

2

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Cervical Spine Whiplash Kinematics and Its Effect on Neural Space Integrity

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ABSTRACT

Despite the prevalence of whiplash-related injuries, a connection between clinical symptoms and injury mechanism has been elusive. Previous studies have attempted to correlate the whiplash kinematic response to injury mechanisms; however, none has specifically examined the potential for neurologic involvement from foraminal occlusion. This biomechanical study measured cadaver cervical spine whiplash kinematics and compared these with changes in the neural space geometry of the cervical spine, providing a measure of the direct neurologic injury potential. Extension and shear displacements of each cervical level were measured and found to be similar to that reported in the literature and within the tissue's physiologic limits. Further, changes to the spinal canal and intervertebral foraminal geometry were recorded during whiplash and cross-sectional area changes were documented (up to 15.3%). Because these foraminal occlusions were smaller in magnitude than those resulting from normal cervical motion, our findings do not support direct neurologic injury resulting from segmental vertebral kinematics as a whiplash injury mechanism.

INTRODUCTION

A number of recent vehicle designs have incorporated specially developed systems to prevent whiplash injuries. Modified seat backs and/or head restraint systems designed to reduce relative head-to-torso motion have emerged in an effort to address this common automotive injury. However, until the mechanism of injury associated with whiplash can be clearly identified, prevention strategies will be unable to specifically target the cause of these injuries. This study was undertaken to examine the potential for direct neurologic tissue insult (neural tissue compression) as a mechanism for whiplash injury.

Although neck injuries account for a significant number of the reported injuries in rear-end automobile crashes, the mechanism of cervical spine whiplash injury is not well understood (Deans, 1987; Van Den Kroonenberg, 1998). During rear-end impacts/accelerations, the human

spine has been shown to undergo non-physiological S-shaped kinematics that could be the source of whiplash injury (Grauer, 1997; Siegmund, 1997). Unfortunately, individuals experiencing these collisions present with a myriad of symptoms: headaches, soreness in the neck, transient numbness or tingling of the upper extremities, and pain of undefined origin, making it impossible to identify a specific clinical injury (Barnsley, 1994). Some investigators have attempted to link whiplash kinematics with specific and appropriate injury mechanisms including: muscular damage (Szabo, 1994), osteoligamentous injury, capsular ligament elongation (Panjabi, 1998), facet joint injury (Ono, 1997), and facet joint pinching (Cusick, 2001). These mechanisms beget neurologic symptoms indirectly. However, the potential for direct compression of the neurologic tissues in the spinal canal and intervertebral foramen has not been investigated as a whiplash injury mechanism.

Cervical spine vertebral displacements in excess of those related to normal daily activity might compromise the neural tissues of the spine, thus producing direct neurologic injury. A number of studies have investigated cervical spine kinematics of whiplash incidents (Cusick, 2001; Deng, 2000; Grauer, 1997; Kaneoka, 1997; Matsushita, 1994; Ono, 1997; Panjabi, 1998; Siegmund, 1997). Many of these studies raise the possibility that the initial S-phase, and not the subsequent period of hyperextension (C-phase), is the source of most injury (Grauer, 1997; Kaneoka, 1997; Matsushita, 1994; Panjabi, 1998). The angular displacements associated with this S-phase have been shown to include extension at the lower levels, little angular change in the middle cervical spine and relative flexion in the upper cervical spine (Cusick, 2001; Deng, 2000; Grauer, 1997; Ono, 1997). Grauer et al. (Grauer, 1997) evaluated different whiplash acceleration impulses (2.5g, 4.5g, 6.5g and 8.5g) and recognized a trend towards increasing maximum angular displacement with increasing acceleration. Others have shown that the shear (translational) displacements of the cervical vertebra increased as you move down the cervical spine with C7-T1 having the greatest shear displacement (Cusick, 2001; Deng, 2000). Individually, the shear and angular displacements of the cervical spine in response to head inertial loading were within the tissues' physiologic range; however, the combined (shear and angular displacement) whiplash response motions have been reported to be non-physiologic (Grauer, 1997; Ono, 1997; Siegmund, 1997). Since these cervical spine motions can exceed normal kinematics, it is conceivable that direct compromise of the neural tissues may occur as a result of these displacements (Figure 1).

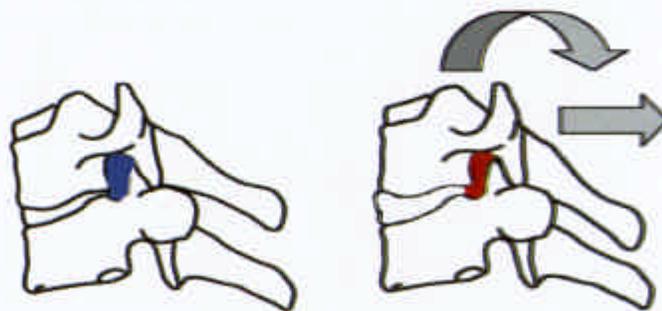


Figure 1. Schematic demonstrating whiplash changes in cervical spine kinematics which might affect the intervertebral neural spaces. It is this relationship between cervical spine whiplash kinematics and the spines ability to protect the neural tissues that is under investigation.

