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Multiplanar Cervical Spine Injury Due to Head-Turned Rear Impact

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Study Design. Head-turned whole cervical spine model was stabilized with muscle force replication and subjected to simulated rear impacts of increasing severity. Multiplanar flexibility testing evaluated any resulting injury.

Objectives. To identify and quantify cervical spine soft tissue injury and injury threshold acceleration for head-turned rear impact, and to compare these data with previously published head-forward rear and frontal impact results.

Summary of Background Data. Epidemiologically and clinically, head-turned rear impact is associated with increased injury severity and symptom duration, as compared to forward facing. To our knowledge, no biomechanical data exist to explain this finding.

Methods. Six human cervical spine specimens (C0–T1) with head-turned and muscle force replication were rear impacted at 3.5, 5, 6.5, and 8 *g*, and flexibility tests were performed before and after each impact. Soft tissue injury was defined as a significant increase ($P < 0.05$) in intervertebral flexibility above baseline. Injury threshold was the lowest T1 horizontal peak acceleration that caused the injury.

Results. The injury threshold acceleration was 5 *g* with injury occurring in extension or axial rotation at C3–C4 through C7–T1, excluding C6–C7. Following 8 *g*, 3-plane injury occurred in extension and axial rotation at C5–C6, while 2-plane injury occurred at C7–T1.

Conclusions. Head-turned rear impact caused significantly greater injury at C0–C1 and C5–C6, as compared to head-forward rear and frontal impacts, and resulted in multiplanar injuries at C5–C6 and C7–T1.

Key words: cervical spine, rear impact, head turned, instability, flexibility testing. **Spine 2006;31:420–429**

Soft tissue injuries of the cervical spine due to whiplash may cause chronic head and neck pain.[1,2] In a 5-year retrospective study of 11,000 patients with cervical spine injury, 87.4% had soft tissue injuries, while only 12.6% had cervical fractures or luxation.[3] In addition, more than 25% of the patients reported symptoms more than 5 years following whiplash. Clinical symptoms commonly include cranial nerve irritation, neck and upper-extremity pain, restricted neck motion, and paresthesias.[4–9]

In several clinical studies, rotated head posture at the time of rear impact was associated with more severe neck injury and longer duration of symptoms as compared to head forward.[10–13] An epidemiologic survey of 163 occupants of rear-end collisions showed that those with their head turned at rear impact were at higher risk for sustaining injuries with symptoms lasting longer than 3 months, as compared with those facing forward.[10] A year-long prospective cohort study of 117 whiplash patients investigated the effect of impact direction, presence of a head restraint, state of preparedness, and head posture on the cervical spine injury severity.[11,12] Of all the variables studied, only rotated head posture at the time of impact was correlated with chronic symptoms. Finally, a study of 80 patients with whiplash showed that rotated head posture caused significantly higher neck pain intensity, reduced function in daily activities, prolonged incapacity, and reduced neck mobility, as compared with those facing forward.[13]

Pre- and postimpact flexibility testing has been used to determine, *in vitro*, the injury severity and injury threshold acceleration due to simulated head-forward rear and frontal impacts.[14–16] Increases in the intervertebral flexibility parameters of neutral zone or range and motion due to the impacts enabled quantification of mechanical spinal instability due to soft tissue injury. Using the whole cervical spine model with muscle force replication, Ito *et al*[16] found that 5 *g* was the injury threshold acceleration due to rear impact with head forward, as determined by a significant increase in the C5–C6 extension neutral zone. At 6.5 *g*, soft tissue injury was detected at C4–C5 and C5–C6, while C0–C1, C4–C5, C5–C6, and C7–T1 were injured at 8 *g*. Using a similar methodology in frontal impacts, Pearson *et al*[15] observed that the injury threshold acceleration was 8 *g* due to significant increases in the C4–C5 and C6–C7 flexion neutral zones. Following 10 *g*, injury was documented at C2–C3 through C7–T1, excluding C5–C6.

To our knowledge, no previous studies have quantified the mechanical instability of the cervical spine caused by simulated head-turned rear impact. Such data may provide a biomechanical explanation of greater injury severity due to head-turned rear impact, as compared to head-forward, as documented in clinical and epidemiologic studies. Identification of the injury modes

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and intervertebral levels injured may assist automotive engineers in designing injury prevention systems, and clinicians in developing improved diagnostic and treatment protocols. The purpose of this study was to identify, quantify, and determine the mode of cervical spine injury sustained during head-turned rear impact, as well as the injury threshold acceleration using a biofidelic whole cervical spine model with muscle force replication. A second goal was to compare the present data with previously published head-forward rear[16] and frontal impact[15] results.

Methods

Overview. Soft tissue injury and the injury threshold acceleration are defined, and the various steps of the protocol are elucidated (Figure 1). The whole cervical spine (WCS) model, together with muscle force replication (MFR) and a surrogate head, were designated the WCS+MFR model and used to provide a biofidelic kinematic response to head-turned rear impact loading. The WCS model, without the MFR, was used in 3-plane flexibility testing to document the soft tissue injury. Baseline flexibility data were obtained after dynamic preconditioning (a noninjurious 2 *g* acceleration). After each subsequent impact, flexibility testing was repeated to determine the soft tissue injury, if any. All specimens underwent fluoroscopic examination following trauma to identify bony fractures, if any.

Specimen Preparation. Six fresh-frozen human osteoligamentous WCS specimens (occiput–T1) were mounted in resin (Polyester Fiberglass Resin; Bondo Corp., Atlanta, GA) at the occiput and T1 such that the foramen magnum was parallel to the occipital mount, and the T1 vertebra was tilted anteriorly by 24°.[17] The average age of the specimens was 80.2 years (range 79–93), and there were 4 male and 2 female donors. Apart from typical age-related degenerative changes, the donors had not had head or neck trauma and did not have any disease that could have affected the osteoligamentous structures. To attach motion-measuring flags, a custom plastic support was fitted rigidly onto the anterior aspect of each vertebra (C1 to C7). The flags, each with 3 noncollinear markers, were rigidly fixed onto the plastic supports. Additional flags were rigidly mounted to the occipital and T1 mounts. A lateral radiograph of the specimen in the neutral posture, with motion tracking flags, was taken to establish anatomic coordinate systems fixed to each vertebra.

