models have been used to quantify narrowing of the cervical spinal canal due to static pure moment application, and dynamic simulated rear impacts [7, 22, 32, 33]. Using spinal canal and intervertebral foramen occlusion transducers, Nuckley et al. [33] simulated rear impacts up to 8 g using a whole cervical spine model and reported non-significant spinal canal diameter narrowing. Using the whole cervical spine model with muscle force replication, Ito et al. [22] simulated rear impacts up to 8 g and calculated the dynamic spinal canal narrowing using rigid body transformation of intervertebral kinematic data recorded optically. They reported that spinal cord injury during rear impact is unlikely in subjects with normal cervical canal diameters; however, those with severe cervical spinal canal stenosis may be at risk. Neural tissue injury and resultant neurological symptoms may be associated with cervical spinal cord compression, nerve root impingement, or both. No previous biomechanical studies have quantified the cervical spinal canal narrowing due to simulated frontal impact.

In order to evaluate the potential for cervical cord injury due to frontal impact, the goals of this study were to: (1) quantify the cervical spinal canal narrowing during simulated frontal impacts of increasing severity using the whole cervical spine model with muscle force replication, (2) determine if frontal impact resulted in a narrower post-impact canal diameter, and (3) extrapolate the present results to evaluate the potential for cord

compression in individuals with normal canal diameters and in those with cervical canal stenosis.

Materials and methods

Specimen preparation

Six fresh-frozen human osteoligamentous whole cervical spine specimens (occiput-T1) were mounted in resin (Fiberglass Evercoat, Cincinnati, OH, USA) at the occiput and T1 in normal neutral posture with the foramen magnum parallel to the occipital mount, and the T1 vertebra tilted anteriorly by 24°. The average age of the specimens was 71.3 years (range: 54–87 years) and there were three male and three female donors. The donors did not suffer from any disease that could have affected the osteoligamentous structures. To attach lightweight motion-tracking flags, a headless wood screw was drilled into the anterior aspects of C1 and C2 and into the lamina of C3–C7. Each flag consisted of a 3-mm diameter hollow brass tube with two white, spherical, radio-opaque markers. The flag was fitted rigidly onto each wood screw, and additional flags were attached to the occipital and T1 mounts. The flag markers were used to determine the spinal kinematics during flexibility testing and during frontal impact. This constituted the whole cervical spine (WCS) model prepared for intact flexibility testing.

To prepare a specimen for frontal impact simulation, a surrogate head (mass 3.3 kg and sagittal plane moment of inertia 0.016 kg m²) was rigidly attached to the occipital mount of the WCS model such that the surrogate head center of mass, relative to the spine specimen, corresponded with values measured from cadavers [4, 48]. The surrogate head and spine were stabilized

Fig. 1 Schematic of the whole cervical spine model with muscle force replication ready for simulated frontal impact in the mini-sled apparatus

Fig. 2 Lateral schematic of the upper cervical spine to demonstrate the canal pinch diameter (CPD) and vertebral motion-tracking flags. For C0-dens, the sagittal CPD was defined as the distance from the posterior point of the foramen magnum and the postero-superior point of the dens. At C1–C2, the CPD was defined as measured from the postero-inferior corner of C1 and the corner of the dens as it joins with the C2 vertebral body. For C2–C3, and the lower intervertebral levels, the CPD was the distance between the postero-inferior corner of the spinolaminar line of the upper vertebra and the postero-superior corner of the lower vertebral body

using the compressive muscle force replication (MFR) system (Fig. 1) [21, 22]. The MFR system was symmetric about the mid-sagittal plane and consisted of anterior, posterior and lateral cables attached to pre-loaded tension springs anchored to the base. The stiffness coefficients of the anterior, lateral and posterior springs were 4.0, 4.0, and 8.0 N/mm, respectively. Two anterior cables, each consisting of two strands, originated at the occipital mount, ran through separate guideposts at C4, through pulleys within the T1 mount and were each connected to a separate spring. The preload in each anterior spring was 15 N. To apply the posterior MFR, small holes were drilled bilaterally into each lamina (C3–C7) and wire loops were inserted into the spinal canal through the laminae and tightly secured above each vertebral spinous process. The two posterior MFR cables originated from the occipital mount, ran through the wire loops, through a pulley at the T1 mount and each were connected to a spring, pre-loaded to 15 N. The lateral cables originated from C0, C2, C4, and C6, passed alternately along lateral guide rods, and were each connected to a spring with a 30 N preload. With this MFR arrangement the compressive pre-loads at each intervertebral level were: 120 N (C0–C1, C1–C2); 180 N (C2–C3, C3–C4); 240 N (C4–C5, C5–C6), and 300 N (C6–C7, C7–T1). A C0–C2 flexion limiter was secured to the occipital mount and to the C2 spinous process that allowed approximately 30° of sagittal

rotation, consistent with the in vivo rotation of the normal cervical spine [11, 34]. This constituted the WCS+MFR model, ready for frontal impact simulation.