We have defined the ability of the vertebral column to preserve the space available to the spinal cord and nerve roots as its neural space integrity (Nuckley, 2002). Neural space integrity of both the spinal canal and intervertebral foramen has been investigated in a number of experimental studies (Carter, 2000; Chen, 1995; Ching, 1998; Holmes, 1996; Nuckley, 2002; Panjabi, 2001; Reid, 1960; Yoo, 1992). These studies have quantified the space available to the nerve roots and spinal cord in intact, injury, and post-injury experiments. Nuckley et al.(Nuckley, 2002) measured the spinal canal mid-sagittal diameter and intervertebral foramen area for quasi-static tests of normal cervical spine range of motion. At maximal extension of the intact spine, they found the spinal canal was unoccluded at C4-5 and occluded 6.3% (C5-6) and 9.2% (C6-7) in the lower cervical spine. Likewise, the intervertebral foramen were occluded 16.7% (C4-5), 20.0% (C5-6), and 13.8% (C6-7) in maximal physiologic extension. These values of intact neural space geometries agreed with the work of Yoo, et al.(Yoo, 1992) in the intervertebral foramen and Holmes, et al.(Holmes, 1996) in the spinal canal. Since these values were measured for the intact cervical spine, they likely represent occlusions, less than which, no neurologic injury would occur. However, it is conceivable that excessive spinal motions produced during whiplash impact/acceleration events may exceed the normal kinematics of the cervical spine whereby generating deleterious levels of neural space occlusion.

Therefore, the goal of this research study was to examine the relationship between rear-impact whiplash kinematics and neural space integrity using a human cadaver model. The results for two distinct whiplash acceleration magnitudes were compared to investigate a dose-dependent response. These data will elucidate whether direct neurologic injury (i.e., nerve root or spinal cord compression caused by spinal canal or intervertebral foramen occlusion) may serve as a whiplash injury mechanism. *The hypotheses for this experimental work were (i) segmental spinal kinematics (angular and shear displacements) are unique for each cervical level for a given acceleration input, (ii) segmental spinal motions are different for low and high whiplash acceleration magnitudes, (iii) spinal canal and intervertebral foramen integrity is significantly diminished during whiplash events, and (iv) the severity of spinal canal and intervertebral foramen occlusion is dependant upon the whiplash acceleration magnitude.*

METHODS

Specimen Preparation.

Six fresh-frozen cadaveric head and cervical spine specimens (Occiput-T5) were obtained through the Department of Biological Structure's Willed Body Program at the University of Washington and the International Institute for the Advancement of Medicine (Jessup, PA), and were selected based on a lack of significant degenerative changes, surgery, or prior neck injury. The mean age of the two females and four males was 45-years (range: 16-68-years). The spinal musculature, nerve roots and associated tissue, and spinal cord and dura were removed from each specimen taking care not to damage the discs and ligaments. The superior portion of the head was removed using a modified Beier procedure cutting posterior-to-anterior through the Frankfurt plane retaining the functional anatomy of the upper cervical spine and occiput (Beier, 1980). In an effort to provide consistent and repeatable inertial loading to each specimen, a Hybrid III anthropomorphic test dummy head was mounted to the remaining occiput. This procedure facilitated the measurement of the angular and linear accelerations (via the Hybrid-III head instrumentation package) at the center of gravity of the head while minimizing loading variability. The inferior vertebrae (T3-T5) were potted in dental plaster (Labstone Buff, Bayer Corp., South Bend, IN) with the C6 posterior vertebral end plate in 20-degrees flexion to mimic the normal lordotic position of the cervical spine (Cusick, 2001). Immediately after dissection, each specimen was hydrated, wrapped in

towels, sealed in a plastic bag, and frozen at -20°C to preserve their mechanical properties (Panjabi, 1985).

Instrumentation.

Spinal canal and intervertebral foramen integrity were measured utilizing transducers developed in our laboratory at the University of Washington (Ching, 1998). The spinal canal occlusion transducer (SCOT) and intervertebral foramen occlusion transducer (IFOT) are capable of measuring both static and dynamic occlusions to the neural spaces of the cervical spine. These transducers are constructed of closed flexible tubing (SCOT: Tygon R-1000, Norton Performance Plastics, Canton, OH; IFOT: Natural rubber, Primeline Industries, Akron, OH) filled with a conductive media (0.9% saline). When a constant-amplitude electric current (2.0-kHz, $20\mu\text{Amp}$) is passed through the saline, the voltage across the tube's length provides a measure of the system's resistance in accordance with Ohm's Law. Since the resistance in the tubing is proportional to its cross-sectional area, monitoring the differential voltage across the tube's length gives a direct measure of its cross-sectional area. During static calibration to a known deformation, both the SCOT and IFOT exhibited an exponentially shaped calibration curve, indicating that measurement sensitivity increased with increasing occlusion of the tubing. The SCOT did not fill the entire bony canal laterally, but rather encompassed the entire mid-sagittal space giving a measure of the minimum mid-sagittal diameter of the spinal canal. The IFOTs fit snugly into the intervertebral foramen and measured the minimum cross-sectional area of the foramen. The functional characteristics these neural space occlusion transducers are presented in Table 1 and apply to quasi-static as well as dynamic events (Raynak, 1998). These transducers measure neural space geometries with a high degree of accuracy ($<6.5\%$ full scale error) to elucidate changes in the integrity of the cervical spine neural spaces.

Table 1. NEURAL SPACE OCCLUSION TRANSDUCER

	IFOTs			SCOT
	6.35mm	7.95mm	9.54mm	15.88mm
Range	17.36 - 30.56 mm^2	22.72 - 48.24 mm^2	31.16 - 68.83 mm^2	5.40 - 14.30 mm^2
Accuracy	$\pm 1.71 \text{ mm}^2$	$\pm 2.01 \text{ mm}^2$	$\pm 2.93 \text{ mm}^2$	$\pm 0.93 \text{ mm}^2$
Sensitivity	0.05 - 1.63 V/mm^2	0.02 - 0.97 V/mm^2	0.02 - 0.34 V/mm^2	0.04 - 3.44 V/mm^2
Resolution	0.007 mm^2	0.014 mm^2	0.021 mm^2	0.005 mm^2

Transducer characteristics were determined through an analysis of the transducers' output (volts) and physical input (area) as measured using CT techniques.