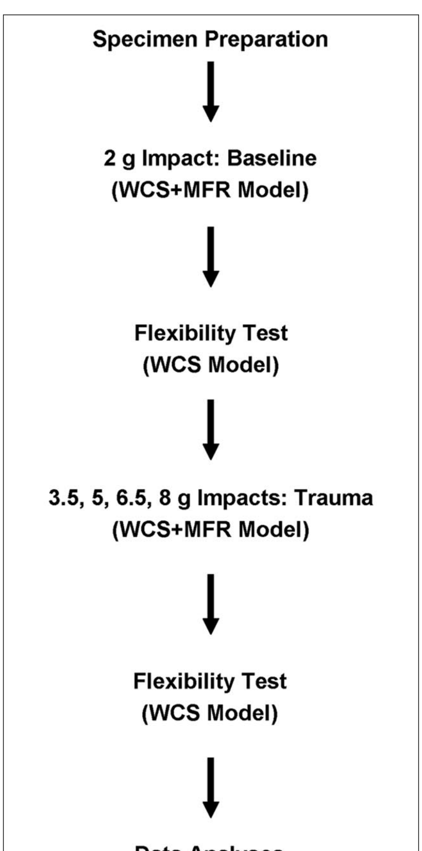


Figure 1. Experimental protocol showing various steps.

WCS Model Preparation for 3-Plane Flexibility Testing. A loading jig was applied to the occipital mount, while the T1 mount was fixed to the test table. The weights of the loading jig and occipital mount were counterbalanced during flexibility testing.

WCS+MFR Model Preparation for Head-Turned Rear Impact. To prepare the specimen for impact, a custom surrogate head (mass 3.3 kg and sagittal, horizontal, and frontal plane moments of inertia of 0.019, 0.014, 0.015 kg m^2, respectively)[18,19] was rigidly attached to the occipital mount. The surrogate head and spine were stabilized using the compressive MFR system.[15,16] The MFR system was symmetric about the midsagittal plane, and consisted of anterior, posterior, and lateral cables attached to preloaded 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. The 2 anterior cables, each consisting of 2 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, wire loops were inserted into the spinal canal through the laminae (C2 to C7) and tightly secured above each vertebral spinous process. The 2 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, preloaded to 15 N. Bilateral cables originated at 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 preloads in the neutral posture 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).

Before impact, the head was rotated such that the average (standard deviation) head-T1 rotation relative to the neutral posture was 28.4° (5.3°) of left axial rotation, 17.9° (4.7°) of left lateral bending, and 3.5° (3.7°) of flexion.

Three-Plane Flexibility Testing. Anatomic coordinate systems were established to determine the motion of each vertebra relative to the adjacent inferior vertebra (Figure 2). The origins of the 3-dimensional coordinate systems were positioned in the midsagittal plane, and fixed to the posteroinferior corner of each vertebral body for C2 through T1 and to the posterior border of the posterior arch of C1. The positive x-axis was directed to the left and was perpendicular to the midsagittal plane. The positive y-axis was oriented superiorly, and the positive z-axis was oriented anteriorly through the anteroinferior corner of each vertebral body for C2 through T1, and through the anterior border of the anterior arch for C1. The yz, xz, and xy axes defined the sagittal, transverse, and frontal planes, respectively.

Pure moments were applied to the occipital mount of the WCS specimen in 4 equal steps up to peak pure moments of 1.5, 3, and 1.5Nm in flexion-extension, axial torque, and lateral bending, respectively (Figure 3).[20] To minimize viscoelastic effects, 30-second wait periods were given following each load application. Two preconditioning cycles were performed, and data were recorded on the third loading cycle. A custom-built loading apparatus, motion monitoring system, and customized software were integrated for automated flexibility testing. Flexion-extension flexibility data were recorded using 2 digital motion analysis cameras (MotionPro, Redlake; MSAD Inc., San Diego, CA). Mean error for sagittal rotation was 0.07° (SD 0.12°). Flexibility data during axial rotation and lateral bending tests were measured using the Optotrak 3-dimensional motion measuring system (Northern Digital, Waterloo, Ontario, Canada). Mean error for rotations within a 24° measurement range was −0.014° (SD 0.14°).[21] The flexibility data were used to calculate the intervertebral Euler angles at each load increment for the occiput relative to C1, and for each vertebra (C1 through C7) relative to, and in the coordinate system of the directly inferior vertebra, in the sequence Rx,

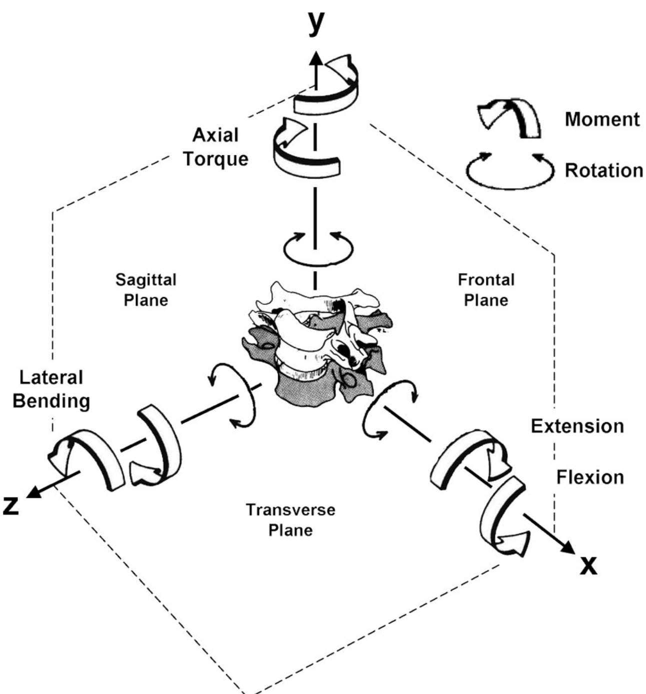


Figure 2. The 3-dimensional coordinate system fixed to a moving vertebra. The coordinate system was established to determine the motion of each vertebra relative to the directly inferior vertebra. The origins were fixed to the posteroinferior corner of each vertebral body, C2 through T1, and to the posterior border of the posterior arch of C1. The positive x-axis was directed to the left and was perpendicular to the midsagittal plane. The positive y-axis was oriented superiorly, and the positive z-axis was oriented anteriorly through the anteroinferior corner of each vertebral body for C2 through T1, and through the anterior border of the anterior arch for C1. The yz, xz, and xy axes defined the sagittal, transverse, and frontal planes, respectively. The broad arrows illustrate pure moments, while the thin circular arrows show the rotations in flexion, extension, axial rotation, and lateral bending.

followed by Ry and Rz. For each intervertebral level,9 neutral zones and 9 ranges of motion (ROMs) were determined (Table 1).

Head-Turned Rear Impact Simulation. Head-turned rear impact simulation was performed using a previously developed bench-top sled apparatus.[22] The incremental trauma protocol was used to impact the WCS+MFR model at nominal T1 horizontal accelerations of 3.5, 5, 6.5, and 8 g.[23] The 2 high-speed digital cameras (MotionPro, Redlake) recorded the 3-dimensional motion of each vertebra immediately following impact at 500 frames/s.