Definitions

The canal pinch diameter (CPD) (Fig. 2) at each intervertebral level was determined under various loading conditions, as defined below (Fig. 3):

<i>Neutral posture CPD</i> (mm)	CPD of intact WCS in the neutral posture
<i>Pre-impact CPD</i> (mm) :	CPD of intact WCS under static 1.5 Nm flexion-extension moment prior to impact
<i>Dynamic impact CPD</i> (mm) :	minimum CPD observed during impact of WCS+MFR model during the total head-T1 flexion time period (i.e., from the T1 horizontal acceleration onset to return to neutral posture, after maximum head-T1 flexion)
<i>Post-impact CPD</i> (mm) :	CPD of WCS under static 1.5 Nm flexion-extension moment following each impact
<i>Pre-impact CPD narrowing</i> (mm) =	neutral posture CPD–pre-impact CPD
<i>Pre-impact CPD narrowing</i> (%) =	100* (pre-impact CPD narrowing)/(neutral posture CPD)
<i>Dynamic impact CPD narrowing</i> (mm) =	neutral posture CPD—dynamic impact CPD

Fig. 3 Schematics demonstrating the experimental protocol and definition of the canal pinch diameter (CPD) under various loading conditions

Dynamic impact CPD narrowing (%) = $100 \times (\text{dynamic impact CPD narrowing}) / (\text{neutral posture CPD})$

Post-impact CPD narrowing (mm) = $\text{neutral posture CPD} - \text{post-impact CPD}$

Post-impact CPD narrowing (%) = $100 \times (\text{Post-impact CPD narrowing}) / (\text{neutral posture CPD})$

Pre-impact and post-impact CPDs

Sagittal plane flexibility testing was performed on the intact WCS model and after each impact to determine the pre-impact and post-impact CPDs, respectively, at each intervertebral level. Pure flexion and extension moments to a maximum of 1.5 Nm were applied to the occipital mount in four equal steps and for three load-unload cycles. To minimize viscoelastic effects, 30-s wait periods were given following each load application. Kinematic data were recorded on the third load cycle using a high-speed digital camera (MotionPRO, Redlake, MSAD Inc., San Diego, CA, USA). The pre-impact and post-impact CPDs were calculated using a procedure analogous to that used for the dynamic impact CPD calculation as described below.

Frontal impact simulation and monitoring

Frontal impact simulation was performed using a previously developed bench-top sled apparatus [36]. The incremental trauma protocol was used to apply frontal impact loading to the WCS+MFR model at nominal T1 horizontal accelerations of 4, 6, 8, and 10 g [15]. A head stop was used to restrict head extension during the rebound phase to within physiological limits and thus prevent extension injuries. The head stop was not designed to mimic an automotive head restraint or injury prevention system. The high-speed camera recorded the spinal motions at 500 frames/s.

Dynamic impact CPD

A lateral X-ray of each intact WCS in the neutral posture, together with two mid-sagittal radio-opaque calibration markers, was taken and digitally scanned

(Adobe Photoshop Version 6.01, San Jose, CA, USA). The calibration markers were used to determine specimen-specific X-ray magnification factor. To determine the neutral posture CPD for C0-dens, the posterior edge of the foramen magnum and the postero-superior point of the dens were selected, and for C1–C2, the postero-inferior corner of C1 and the postero-inferior point of the dens were chosen (Fig. 2). For C2–C3 to C6–C7, two points, the postero-inferior corner of the spinolaminar line of the upper vertebra and the postero-superior corner of the lower vertebral body defined the neutral posture CPD. Geometrical rigid body relationships between the flag markers and the CPD points, established on the lateral X-ray and converted to mm using the specimen-specific X-ray magnification factor, were superimposed onto the first frame of the high-speed movie. Custom motion-tracking software written in Matlab (The Mathworks Inc., Natick, MA, USA) was used to calculate the intervertebral rotations and flag marker translations at each subsequent frame. These data, together with the geometrical rigid body relationships, were used to calculate the translations of the CPD points during impact. The average error in the computation of the CPD was 0.3 mm (SD 0.2 mm) [39]. The CPD narrowing velocity was computed by numerically differentiating the dynamic impact CPD narrowing.