The sagittal plane kinematics of the cervical spine was measured using 1.5-mm diameter threaded pins, which were inserted into the anterior bodies of C3, C4, C5, C6, and C7. These pins held reflective targets which were tracked via high speed video (Kodak Ektapro Imager, Eastman Kodak Co., Rochester, NY). The video files were digitized and analyzed using WINalyze motion analysis software (Mikromak GmbH, Erlangen, Germany). This software offers sub-pixel resolution via its digitizing algorithm (Frischholz, 2001); however, its absolute accuracy is dictated by the 512×384 pixel resolution of the video and for this study was 1.48-mm/pixel. Since our measurements were all normalized to initial conditions, the resolution of the system dictates the error in our differential measurements of shear and angular displacement. Therefore, the resolution and measurement error of the WINalyze motion analysis was 0.08-mm and 0.02-degrees, providing us with excellent kinematic measurement capabilities.

Loading Apparatus.

A custom designed closed-loop acceleration sled was used for bench-top testing of the cadaveric cervical spine with Hybrid III head preparations (Raynak, 2000). This acceleration sled utilizes linear motors (Trilogy Systems, Austin, TX) and a motion control system (Delta Tau Data Systems, Northridge, CA) to propel the specimens to specific acceleration magnitudes and pulse durations. Peak amplitudes of 2, 4, 6, 8, and 10-g of acceleration were generated on a haversine of 140-msec pulse-duration. These sled parameters were generated based upon comparison with human volunteer research performed at the Wright-Patterson Air Force Research Laboratories. Human volunteer responses were compared with Hybrid III ATD responses and then our sled was tuned using just the Hybrid III neck and head portions to replicate on our bench-top the same environment 'seen' by the human subjects.

Experimental Procedure.

Each dissected specimen was thawed for experimentation and maintained hydrated with lactated Ringers solution throughout testing. Pins were inserted into the C3, C4, C5, C6, and C7 vertebral bodies for video marker attachment and kinematic analysis. Intervertebral foramen occlusion transducers were inserted into the bilateral foramen of C3-4, C4-5, C5-6, and C6-7 such that each transducer was pre-occluded in its foramen to increase initial sensitivity. The spinal canal occlusion transducer was then inserted into the canal aligning the sensing regions within our spinal region of interest (Figure 2).

The fully instrumented specimen (with the Hybrid III head) was then mounted into the bench-top sled for acceleration testing (Figure 2). Each specimen received each of the acceleration profiles in a semi-random order to preserve the specimen. The 2, 4, and 6-g trials were randomized and performed first, followed by a random choice of the 8 or 10-g trial. This project specifically examined the results of the whiplash acceleration profiles for only the 4 and 8-g runs. In this regard, each specimen was always exposed to the lower (4-g) acceleration run before the higher (8-g) magnitude run.

The specimen was placed on the sled in neutral position prior to acceleration and their posture was maintained with masking tape on the head form. Then the specimen was perturbed from equilibrium by the acceleration profiles listed above. During this whiplash event, 23-channels of data were collected at 4000-Hz. These data included the IFOT and SCOT data as well as the head linear X, Y, Z, and rotational Y accelerations and the sled linear X acceleration. The data were recorded using a LabVIEW data acquisition board (PCI-6071E, National Instruments™, Austin, TX) on a personal computer (E-5200, Gateway™, Sioux Falls, ND) and were synchronized with a high-speed video camera recording the event at 1000-frames/sec.

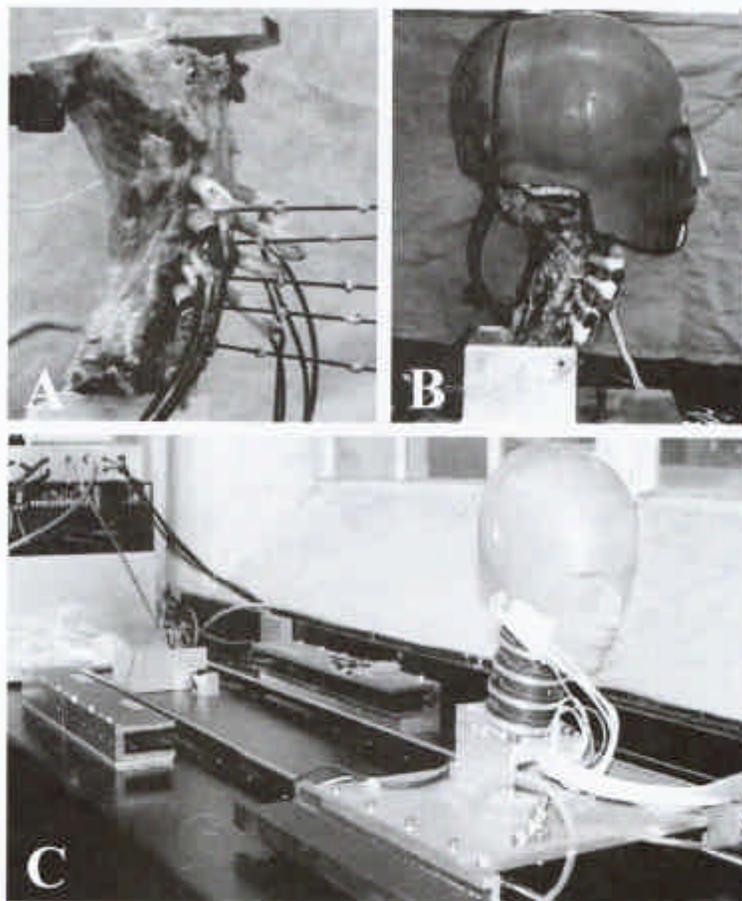


Figure 2. Instrumented human cervical spine prepared for testing on the whiplash acceleration sled. (A) Neural space integrity was measured by custom occlusion transducers shown here emanating from the bilateral intervertebral foramen of C3-4, C4-5, C5-6, and C6-7 (not shown: spinal canal occlusion transducer). Steel rods inserted into the C3, C4, C5, C6 and C7 vertebral bodies contain white markers to facilitate the video measurement of spinal kinematics. (B) The specimen was next fit with the Hybrid III head on its occiput mounting plate and rigidly attached to the acceleration sled. (C) The inertial loading apparatus includes fixed magnets (back left) which repel the sled once current is passed through the electromagnets attached to the sides of the sled. This sled acceleration pulse provides the whiplash input to the specimen.