Definitions

- Soft tissue injury: A statistically significant increase ($P < 0.05$) in any of the 18 flexibility parameters (Table 1) due to the impact as compared to the corresponding baseline values.
- Two-plane injury: The soft tissue injury occurring in any 2 planes.
- Three-plane injury: The soft tissue injury occurring in 3 planes.
- Injury threshold: The lowest T1 horizontal peak acceleration that caused the soft tissue injury at any intervertebral level.

Data Analyses

Injury Potential. The injury potential (IP) was used for graphic visualization of each of the normalized flexibility parameters of Table 1.[14–16] The IP was defined as the relative percentage increase in the intervertebral flexibility following each impact ($\mathrm{Flex}_{\mathrm{Impact}}$), above the corresponding 2 g baseline value (Flex_{2g}), and was calculated for each specimen and averaged for the group.

$$\mathrm{IP}\ (\%) = 100\left(\frac{\mathrm{Flex}_{\mathrm{Impact}} - \mathrm{Flex}_{2g}}{\mathrm{Flex}_{2g}}\right)$$

Thus, an IP of 100% indicated that impact caused the intervertebral flexibility to be double that of its 2 g baseline value.

To quantify the multiplanar injuries and compare injury severity among various impact configurations of previous studies,[15,16] a 3-plane IP function was calculated for all injured intervertebral levels.

$$\text{Three-Plane IP} = +\sqrt{(\mathrm{IP}_{\mathrm{Sagittal}})^2_{\max} + (\mathrm{IP}_{\mathrm{Transverse}})^2_{\max} + (\mathrm{IP}_{\mathrm{Frontal}})^2_{\max}}$$

where $\mathrm{IP}_{\mathrm{Sagittal}}$, $\mathrm{IP}_{\mathrm{Transverse}}$, and $\mathrm{IP}_{\mathrm{Frontal}}$ each represented the maximum IP value, either NZ or ROM, at which injury was detected.

Statistics. Single factor, repeated measures analysis of variance ($P < 0.05$) and Bonferonni *post hoc* tests were used to determine significant increases in the intervertebral flexibility parameters (NZ and ROM) above the corresponding 2 g baseline values. To compare the present results to those obtained from previously reported head-forward rear[16] and frontal impact[15] data, 3-plane IPs were compared for each injured intervertebral level, using single factor, nonrepeated measures analysis of variance ($P < 0.05$) and pairwise Bonferonni *post hoc*

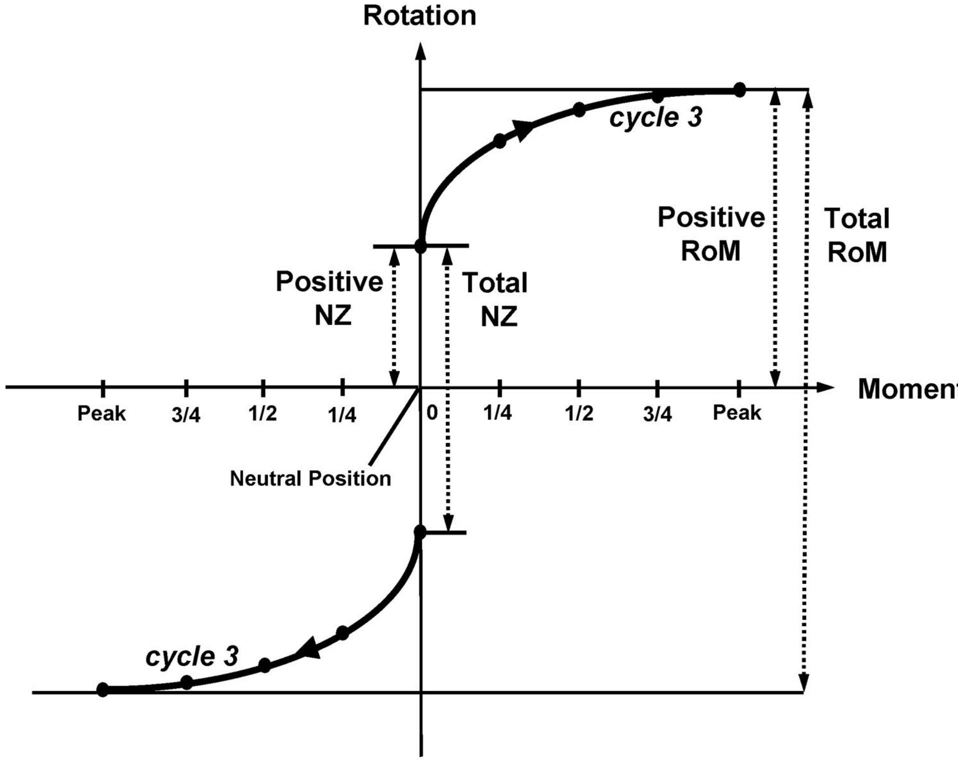


Figure 3. Flexibility testing protocol in which peak pure moments of 1.5, 3, and 1.5 Nm in flexion-extension, axial torque, and lateral bending, respectively, were applied in 4 equal steps, while motion data were collected on the third loading cycle. Flexibility parameters of neutral zone (NZ) and range of motion (RoM) were determined for each intervertebral level.

tests. Adjusted *P* values were computed based on number of *post hoc* tests performed.

■ Results

The average measured T1 horizontal acceleration peaks differed by at most 0.3 *g* from the corresponding nominal maximum accelerations (Table 2). The average duration of the T1 horizontal acceleration pulse ranged between 85.3 and 94.2 milliseconds during the 8 *g* and 3.5 *g* impacts, respectively. The peak T1 horizontal velocity was 10.4 kph during the 8 *g* impact.

Fluoroscopic examination of the specimens following trauma revealed no bony fractures.

All pre- and post impact NZ and ROM data are presented in tabular form in which averages, SDs, and significant increases with respect to 2 *g* baseline values are given for each intervertebral level (C0–C1 through C7–T1) (Tables 3–8 available for viewing on ArticlePlus only). The injury threshold acceleration was 5 *g*, as evidenced by significant increases above the 2 *g* baseline in the C5–C6 and C7–T1 extension NZs (Figure 4A) and in the C3–C4 through C5–C6 total axial rotation ROMs (Figure 5B). Following 8 *g*, significant increases occurred in the extension NZs at C5–C6 through C7–T1, ranging between 72.8% and 125.3% (Figure 4A), total axial rotation NZs at C4–C5 and C5–C6 (Figure 5A), and right lateral bending neutral zone at C5–C6 (Figure 6A). Corresponding significant increases at 8 *g* occurred in the extension ROMs at C5–C6 and C7-T1 (Figure 4B), axial rotation ROMs at C0–C1 and C3–C4 through C7-T1, excluding C6–C7 (Figure 5B), and lateral bending ROM at C5–C6 (Figure 6B).