Data analyses

The dynamic impact CPD narrowing data were low pass, dual-pass digitally filtered at a cut-off frequency of 30 Hz. Residual and Fourier analyses demonstrated that most of the signal power was contained under 20 Hz. Single factor repeated measures ANOVA and Bonferroni post hoc tests (Minitab Rel. 13, State College, PA, USA) were performed to determine statistical differences ($P < 0.05$) between the dynamic impact CPD narrowing and the pre-impact CPD narrowing and between the post-impact and pre-impact CPD narrowing, at each intervertebral level. To determine the temporal event patterns during simulated impact, single factor repeated measures ANOVA and Bonferroni post hoc tests were performed between the times of the peak CPD narrowing at each intervertebral level and the time of the peak head flexion.

Table 1 Average (SD) nominal and measured peak T1 horizontal accelerations (g), acceleration pulse durations (ms), and peak velocities (kph) obtained during simulated frontal impact

Nominal peak T1 Horizontal Acceleration (g)	Measured peak T1 Horizontal Acceleration (g)	Measured T1 Horizontal acceleration Pulse duration (ms)	Measured peak T1 Horizontal Velocity (kph)
4	3.9 (0.2)	113.8 (11.4)	8.4 (1.1)
6	5.7 (0.1)	106.7 (5.4)	11.4 (0.6)
8	8.5 (0.3)	106.8 (14.5)	13.4 (0.5)
10	10.0 (0.4)	99.0 (3.2)	13.8 (1.3)

Fig. 4 A representative example (specimen no. 1) of the T1 horizontal acceleration pulse as a function of time for the 4, 6, 8, and 10 g impacts

Table 2 Specimen biographic data and neutral posture canal pinch diameters (CPDs) (mm)

Specimen No	Age (Years)	Gender	CPDs (mm)						
			C0-dens	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
1	54	M	31.7	19.1	19.1	16.3	15.5	18.0	20.0
2	60	F	32.3	18.9	18.5	18.2	17.8	18.2	16.8
3	87	M	36.0	22.6	19.4	18.8	18.6	19.3	17.4
4	68	F	30.7	15.6	15.6	14.5	17.0	18.4	18.6
5	80	M	32.9	24.0	16.7	16.9	16.9	18.6	18.3
6	79	F	32.6	17.2	18.7	17.0	15.4	18.2	14.7
Average (SD)	71.3 (12.8)		32.8 (1.8)	19.6 (9.6)	18.0 (1.4)	16.9 (1.5)	16.8 (1.1)	18.5 (0.5)	17.6 (1.4)

For *C0-dens*, the CPDs were measured from the posterior point of the foramen magnum to the postero-superior point of the dens. For *C1-C2*, the CPD was measured from the postero-inferior point of C1 to the postero-inferior point of the dens. For *C2-C3*

through *C6-C7*, two points, the postero-inferior corner of the spinolaminar line of the upper vertebra and the postero-superior corner of the lower vertebral body defined the neutral posture CPD (Fig. 2)

Results

The average measured T1 horizontal acceleration peaks were 3.9, 5.7, 8.5, and 10.0 g, corresponding to the nominal maximum accelerations of 4, 6, 8, and 10 g, respectively (Table 1). The average duration of the T1 horizontal acceleration pulse ranged between 99.0 and

113.8 ms during the 10 g (ΔV : 13.8 kph) and 3.9 g (ΔV : 8.4 kph) impacts, respectively. A representative example of the T1 horizontal acceleration pulse as a function of time for the 4, 6, 8, and 10 g impacts is provided for specimen no. 1 (Fig. 4).