Data Analysis.

All of the SCOT and IFOT data were run through a zero-phase shift four-pole Butterworth low-pass digital filter. The cut-off frequency was set at 120-Hz. These filtered data were then calibrated to SCOT mid-sagittal diameter and IFOT cross-sectional areas. The high-speed videos were colorized and converted into AVI file format (Kodak PC Color, Eastman Kodak Co., Rochester, NY and VideoMach, Gromada, www.gromada.com). This video was then imported into WINalyze to digitize and track each vertebra. The WINalyze SSD algorithm for automatic tracking was used and the points were digitally filtered using a 7-kernal Savitzky-Golay smoothing technique. Each vertebra contained two markers, which were used to identify that vertebra's angular displacement directly. The points were also converted into shear (translational) displacements of each vertebra with respect to its inferior (lower) vertebra. These data were obtained via a coordinate transformation (Eq. 2) which converted the inferior vertebra's markers into the x-axis of the cartesian coordinate system, thus eliminating angular displacement contamination of the shear displacements. The (X_1, Y_1) coordinates are for the anterior-inferior aspect of the inferior vertebra, and (X_2, Y_2) are the coordinates of the superior vertebra's anterior-inferior point. Using these data and the angular displacement between the adjacent vertebrae, θ (Eq. 1) the shear translation, X_3 is calculated for its new coordinate system.

$$\theta = \text{Tan}^{-1} \left[\frac{(Y_2 - Y_1)}{X_2 - X_1} \right] \quad (1)$$

$$X_3 = X_2 \text{Cos} \theta + Y_2 \text{Sin} \theta - X_1 \text{Cos} \theta - Y_1 \text{Sin} \theta \quad (2)$$

Once each vertebra's angular and shear displacements were determined with respect to the adjacent inferior vertebra, the data were grouped for analysis. This study investigated differences between spinal levels (C3-4, C4-5, C5-6, C6-7) and acceleration treatments (4-g and 8-g) with regard to kinematics and neural space integrity. A two-way ANOVA was performed on the data at discrete time points 100-msec (arbitrary) and 130-msec (this was the greatest common time-point before the head contacted restraint across all sled runs) and using the peak displacements for each level and acceleration. Further, the neural space integrity data was examined and the peak values were compared using a two-tailed Student's t-test to examine if dynamic changes to the neural spaces occurred which were significantly different from baseline neural space dimensions. Significance for each of these analyses was established at an alpha level of 0.05.

RESULTS

Whiplash Segmental Kinematics.

In general, the kinematic response (S-shape) of the cadaveric specimens in our experiment was very similar to those in the literature (Figure 3). The linear and angular displacements measured for each intervertebral joint (C3-4, C4-5, C5-6, C6-7) are illustrated in the time histories of Figures 4 and 5. Figure 4 illustrates the mean angular displacement time histories for both the 4-g and 8-g tests. These data demonstrate angular displacements increasing in extension for the lower cervical spine while the middle cervical spine shows very little angular displacement. Extension of the lower cervical spine appeared to increase at a greater rate for the 8-g trials compared with the 4-g tests. However, comparison of the 4-g and 8-g trials revealed no statistical difference in angular displacement at 100-msec, 130-msec, or peak angulation. There were statistical differences between the spinal levels for both the 4-g and 8-g tests. At both the 100-msec, and 130-msec time

points, the angular displacement of the C3-4 level was different from each of the other levels ($p < 0.013$). Significant differences were not observed between the other levels of the lower cervical spine although the C4-5 and C6-7 levels showed a trend ($p < 0.051$) of greater angulation than the lower cervical spine (C6-7).