Following 8 *g*, 3-plane injury was observed at C5–C6 in extension, axial rotation, and lateral bending, while 2-plane injury was documented at C7–T1 in extension and axial rotation. Injury was also documented at C0–C1, C3–C4, C4–C5 in axial rotation and C6–C7 in ex-

Table 1. The 18 Flexibility Parameters at Each Intervertebral Level Consisted of Neutral Zones and Ranges of Motion in Each of the 3 Planes of Motion

Measured Parameters	Sagittal Rotation	Axial Rotation	Lateral Bending
NZ	Flexion	Right	Right
NZ	Extension	Left	Left
NZ	Total (flexion+extension)	Total (right+left)	Total (right+left)
RoM	Flexion	Right	Right
RoM	Extension	Left	Left
RoM	Total (flexion+extension)	Total (right+left)	Total (right+left)

NZ indicates neutral zone; RoM, range of motion.

Table 2. Average (SD) Nominal and Measured Peak T1 Horizontal Accelerations (1 g = 9.81 m/s^2), Acceleration Pulse Durations, and Peak Velocities Obtained During Head-Turned Rear Impact

Nominal Peak T1 Horizontal Acceleration (*g*)	Measured Peak T1 Horizontal Acceleration (*g*)	Measured T1 Horizontal Acceleration Pulse Duration (milliseconds)	Measured Peak T1 Horizontal Velocity (kph)	Measured Peak T1 Horizontal Velocit (mph)
3.5	3.3 (0.2)	94.2 (3.2)	5.1 (0.8)	3.1 (0.5)
5	4.7 (0.2)	94.8 (6.1)	8.0 (0.9)	5.0 (0.6)
6.5	6.3 (0.3)	90.2 (7.9)	9.9 (1.4)	6.2 (0.8)
8	8.1 (0.3)	85.3 (2.4)	10.4 (0.7)	6.4 (0.4)

tension. Thus, although the principle mode of injury was extension, the injuries also occurred due to coupled axial rotation, and lateral bending.

Discussion

Epidemiologic studies have documented an increased risk of incurring severe and chronic injuries in rear impact, with head turned as compared to facing forward.[10,11] The current study, using the head-turned WCS model with MFR, identified the injury threshold acceleration and injured intervertebral levels by using 3-plane flexibility testing before and after each rear impact (3.5, 5, 6.5, and 8 *g*). Injury was defined at each spinal level as a significant increase in any 1 of the 18 flexibility parameters (Table 1), above its corresponding baseline value. The injury threshold acceleration was *5 g*, with extension injuries occurring at C5–C6 and C7–T1, and axial rotation injuries occurring at C3–C4 through C5–C6. At 8 *g*, injuries occurred throughout the cervical spine from C0–C1 through C7–T1, except C2–C3 and C3–C4. Three-plane injury occurred at C5–C6, while 2-plane injury was documented at C7–T1 in extension and axial rotation. The peak injury potentials (NZ or ROM) after 8 *g* impact showed the 3-plane injury at C5–C6 and the high injury severity in extension at C5–C6 through C7–T1, as compared to axial rotation and lateral bending (Figure 7).

The limitations of this study must be considered before interpreting the present results. The MFR system provided passive resistance to intervertebral motion and did not include the active *in vivo* neuromuscular response, thus simulating the response of an unwarned subject. The specimens of the present study, with an average age of 80.2 years, were weaker than the population most likely to sustain rear impact trauma. To ensure that no specimen was prematurely injured due to excessive head inertia loads and to ensure the validity of the injury threshold acceleration, 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.[18,19] The current study used the incremental trauma approach to determine the soft tissue injury threshold acceleration. A previous study using a porcine cervical spine model showed that the incremental and single trauma protocols produced equivalent subfailure soft tissue injury severity, thus justifying the incremental trauma approach.[23]

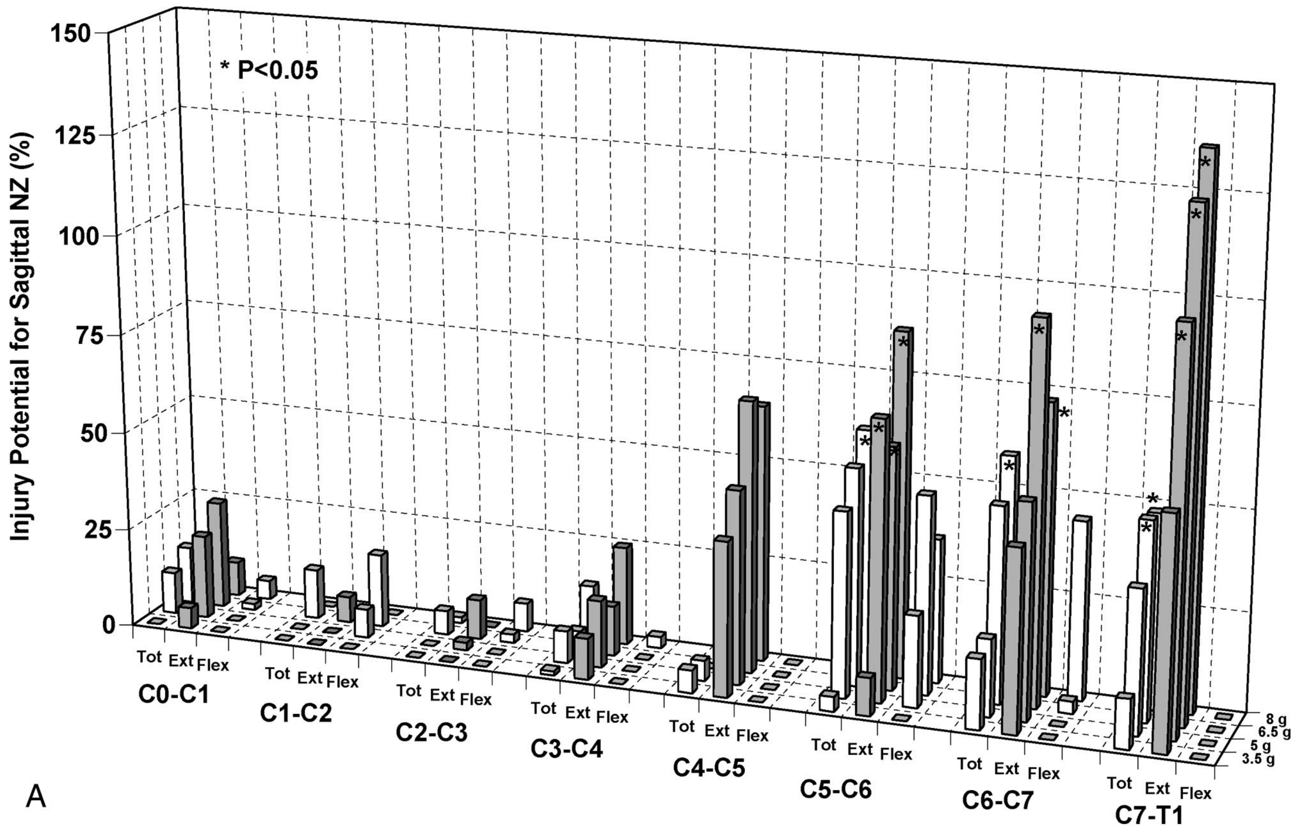
The present IP and injury threshold data, together with the previously reported results of head-forward rear impact[16] and frontal impact[15] studies using similar methodology, provide valuable comparative information regarding potential injury sites, severity, and mechanisms. Multiplanar injury was documented in the present head-turned rear impact study only at C5–C6 and C7–T1, and it did not occur in head-forward rear or frontal impacts. At 8 *g*, the 3-plane IPs at C0–C1 and C5–C6 for head-turned rear impact significantly exceeded ($P < 0.05$) the corresponding head-forward rear impact IPs,[16] while no injury was observed at C0–C1 or C5–C6 in head-forward frontal impact (Figure 8).[15]