The average neutral posture CPD ranged from 32.8 at C0-dens to 16.8 mm at C4-C5 and compared

Fig. 5 A representative example (human no. 1, 8 g impact) of dynamic **A** intervertebral rotations and **B** canal pinch diameter (CPD) narrowing throughout the cervical spine. Positive CPD narrowing is defined as a decrease in the CPD. Flexion is defined as positive rotation, while extension is defined as negative rotation

Table 3 Average (SD) dynamic impact and pre-impact canal pinch diameter (CPD) narrowing (mm) throughout the cervical spine during 4 to 10 g impacts

	CPD Narrowing (mm)						
	C0-dens	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Pre-Frontal impact	1.5 (1.4)	1.1 (0.5)	1.2 (0.8)	0.6 (0.4)	0.9 (0.9)	0.6 (0.3)	0.5 (0.4)
4 g	3.2 (2.1)	1.3 (0.7)	2.4 (1.5)	0.5 (0.4)	0.6 (0.3)	0.3 (0.4)	0.2 (0.5)
6 g	3.5 (2.5)	2.6 (1.5)	3.3 (1.7)*	0.5 (0.6)	0.8 (0.8)	0.5 (0.4)	0.5 (0.7)
8 g	5.7 (1.9)**	1.7 (0.5)	3.9 (2.6)*	0.5 (0.6)	1.0 (1.4)	0.4 (0.3)	0.4 (0.5)
10 g	8.5 (3.1)*	2.0 (1.5)	3.9 (3.0)*	1.3 (0.6)	2.1 (1.5)	0.9 (1.5)	2.2 (1.0)*

For each impact and spinal level, significant differences between the dynamic impact and corresponding pre-impact CPD narrowing are indicated by *($P < 0.05$) and ** ($P < 0.1$)

Fig. 6 Average peak dynamic impact (4 to 10 g) and pre-impact spinal canal pinch diameter (CPD) narrowing (%) throughout the cervical spine

favorably with previous reports of average normal bony canal diameters, demonstrating that the present specimens and did not exhibit cervical canal stenosis (Table 2) [16, 18, 20, 29, 35, 38, 42]. The dynamic impact CPD narrowing varied among specimens, intervertebral levels and accelerations. In general, the peak CPD narrowing at C0-dens and C1–C2 occurred immediately following the peak T1 acceleration during intervertebral extension of the upper cervical spine, while the peak CPD narrowing at C2–C3 to C6–C7 occurred later during peak intervertebral flexion. This is exemplified by the dynamic rotations and CPD narrowing of human specimen no. 1 during the 8 g impact (Fig. 5a, b).

Table 4 Average (SD) times (ms) of peak canal pinch diameter (CPD) narrowing throughout the cervical spine and peak head flexion during frontal impact relative to the onset of the T1 horizontal acceleration

Chronology of Events	Time (ms) after impact
Peak C1–C2 CPD narrowing	94.8 (38.5)
Peak C0-dens CPD narrowing	100.5 (38.8)
Peak C5–C6 CPD narrowing	109.2 (90.1)
Peak C3–C4 CPD narrowing	116.7 (59.9)
Peak C2–C3 CPD narrowing	132.0 (48.1)
Peak head flexion	165.8 (17.3)
Peak C6–C7 CPD narrowing	184.1 (109.5)
Peak C4–C5 CPD narrowing	240.4 (87.3)

Peak CPD narrowing at C1–C2 and C0-dens occurred significantly earlier ($P < 0.05$) than peak head flexion, while peak C4–C5 CPD narrowing occurred significantly after peak head flexion

All frontal impact CPD narrowing data are presented in tabular form in which averages, standard deviations, and significant increases with respect to pre-impact values are given for each intervertebral level (C0–C1 to C6–C7) and each impact (Table 3). The average pre-impact CPD narrowing varied among intervertebral levels, ranging from 1.5 mm at C0-dens to 0.5 mm at C6–C7 (Table 3), which represented 3.7 and 4.6% narrowing of the corresponding neutral posture CPDs, respectively (Fig. 6). Dynamic impact caused the greatest CPD narrowing at C0-dens and generally increased as impact severity increased. A significantly narrower ($P < 0.05$) dynamic impact CPD, as compared to the corresponding pre-impact CPD, was first observed at C2–C3 during the 6 g impact and subsequently spread to C0-dens and C6–C7 at higher impact accelerations. The narrowest CPD was observed at C0-dens during the 10 g impact and was 8.5 mm or 25.9% narrower than the corresponding neutral posture CPD.