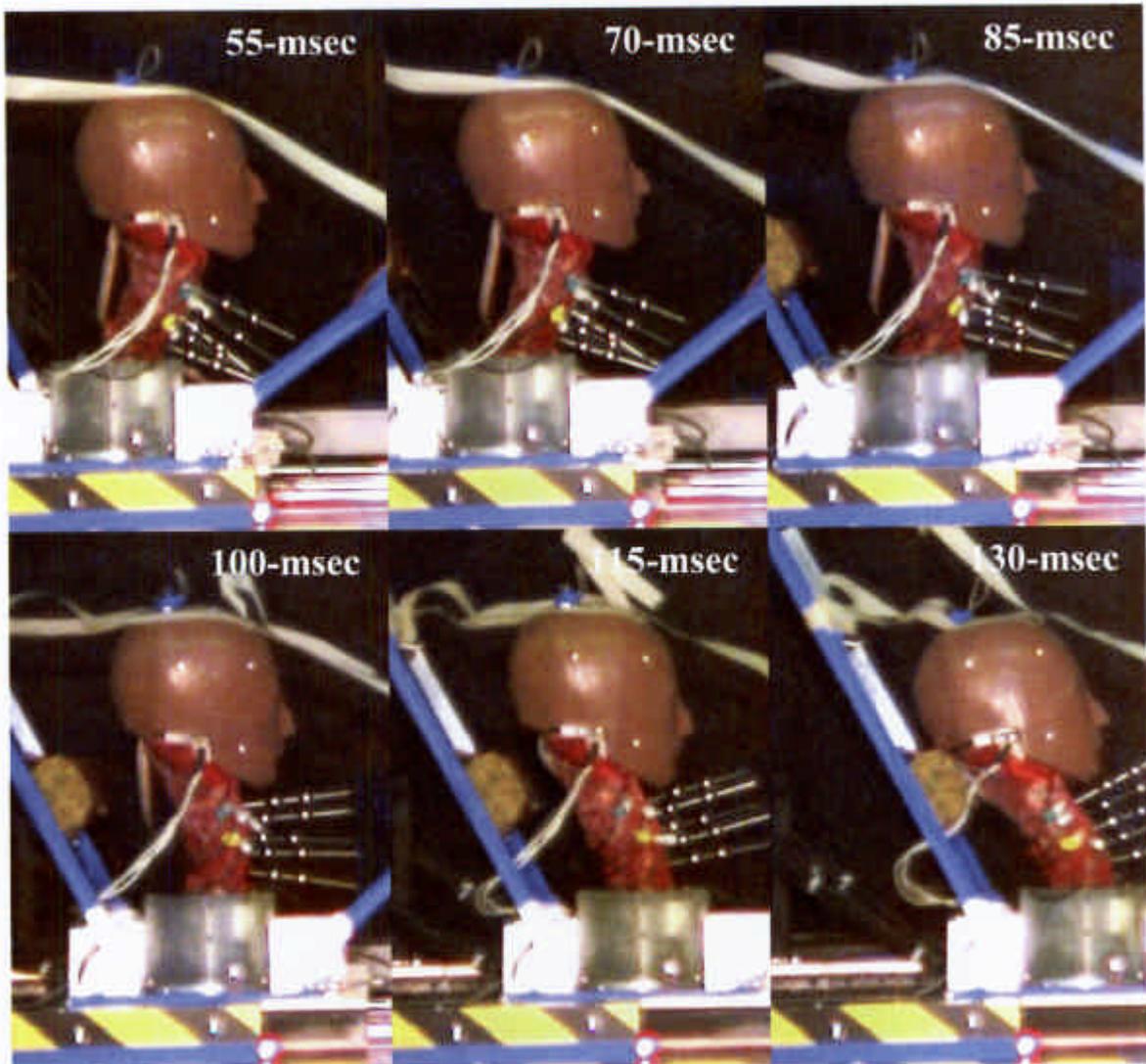


Figure 3. High speed video stills of an 8-g whiplash acceleration test. These images demonstrate pictorially the time history of the whiplash event. Time $t = 0$ is the initiation of sled acceleration and the peak sled acceleration occurred at 80-msec. Note the marker movement showing increasing extension angular displacement of the lower cervical spine with little change in the head position.

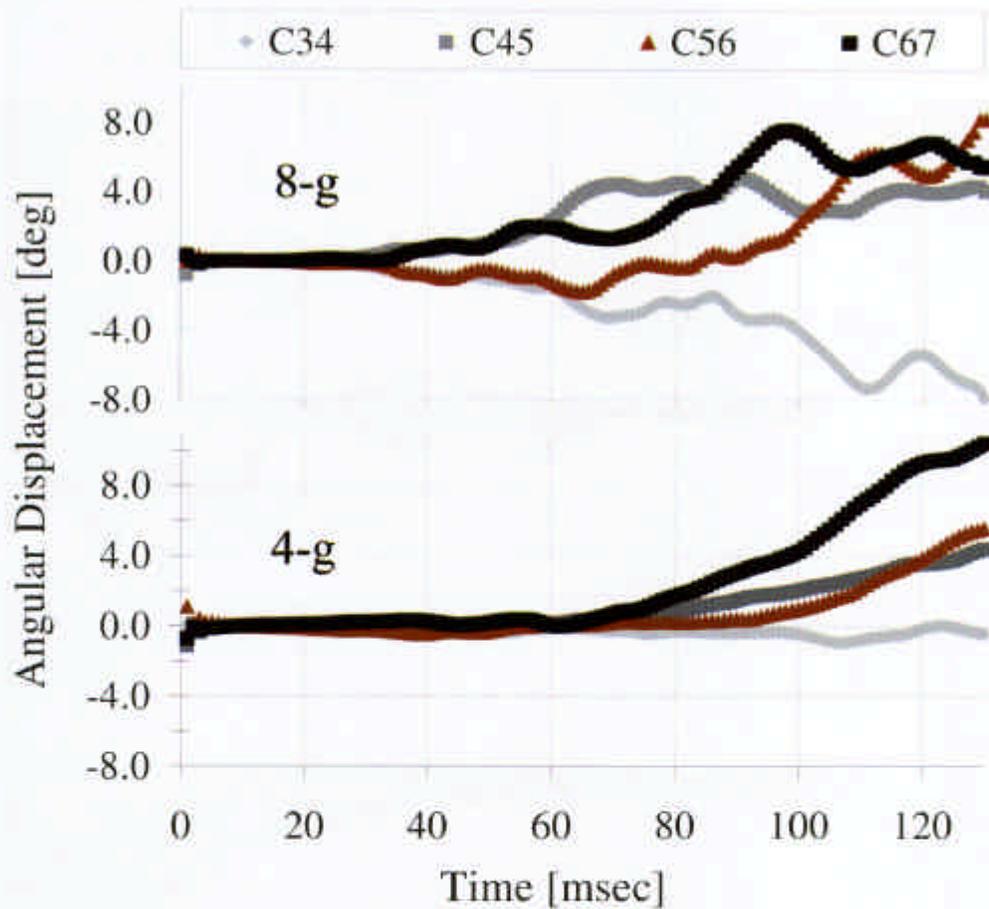


Figure 4. Cervical spine angular displacement kinematic response to a rear-end accelerations. These plots show the mean angular displacement of the specimens for 8-g [N=4] and 4-g [N=6] whiplash acceleration inputs. The relative angular change for each functional spinal unit shows a unique pattern for the lower cervical spine (C4-5, C5-6, and C6-7) compared with the C3-4 level for both acceleration treatments. No statistical differences were uncovered between the 4-g and 8-g angular displacement data although their time histories appear quite different.