These biomechanical findings are supported by an epidemiologic study of 163 occupants involved in rear-end collisions, which analyzed the effect of vehicle type, impact configuration, and occupant seating position on injury severity and duration.[10] It was found that occupants who had their head turned at impact were more than 100% more likely to have injuries lasting longer than 3 months, as compared to forward-facing occupants. Previous clinical studies have also found that rotated head posture at rear impact caused increased frequency, duration, and severity of symptoms, as well as a decrease in active neck mobility, as compared to forward facing.[10–13]

The 3-plane IP at C5–C6 due to the 8 *g* head-turned rear impact significantly exceeded and was more than double the corresponding head-forward rear impact IP (Figure 8).[16] C5–C6 has also been identified as a primary injury site in clinical and epidemiologic studies.[24–26] Yoganandan *et al*[26] reported the highest frequency of injury at C5–C6 in survivors of motor vehicle-related trauma using the National Accident Sampling System. A retrospective analysis of a prospectively collected trauma database of 468 patients with cervical spine injury showed that C5–C6 was associated with the highest proportion of severe injury, followed by C4–C5.[24]

Differences in the injury threshold acceleration among the impact configurations were also observed. The 5 *g* injury threshold acceleration observed for head-forward and head-turned rear impact was lower than the 8 *g* threshold documented for head-forward frontal impact.[15,16] Thus, the soft tissue injuries sustained during rear impact may occur at lower accelerations than during frontal impact, with multiplanar injuries occurring if the head is turned prior to rear impact.

The current study of head-turned rear impact used a biofidelic WCS model with MFR to quantify soft tissue



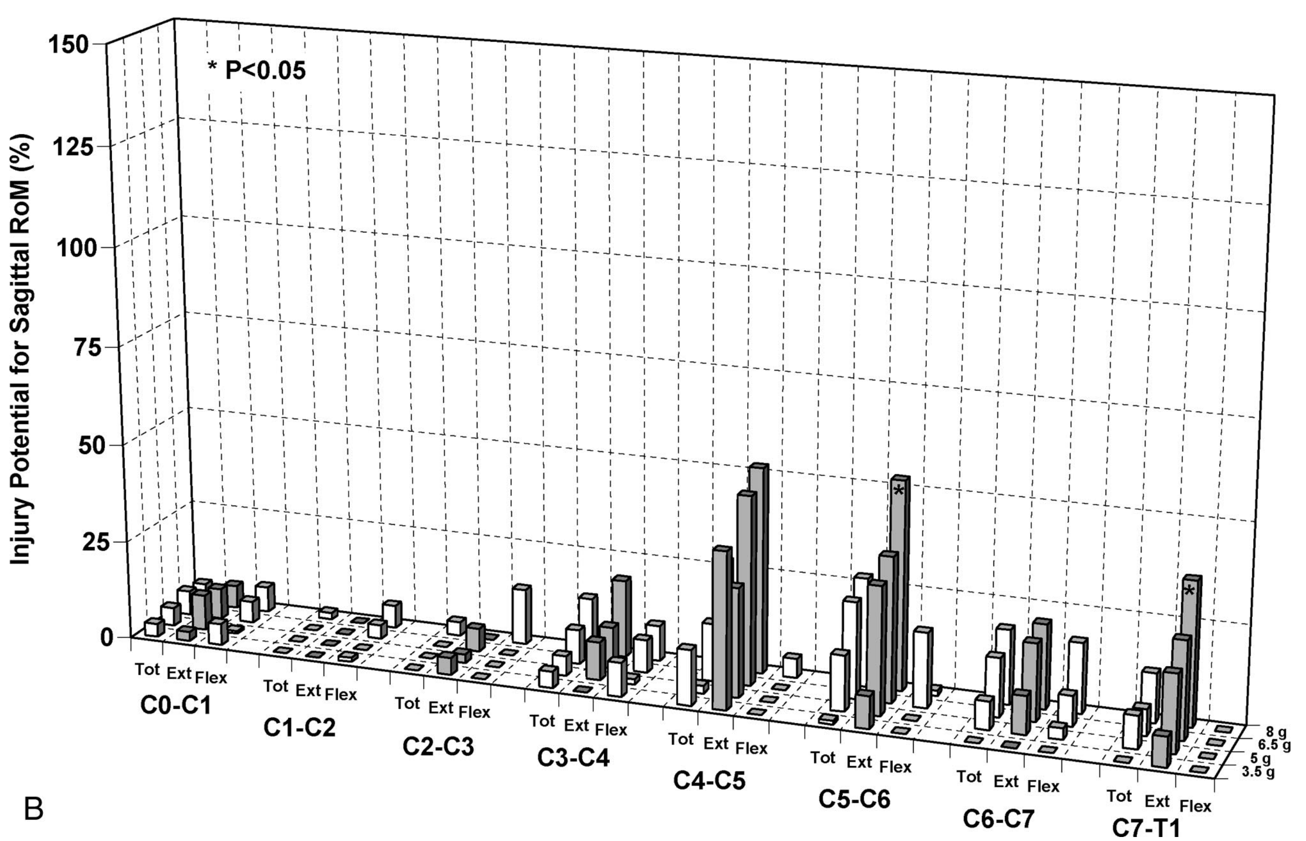
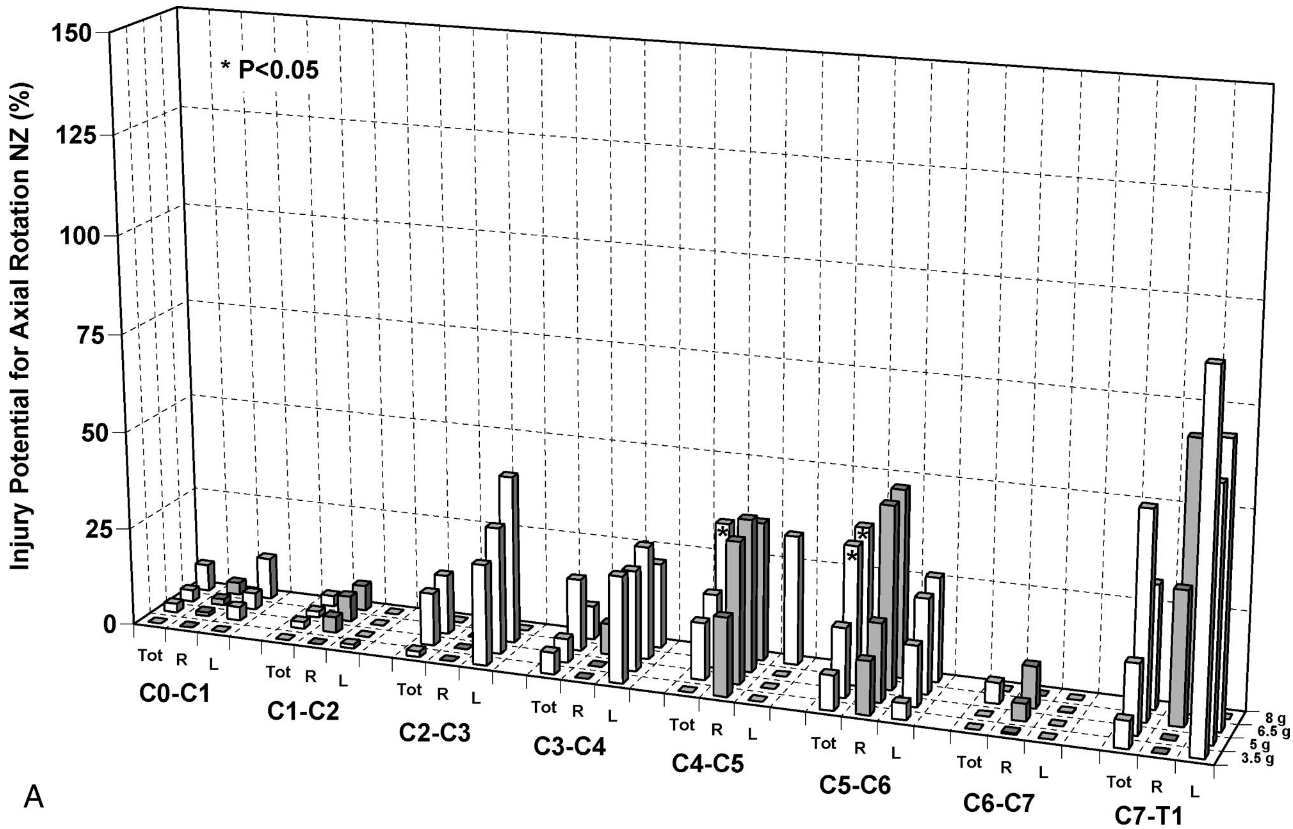