Since no statistically significant differences between the times of peak CPD narrowing and peak head flexion were observed among impact acceleration, the data from all accelerations were combined for the subsequent temporal analyses (Table 4). The chronology of event times demonstrated that the peak CPD dynamic narrowing occurred first at C1–C2, 94.8 ms after impact, followed by C0-dens at 100.5 ms; both significantly earlier ($P < 0.05$) than the time of the peak head flexion (165.8 ms). The peak C4–C5 CPD narrowing occurred at 240.4 ms, significantly after the peak head flexion.

Table 5 Average (SD) peak velocity (m/s) of canal pinch diameter (CPD) narrowing throughout the cervical spine during frontal impact

	CPD narrowing velocity (m/s)						
	C0-dens	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
4 g	0.09 (0.04)	0.04 (0.02)	0.05 (0.02)	0.02 (0.01)	0.02 (0.01)	0.03 (0.01)	0.04 (0.02)
6 g	0.21 (0.08)	0.11 (0.04)	0.08 (0.04)	0.03 (0.01)	0.03 (0.01)	0.04 (0.02)	0.03 (0.02)
8 g	0.23 (0.04)	0.08 (0.03)	0.12 (0.08)	0.03 (0.01)	0.03 (0.01)	0.04 (0.01)	0.03 (0.01)
10 g	0.98 (0.11)	0.15 (0.07)	0.22 (0.13)	0.12 (0.09)	0.14 (0.04)	0.08 (0.09)	0.17 (0.08)

Table 6 Average (SD) post-impact and pre-impact (4 to 10 g) canal pinch diameter (CPD) narrowing (mm) throughout the cervical spine under 1.5 Nm static sagittal loading

	CPD narrowing (mm)						
	C0-dens	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Pre-frontal impact	1.5 (1.4)	1.1 (0.5)	1.2 (0.8)	0.6 (0.4)	0.9 (0.9)	0.6 (0.3)	0.6 (0.4)
Post 4 g	4.3 (1.5)*	1.4 (0.4)	1.5 (0.3)	0.6 (0.4)	0.8 (0.9)	1.1 (0.6)	0.4 (0.4)
Post 6 g	4.8 (1.5)*	1.3 (0.9)	1.6 (0.8)	0.6 (0.3)	1.0 (1.0)	1.3 (0.7)	0.8 (0.8)
Post 8 g	4.7 (0.5)*	1.0 (0.4)	1.8 (0.6)*	0.7 (0.5)	0.9 (1.1)	1.6 (1.2)*	0.6 (0.5)
Post 10 g	6.3 (0.2)*	1.5 (1.0)	1.7 (0.3)	0.7 (0.4)	1.0 (1.1)	1.5 (1.1)*	0.6 (0.3)

Significant differences between post-impact and pre-impact CPD narrowing are indicated by * ($P < 0.05$) and ** ($P < 0.1$)

The peak CPD narrowing velocity ranged between 0.02 m/s at C3–C4 and C4–C5 during the 4 g impact to 0.98 m/s at C0-dens during the 10 g impact (Table 5). The peak CPD narrowing velocity generally increased as the impact acceleration increased.

The post-impact CPDs were significantly narrower than the corresponding pre-impact CPDs beginning following the 4 g impact at C0-dens, the 8 g impact at C2–C3, and at C5–C6 following the 8 and 10 g impacts (Table 6, Fig. 7).

Discussion

Clinical studies have documented neurological symptoms such as neck pain, headache, weakness, and paresthesias due to frontal collisions [3, 19, 25, 43]. Using the whole cervical spine (WCS) model with muscle force replication (MFR), the present study has quantified the cervical spinal canal narrowing during dynamic frontal impact and pre-impact and post-impact flexibility testing. The results indicated that both the dynamic impact CPD and the post-impact CPD were significantly narrower ($P < 0.05$) than the corresponding pre-impact CPD. The dynamic impact CPD became progressively narrower with increased impact severity; the greatest narrowing of 8.5 mm, or 25.9%, occurred at C0-dens at 10 g (Fig. 6). The peak CPD narrowing at C0-dens and C1–C2 preceded the peak head flexion indicating that peak upper cervical spine CPD narrowing occurred during the inverse S-phase, defined by upper cervical

spine extension and lower cervical spine flexion [37]. Peak dynamic impact narrowing at C2–C3 and C6–C7 exceeded physiologic limits (pre-impact CPD) with corresponding canal occlusion of 3.9 mm (21.9%) and 2.2 mm (14.9%), respectively. The peak CPD narrowing velocity remained under 1 m/s. Animal studies have shown that 40–60% occlusion is required to cause impaired neuronal conduction at impact velocities below 3 m/s, thus indicating that frontal impact is unlikely to cause neurological symptoms due to dynamic cord compression for the experimental conditions studied [2, 6, 24]. Post-impact CPDs were significantly narrower at C0-dens, C2–C3, and C5–C6, which corresponded to peak narrowing of 6.3 mm (19.1%) at C0-dens, 1.8 mm (9.9%) at C2–C3, and 1.6 mm (8.9%) at C5–C6.