The translational (shear) displacements (superior-posterior) are shown in Figure 5. These data also illustrate an increasing rate of shear displacement with increased magnitude of the acceleration pulse. The shear displacements measured herein are not statistically different between the 4-g and 8-g trials. Unfortunately, we could not demonstrate spinal level shear differences although a trend towards increased shear displacement at lower cervical levels existed.

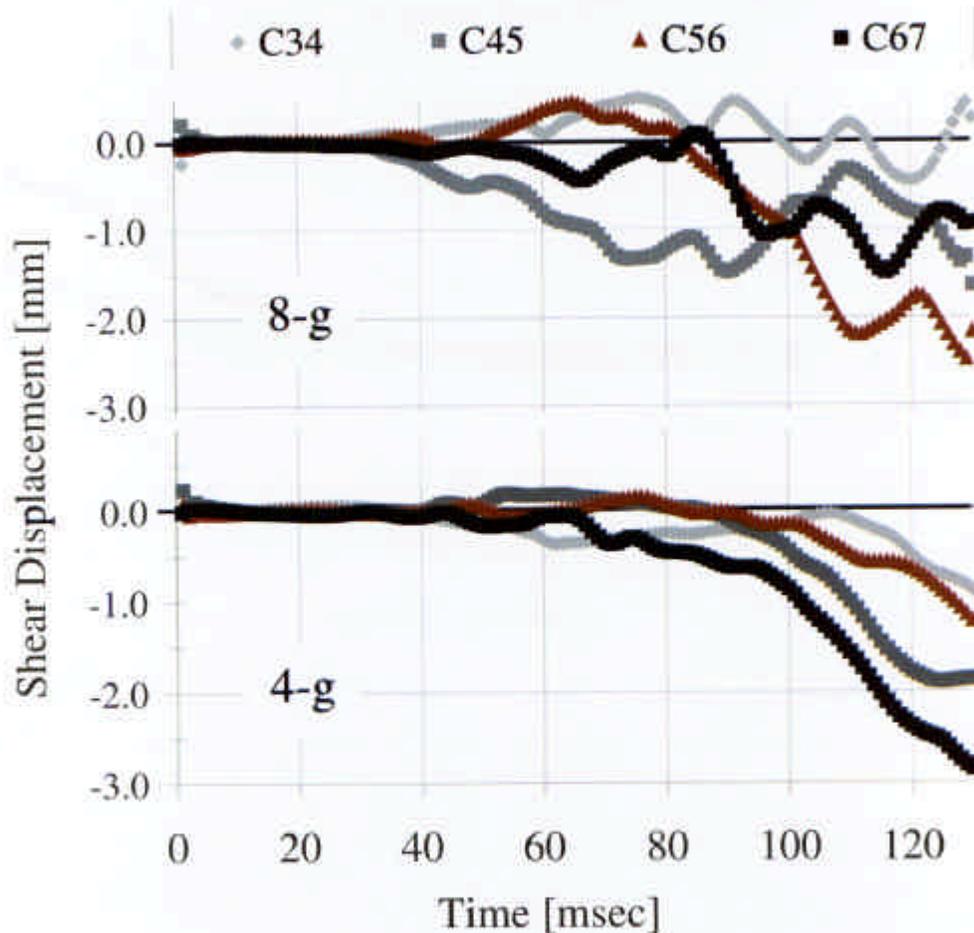


Figure 5: Cervical spine shear displacement kinematic response to whiplash. These plots show the mean shear displacement of the specimens for 8-g [N=4] and 4-g [N=6] whiplash acceleration inputs. The relative superior-posterior shear displacements for each functional spinal unit demonstrate no statistical differences between the levels. Further, comparison of the acceleration treatment (4-g vs. 8-g) produced no statistical relationship in spite of their different rates of shear displacement.

Spinal Canal Integrity.

Measurable changes in the spinal canal revealed that the spinal canal mid-sagittal diameter was minimally affected throughout the whiplash events. None of these occlusions were statistically significant nor was there a significant correlation between occlusion, cervical level, or acceleration treatment. There were, however, trends demonstrating that the upper cervical spinal canal (C1-C2) increased in mid-sagittal diameter for both the 4-g and 8-g experiments.

Intervertebral Foramen Integrity.

Significant changes to the intervertebral foramen area were observed at different foraminal levels for each of the acceleration treatments. The 4-g experiments revealed significant occlusion at C5-6 intervertebral foramen (bilaterally), reducing its cross-sectional area by 11.4% on average ($p = 0.029$) from the intact baseline condition. Whiplash insults of 8-gs produced significant changes in both the C5-6 (12.2%, $p = 0.031$) and the C6-7 (15.3%, $p = 0.006$) intervertebral foramen. These occlusions were the only significant measures on average, although trends were seen in the lower cervical spine suggesting a possible relationship between foraminal occlusion and acceleration impulse. Further, progression up the cervical spine from C6-7-to-C3-4 demonstrated decreasing foraminal occlusion to the point of foraminal opening at the C3-4 level. Finally, the 4-g experiments had a calculated average occlusion velocity of 0.06-m/sec compared with the 8-g trials average occlusion velocity of 0.18-m/sec.

DISCUSSION

The objective of this research was to determine whether the cervical spine kinematics associated with whiplash has the potential for causing direct neurologic injury (neural tissue compression via neural space occlusion). Several hypotheses examining the relationship between whiplash kinematics and neural space integrity were tested using an experimental human cadaver model. The results herein demonstrate that the kinematic response of the cervical spine is level specific (varies by spinal level) and produces significant geometric changes in the neural space boundaries which house the neural tissues. This discussion will look closely at each of the tested hypotheses and critically evaluate their significance towards establishing a relationship between whiplash kinematics and direct neurologic injury.