Figure 4. Average injury potentials for sagittal neutral zone (NZ) (**A**) and range of motion (RoM) (**B**) for flexion (Flex), extension (Ext), and total (Tot) (flexion plus extension) for each intervertebral level (C0–C1 to C7–T1) due to impact. The nominal T1 horizontal maximum accelerations were 3.5, 5, 6.5, and 8 *g*. Statistically significant increases ($P < 0.05$) in the NZ and ROM above corresponding 2 *g* baseline values are indicated (*).



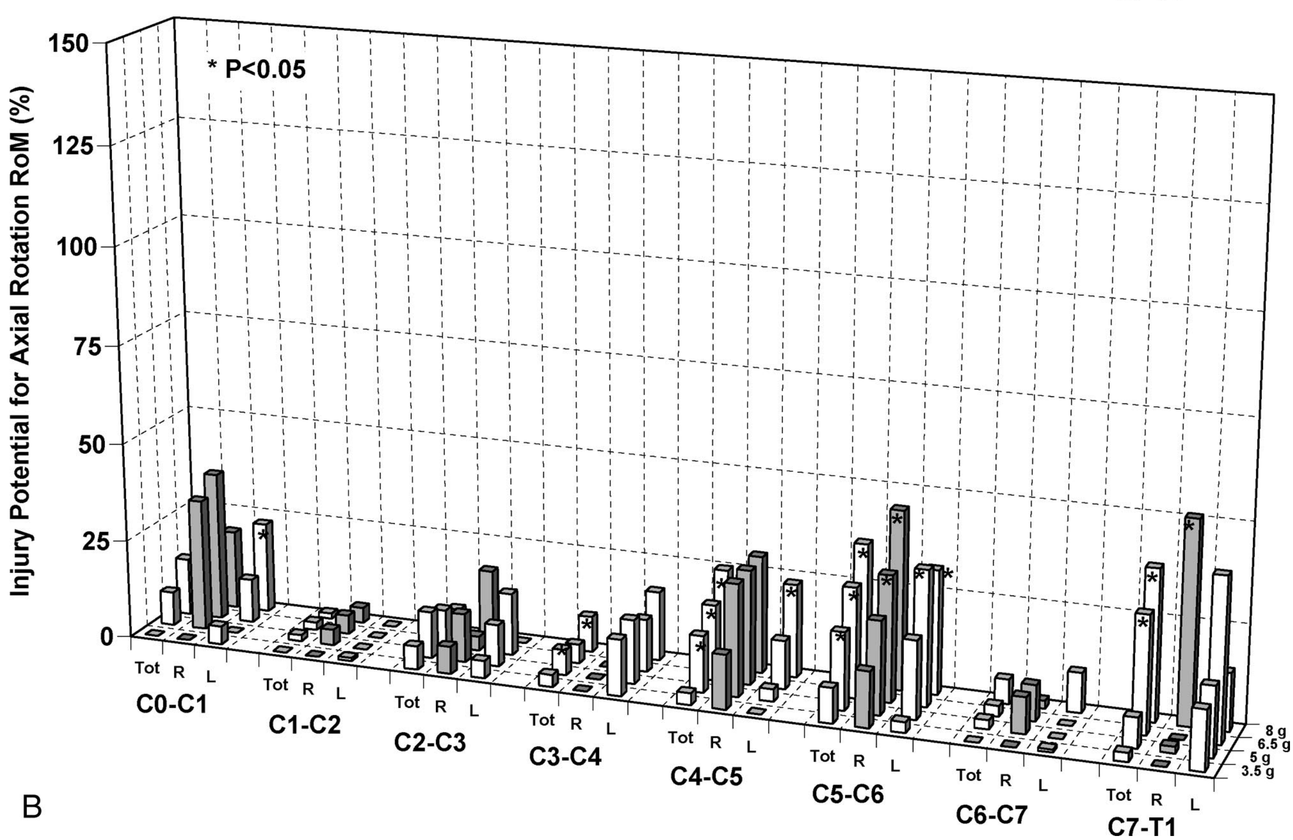


Figure 5. Average injury potentials for axial rotation neutral zone (NZ) (**A**) and range of motion (RoM) (**B**) for left (L), right (R), and total (left plus right) for each intervertebral level (C0–C1 to C7–T1) due to impact. The nominal T1 horizontal maximum accelerations were 3.5, 5, 6.5, and 8 *g*. Statistically significant increases ($P < 0.05$) in the NZ and ROM above corresponding 2 *g* baseline values are indicated (*).