The limitations of the current model and the CPD computational methodology must be considered when formulating conclusions regarding neural injury mechanisms. The specimens of the present study, with an average age of 71.3 years, were weaker than the population most likely to suffer frontal impact trauma. To ensure that no specimen was prematurely injured due to excessive head inertia loads, a 3.3 kg surrogate head mass was used, which was at the lower end of values measured from cadavers, ranging between 2.8 and 5.8 kg [4, 48]. While spinal cord compression may be caused by soft tissue protrusions into the canal, it was not possible to track the displacements of the soft tissues using the rigid body analysis of the current study. Previous studies have provided soft tissue canal dimensions thus enabling extrapolation of the current results [7, 20,

Fig. 7 Average peak post-impact and pre-impact canal pinch diameter (CPD) narrowing throughout the cervical spine

31, 32]. The MFR system provided passive resistance to intervertebral motion following impact simulating the response of an unwarned occupant and thus did not

Fig. 8 Minimum soft tissue canal diameter for frontal and rear impact in those with normal canals and in those with spondylotic canals. The minimum soft tissue canal diameters were calculated by subtracting the peak dynamic frontal impact narrowing of the present study from the average antero-posterior soft tissue canal diameters [7, 20, 31, 32]. Rear impact data, from a previous experiment is shown for comparison [22]. To evaluate the potential for cord compression, the average dural diameters are also shown [10, 20, 23, 31, 45]

model the active neuromuscular response. A recent study of cervical muscle reaction times during simulated frontal impacts up to 1.4 g found that peak muscle activity occurred at 189 ms [27], indicating that the cervical muscles may not respond quickly enough to protect the neural tissue from injury. Irrespective of the timing, sufficient muscle strength is needed to affect the neck kinematics. The average peak extension moment of 18.7 Nm at C7-T1 produced by the present MFR exceeds the peak extension moment developed by weaker subjects (in vivo female range: 9–33 Nm) [47]. Thus, the posterior cervical muscles may neither be able to develop

sufficient strength, nor respond quickly enough to restrict head flexion or protect the neck following frontal impact. In the present study, the incremental trauma approach was utilized. A previous study using a porcine cervical spine model demonstrated that incremental and single trauma protocols produce equivalent soft tissue injury severity due to frontal impacts [15]. Although interpretation of our results is limited by these factors, we believe that the observed kinematic patterns may improve our understanding of the neural injury mechanisms during frontal impact.

Based on the average antero-posterior spinal cord and soft tissue canal diameters reported in previous studies [7, 10, 20, 23, 31, 32, 45], it is possible to extrapolate the present results to evaluate the potential for spinal cord compression during frontal impact in subjects with normal canal diameters and in those with cervical spinal canal stenosis (Fig. 8). The minimum soft tissue canal diameter, obtained by subtracting the peak CPD narrowing obtained in the present study from the corresponding average soft tissue canal diameters, demonstrated that there is no risk for cord compression during frontal impact in individuals with normal canal diameters or in those with cervical canal stenosis. Based upon a recent rear impact study, using a similar meth-

odology, there does exist potential for rear impact-induced cord compression at C4–C5 and C5–C6 in those with cervical canal stenosis [22].

Conclusions

Using a biofidelic model to simulate frontal impact, this study has shown that the dynamic and post-impact CPDs were significantly narrower than the pre-impact CPDs but were of insufficient magnitude to cause cord compression or injury. Extrapolation of the current data produced a similar conclusion for patients with cervical spinal canal stenosis. A previous biomechanical study using similar methodology has documented the potential for cord compression during rear impact in individuals with cervical spinal canal stenosis. Neurological symptoms have been reported following whiplash-type injury and may be associated with cervical cord compression, however interpretation of the present results indicated that this mechanism does not play a role in frontal impact injury.

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