Whiplash Kinematics.

The segmental spinal kinematics measured for each specimen during the 4-g and 8-g tests demonstrated a large degree of inter-specimen variability. Although not uncommon for experimental biological testing, this variability, made establishing statistically significant cervical level or acceleration effects difficult and perhaps improbable without a larger number of test specimens. Nevertheless, some of the findings are noteworthy. The angular displacements were distinctly different for the middle cervical spine compared with the upper cervical spine. While trends were observed, no statistically significant changes by cervical level were able to be determined for segmental shear. Since hypothesis (i) was concerned with measuring any significant level kinematics distinction, we reject the null hypothesis and purport that segmental spinal kinematics vary by cervical level (i.e., the upper vs. mid-cervical spine differ in angular displacement). Hypothesis (ii) examined the effect of whiplash acceleration magnitude and while the data appear to show different displacement patterns over the time history, differences between the 4-g and 8-g kinematics could not be supported. Thus, we accept the null hypothesis that for the 4-g and 8-g whiplash inputs, there was no statistical difference in whiplash kinematics.

The kinematic response of the cervical spine to whiplash has been reported by others to fall within the physiologic range of the tissues (Cusick, 2001; Grauer, 1997; Ono, 1997; Siegmund, 1997). In agreement with these previous studies, the gross angular displacements measured herein were less than those reported as the end range of physiologic motion for each spinal level (Dvorak, 1988; Lysell, 1969; Nuckley, 2002; Panjabi, 1994; Voo, 1998). Although non-physiologic shear strains have been documented in the facet capsules during whiplash, (Cusick, 2001; Grauer, 1997; Panjabi, 1998) the gross shear displacements of the entire functional spinal unit have not been shown to exceed shear failure displacements (Moroney, 1988; Panjabi, 1986). These non-physiologic (sub-failure) displacements of the facet joint have been implicated as a possible source of whiplash pain through pinching of the capsule which is rich in nociceptors (Barnsley, 1995; Cusick, 2001; Inami, 2000). Both the angular and shear displacements measured herein and for previous studies are within their specific physiologic ranges, however, this study was concerned with the summed (angular and shear) gross segmental motions induced by whiplash and whether they were sufficient to reduce the space available to the neural tissues.

Spinal Canal Integrity.

During the experimental whiplash event, the spinal canal integrity measurements demonstrated little change in canal mid-sagittal diameter from neutral position. Thus, we accept the null hypothesis (iii) indicating that the intact spine maintains its canal space in the face of whiplash accelerations/impacts. Since few individuals experience spinal cord injury in the absence of a structurally altering injury, this result was not unexpected. The final hypothesis (iv) examined the effect of whiplash acceleration magnitude on the severity of the neural space encroachment. The null hypothesis (iv) was accepted since we did not measure any significant changes in the spinal canal for either the 4-g or 8-g inputs.

While significant changes in the geometry of the cervical spinal canal due to whiplash extension were not observed, others have found spinal cord bending and necking to result from flexion and extension of the cervical spine (Breig, 1970; Chen, 1995; Panjabi, 1988; Reid, 1960). Our measurement of the boundary conditions of the spinal cord can predict compression to the neural tissues; however, the tensile mechanical environment surrounding the spinal cord cannot be measured. Other studies have investigated the spinal canal geometry to be significantly altered in compression loading (Carter, 2000; Panjabi, 2001) and subsequent post-injury positioning of the injured cervical spine (Ching, 1997). Thus, our results and those presented above suggest that for spinal cord injury to result from whiplash insults, it must be associated with either a frank (osteoligamentous) injury or excessive elongation of the spinal canal.

Intervertebral Foramen Integrity.

Our sled-induced whiplash inputs created significant intervertebral foramen occlusions to the lower cervical spine. Therefore, the null hypothesis (iii) for intervertebral foramen integrity is rejected. While we observed a trend toward increasing occlusion with an increased acceleration magnitude, null hypothesis (iv) is accepted because none of the foraminal occlusions were significantly different from one another between the 4-g and 8-g acceleration inputs. Despite significant changes in the intervertebral foramen integrity, none were greater than those experienced in normal quasi-static cervical motion. Therefore, it is not expected that the occlusions to the intervertebral foramen will bring about nerve root injury as a result of whiplash events.

The lower cervical spine (C5-6, C6-7) exhibited the largest occlusions to the intervertebral foramen in agreement with epidemiological neurologic injury data (Sances, 1984). Further, these levels exhibited the greatest angular (extension) and shear displacements, which typically result in reduced intervertebral foramen dimensions (Nuckley, 2002). Moving up the cervical spine revealed opening of the intervertebral foramen at C3-4. This is consistent with the relative flexion that is occurring during the initial S-phase of the whiplash event. These trends demonstrate the

association between intervertebral foramen integrity and cervical spine kinematics for whiplash (inertial) loading.

The Potential for Direct Neurologic Injury from Whiplash.

Although we have measured the geometric integrity of cervical spine neural spaces (i.e., boundary conditions) resulting from whiplash kinematics, we have not directly examined the effects of these neural space changes on the neural tissues themselves. The spinal cord fills approximately 48% of the canal by mid-sagittal diameter and nerve roots comprise 55-70% of the intervertebral foramen area (Humphreys, 1998; Lang, 1993). Neural space changes greater than these would surely affect the function of the neurons; however the measured occlusions due to whiplash were of lesser magnitude. Hence, a brief review of the neurologic tissue injury literature may help to clarify — and perhaps challenge— direct neurologic injury as a potential mechanism for whiplash injury.