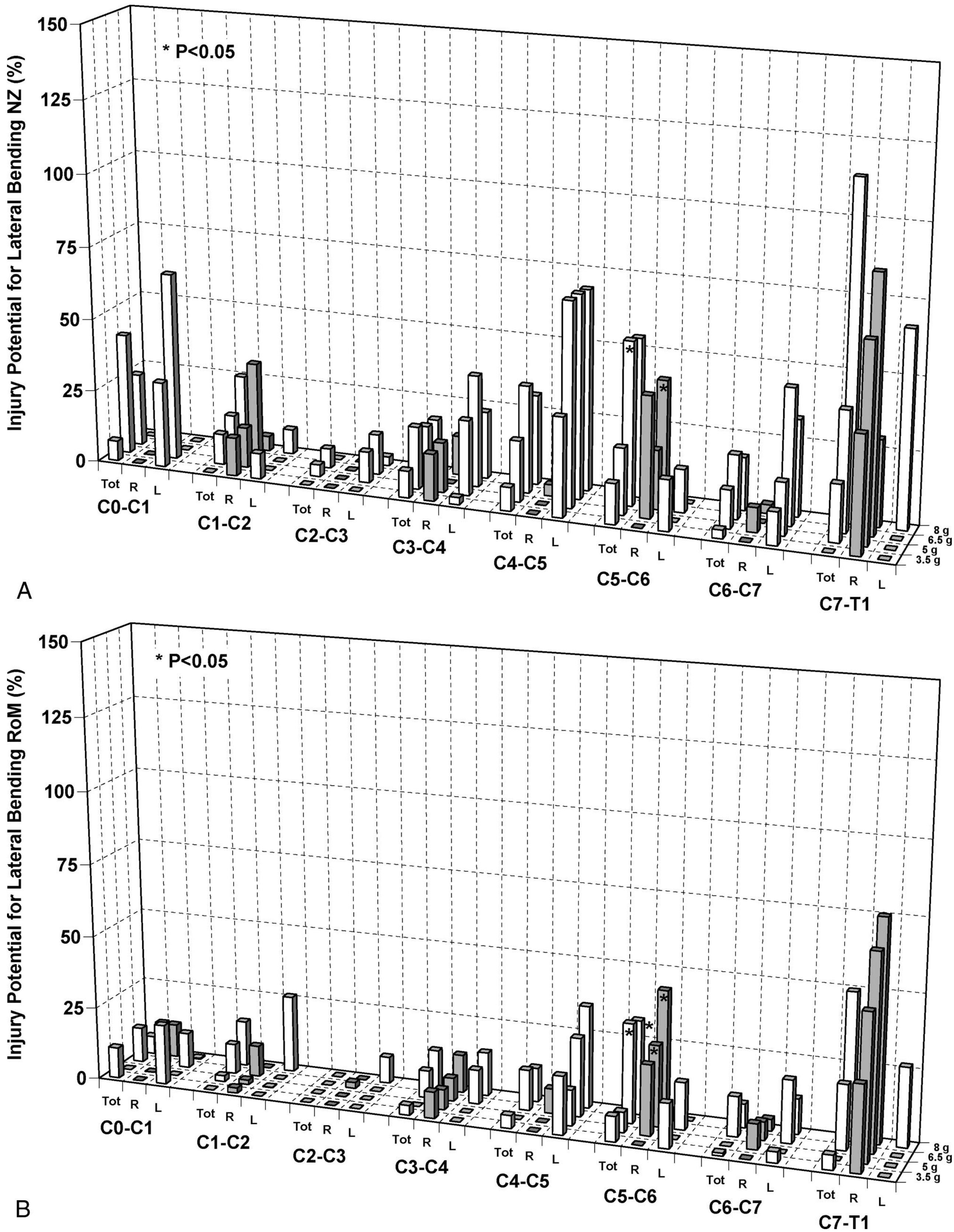


Figure 6. Average injury potentials for lateral bending neutral zone (NZ) (**A**) and range of motion (RoM) (**B**) for left (L), right (R), and total (left plus right) for each intervertebral level (C0–C1 to C7–T1) due to impact. The nominal T1 horizontal maximum accelerations were 3.5, 5, 6.5, and 8 *g*. Statistically significant increases ($P < 0.05$) in the NZ and ROM above corresponding 2 *g* baseline values are indicated (*).

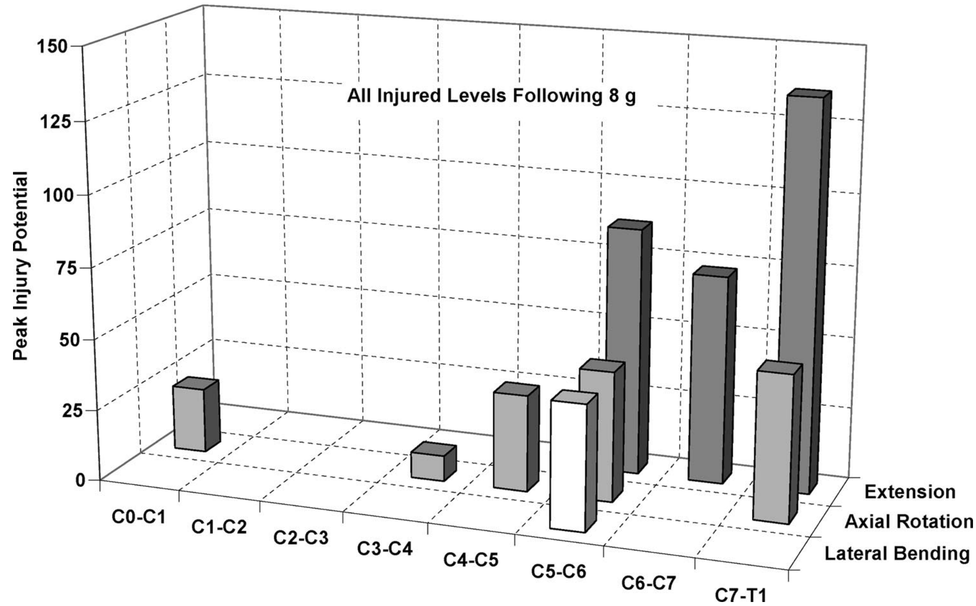


Figure 7. Peak injury potentials for only the intervertebral levels that were injured due to head-turned rear impact, following 8 *g*. Three-plane injury was observed only at C5–C6, while 2-plane injury was documented only at C7–T1. Injury also occurred at C0–C1, C3–C4, and C4–C5 in axial rotation and at C6–C7 in extension.

injuries. The high injury severity observed primarily in extension at C5–C6 through C7–T1 and the 3-plane injury at C5–C6 caused by head-turned rear impact sharply contrasted with the single-plane injury and lower injury severity reported in previous head-forward rear and frontal impact studies using similar methodology. These biomechanical findings are supported by clinical observations of increased frequency and severity of whiplash-related symptoms, when the head was rotated as compared to forward facing, at the time of rear impact.[10–13] The high injury severity at C5–C6 caused by both head-turned and head-forward rear impact is supported by clinical studies, which have found that C5–C6 was most prone to injury in automobile collisions.[24–26] Thus, these data indicate that occupants involved in rear impacts are at increased risk for severe injury, primarily at C5–C6, when the head is turned as compared to head-forward at the time of impact. The extension injuries due

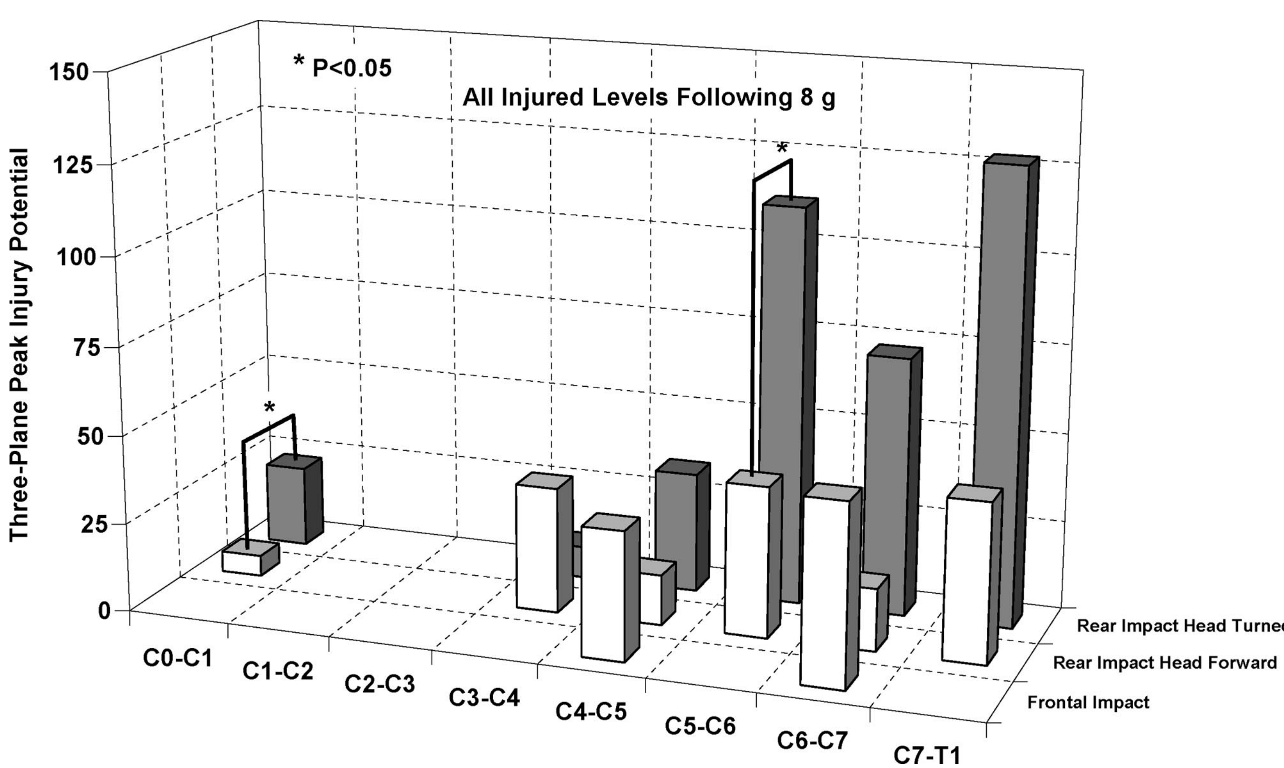


Figure 8. Three-plane injury potentials for only the injured intervertebral levels due to head-turned rear impact and head-forward rear and frontal impacts, following 8 *g*. The 3-plane injury potentials at C0–C1 and C5–C6 for head-turned rear impact significantly exceeded ($P < 0.05$) the corresponding rear impact head-forward injury potentials, while no injury was observed in frontal impact. Statistically significant pairwise increases among impact configuration are indicated for each intervertebral level (*).

to head-forward rear impact suggest the onset of subfailure injury to the anterior longitudinal ligament and anterior anular fibers, and associated facet joint impingement, while the flexion loading during frontal impact may injure supraspinous and interspinous ligaments and ligamentum flavum. In contrast, head-turned rear impact may cause lateral anular fiber and capsular ligament injuries, in addition to the head-forward rear impact injuries. The present finding may provide valuable information to the clinician for diagnosing whiplash-type injuries based on the specific impact configuration.

■ Key Points

- Cervical spine soft tissue injury, injury severity, and injury threshold acceleration were determined for the head-turned WCS model with MFR due to rear impacts of 3.5, 5, 6.5, and 8 *g*.
- Soft tissue injury was defined as a significant increase ($P < 0.05$) in any neutral zone (NZ) or range of motion (ROM) above corresponding 2 *g* baseline values. Injury threshold was defined as the acceleration that caused the injury at any intervertebral level.
- The injury threshold was 5 *g*. Injuries occurred in extension NZs or axial rotation ROMs at C3–C4 through C7–T1, excluding C6–C7.
- Following 8 *g*, 3-plane injury occurred at C5–C6, while 2-plane injury occurred at C7–T1 in extension and axial rotation. Injuries also occurred at C0–C1, C3–C4, and C4–C5 in axial rotation and at C6–C7 in extension.
- Head-turned rear impact caused significantly greater injury at C0–C1 and C5–C6, compared to head-forward frontal and rear impact.

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