Anderson, et al. applied high velocity insults, using a plunger-type device, directly to ferret spinal cords and were able to quantify both the degree of damage and the amount of cord deformation (Anderson, 1982). Their study found a correlation between evoked potentials and ranges of spinal cord occlusion. A 10-30% spinal cord occlusion (by mid-sagittal diameter) did not induce a significant evoked potential latency (from baseline). A 40-60% occlusion generated “impaired neuronal conduction... which probably represents eventual partial recover”, and above 75% occlusion, no functional recovery was observed.

In the most comprehensive neurotrauma experiment to date, Kearney, et al. specifically investigated the rate and deformation dependency of the spinal cord on functional neurologic deficit (Kearney, 1988). They reported that animals subjected to a spinal cord occlusion greater than 50% were unlikely to recover independent of contact velocity. However, as impact velocity increased (above 2-3-m/sec), the amount of deformation necessary to produce non-recoverable injury decreased significantly. Hence, a quasi-static neurologic injury threshold of around 50% spinal cord deformation seems reasonable based on the above two studies. In addition, the rate of cord compression clearly plays a role in determining neurologic injury severity. The spinal canal and intervertebral foramen occlusion thresholds would likely be less than 50%, given that these spaces contain connective tissues, fat, and cerebrospinal fluid which respond viscoelastically and contain a finite mass in the foramen.

In our study, the measured canal and intervertebral foraminal occlusion magnitudes were much less than what would be expected to cause neurologic injury at quasi-static loading rates. However, anatomic variability (e.g., a congenitally narrow spinal canal) or degenerative changes (e.g., osteophytes and bone spurs) which affect the available space for the neural tissues may predispose an individual to neurologic tissue insult. Finally, measured occlusion rates on the order of only 0.18-m/sec suggest that the rate effects reported by Kearney et al. would unlikely play a role in exacerbating a potential neurological injury due to low-g whiplash events.

Other potential neural tissue injury mechanisms such as rapid fluid volume changes (e.g., cerebrospinal fluid or blood in the venous plexus) inducing pressure on the nerve roots or nerve root ganglion injury were not investigated as part of this study (Svensson, 1998; Svensson, 2000). While these represent other potential mechanisms for whiplash-induced neural tissue damage, they were beyond the scope of this study which focused solely on geometric changes to the foraminal spaces caused by segmental vertebral kinematics.

CONCLUSION

Our findings demonstrate physiologic angular and shear displacements in response to whiplash loading of the cervical spine in an experimental cadaver model. Significant changes in neural

space integrity of the lower cervical spine were measured during these tests; however the magnitude of these changes were generally small and on the order of those observed during normal physiologic range of motion. The relatively low rate of these neural space occlusions would be unlikely to affect the neural tissue injury threshold significantly so as to alter our findings. In sum, the data collected in this research effort do not support direct neural injury related to segmental kinematics as a whiplash injury mechanism. However, other mechanisms of direct neural tissue injury should be further investigated. These preliminary findings may help to guide future efforts in improving whiplash prevention strategies.

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DISCUSSION

PAPER: Cervical Spine Whiplash Kinematics and Its Effect on Neural Space Integrity

PRESENTER: David Nuckley, University of Washington

QUESTION: Mike Schlick, Medical College of Wisconsin.

Without getting a label for myself, I am no less than fascinated to find out a Hybrid III was attached to a human cervical spine. Can you share with us some of the details of that connection?

ANSWER: Yes. There is medical terminology. We cut right at the Frantfort plane so we had the base of the occiput was intact. And we potted the base of the occiput and we made a retrofit plate which actually setup right into and screwed into the Hybrid III, base of the Hybrid III head. We could not fit a load cell there because that would move the CG of the head much, much higher where it wouldn't be natural. So we just had the accelerometry package within the head but none of the load cells. We filled the base of the occiput with PMMA and drilled screws up through that into the Hybrid III head. And we didn't have any wobbling or any problems with that fixation.

Q: One other question I had, was the transducers that went through the foramen, I imagine they are flexible?

A: Yes.

Q: Are they a tubing of some sort?

A: They are a flexible tubing that we fill with saline which is a conductive media. We just pass a current, a constant current through that area. And that change in area is just like a resistor, you change the area of that resistor you increase resistance and you can measure that as a change in voltage.

Q: I take it then those passed from one foramen to the other so the spinal cord was gone at this point?

A: Yes. The spinal cord was gone. They actually only inserted into one foramen.

Q: It was bi-axial?

A: Yes.

Q: Okay.

A: Bi-axial, one foramen each. So the data that I presented is a means from both bilateral that we did.

Q: Guy Nusholtz, Daimler Chrysler

It seemed that the response in the 4G versus the 8G comparison that you did that you had more response at the 4G than you did in the 8G although you had a lot more scatter in the 8G. Do you have an explanation for that, why greater input would produce less response? Negative stiffness?

A: The only thing I can think is that in some of those tests we may have had a little bit of confounding data in that the higher the rate the more the inertia of those actual pins that were sticking out could wobble. And we did see quite a bit of wobble in the higher

G tests, just because those pins have mass themselves. It could cancel out, sort of superposition from test to test.

Q: Could that also be partially an explanation for why you had more variability in each case?

A: Yes, I think so. Definitely.

Q: Have you thought of maybe trying to mimic muscles in some way just to see what their effect might be? Obviously, you are not going to be able to duplicate it, but either running straps?

A: Cables or straps. Actually that is a great idea. We haven't done that in the laboratory. We performed an initial experiment a long time ago where we did try to keep the musculature and try and keep everything intact. And we thought that just the passive musculature might contribute but passive musculature doesn't contribute in whiplash. So, it would need to be some sort of active system or strap which were taut.

Q: Something that had some level of force on it?

A: Yes.

Q: Okay. Thank you.