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Whiplash injuries and the potential for mechanical instability

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Abstract Whiplash injury to the cervical spine is poorly understood. Symptoms often do not correlate to the clinical findings. It has been hypothesized that the long-term clinical symptoms associated with whiplash have their basis in mechanical derangement of the cervical spine caused at the time of trauma. Before such a hypothesis can be proven, one needs to document and quantify the soft tissue injuries of the cervical spine in whiplash. The purpose of the study was to quantify the mechanical changes that occur in the cervical spine specimen as a result of experimental whiplash trauma. Utilizing a whiplash trauma model, injuries to human cadaveric cervical spine specimens (C0 – T1 or C0 – C7) were produced by increasingly severe traumas. The flexibility tests determined the motion changes at each intervertebral level in response to 1.0 Nm pure flexion-extension moment. Parameters of range of motion (ROM) and neutral zone (NZ) were determined before and after each trauma. Significant flexibility increases first occurred in the lower cervical spine after 4.5–g rear-end (anteriorly directed) acceleration of the T1 vertebra. At this acceleration magnitude, extension ROM and NZ at C5 – C6 increased ($P < 0.05$) by 98% and 160% respectively. There was also a tendency ($P < 0.1$) for the extension NZ at C0 – C1 and C6 – C7 levels to increase after the 6.5-g acceleration by 52% and 241% respectively. There were no such tendencies for the ROM parameter. We have identified the threshold and sites of whiplash injury to the cervical spine. This information should help the clinician make more precise diagnoses in the case of whiplash trauma patients.

Key words Whiplash · Biomechanics · Spinal instability · Cervical spine · Spine injury

Introduction

Whiplash injury to the neck is one of the most poorly understood disorders of the vertebral column [4]. The severity of the whiplash trauma often does not correlate with the seriousness of the clinical symptoms, which include neck and shoulder pain, headache, dizziness, and blurring of vision. This incongruity often leads to undefined diagnoses [2, 10, 31]. The most recent scientific monogram by the Quebec Task Force on Whiplash Associated Disorders identified very few objective studies that have documented specific spinal lesions associated with whiplash [32].

In a survey of 10,000 cases of cervical spine injuries, the symptoms of pain and headache were present in 25% of the cases 5 years after the accident [9]. In a longer follow-up, 28% of the whiplash victims complained of intrusive symptoms and another 12% suffered from severe problems 10.3 years after the trauma [10]. Eighty-six percent of the patients were believed to have suffered soft tissue injuries. In another study conducted prospectively,

only 38% of the whiplash patients had recovered at their 2-year follow-up [11]. There is a consensus that most whiplash patients suffer soft tissue injuries [8, 16, 19, 20]. Patients with more severe injuries show signs of clinical instability [16], determined using accepted instability criteria [34]. In patients with milder soft tissue injuries, these injuries may go undetected [16], as they are not completely identified by the presently avaiable imaging methods [16]. These subfailure soft tissue injuries may explain the decreased function and pain associated with whiplash trauma. With time, such symptoms may become chronic and result in clinical instability. Therefore, for better understanding of such injuries, there is a need for the study of mechanical derangement of the ligamentous structures associated with whiplash trauma.

We defined functional injury to the cervical spine as a significant increase in its flexibility. The purpose of our in vitro experimental investigation was to quantitate the changes in the intervertebral mechanical flexibility of the cervical spine at all levels due to simulated whiplash trauma, using fresh cadaveric human cervical spine specimens. The injury to the cervical spine was studied by performing intervertebral flexibility tests on intact specimens before and after whiplash trauma of increasing severity.

Materials and methods

Specimen preparation

Eight fresh-frozen human cadaveric cervical spine specimens were studied. Four specimens were occiput to T1; and four were occiput to C7. The specimens were cleaned of all non-ligamentous soft tissue with care so as not to damage osteoligamentous structures. All specimens were radiographed to find any damage or abnormality, beyond the normal degenerative changes. None were found. Each specimen was placed in a neutral posture, and the occiput and T1 (or C7) vertebrae were rigidly secured with screws and embedded in resin mounts (Dynatrom/Bondo, Atlanta, Ga). The specimen was oriented so that the foramen magnum was horizontal and the C6 vertebra was at a 20° anterior tilt, simulating normal spine neutral posture [5].

Flexibility testing

Flexiblity testing was done to characterize the mechanical properties of the cervical spine specimen as previously described [26]. Motion monitoring flags made of Plexiglas were secured to the vertebral body flag mounts with screws (Fig. 1). Each flag had three non-colinear infrared-emitting diodes designed for the Optotrak three-dimensional-motion measurement system (Northern Digital, Waterloo, Canada). A headpiece, was secured to the upper specimen mount. The weights of the headpiece and upper mount were balanced by a counter-force applied with a hung mass via a flexible cable over a pulley system. The flexibility system was designed to apply pure moments to the specimen in an unconstrained manner via a system of low-friction pneumatic cylinders, linear bearings, and pulleys [27]. The accuracy of the flexibility measurements has been previously determined [18]. The mean (SD) of the error for rotations within a 24° measurement range was –0.014° (0.14°).

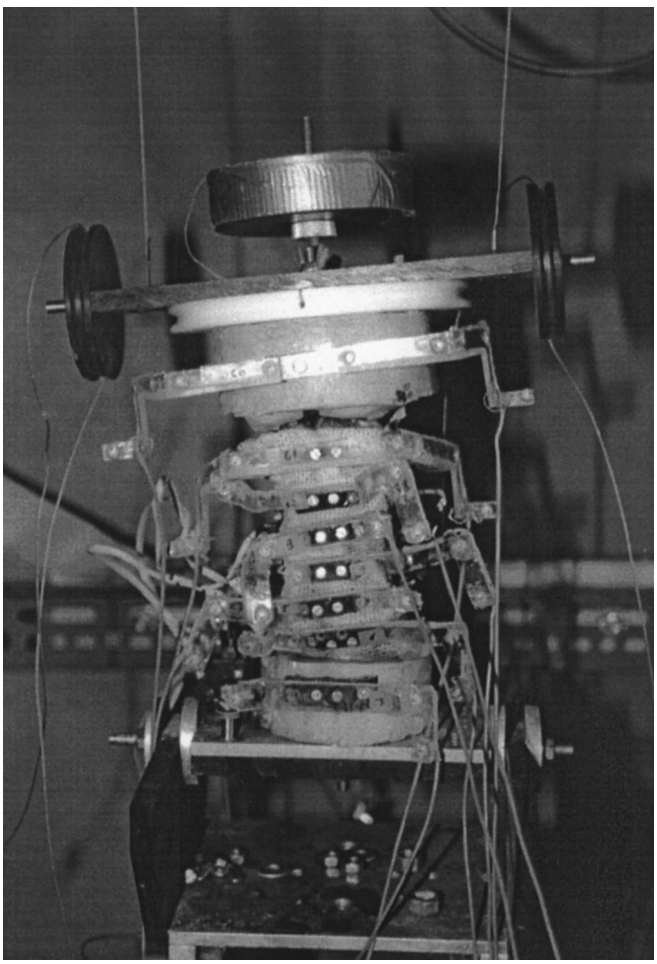


Fig. 1 Photograph of cervical spine specimen in the flexibility testing machine. Pure moments were applied to the headpiece, which was fixed to the upper specimens mount. Individual intervertebral motions were measured with infrared-emitting diodes attached to the detection flags using an optoelectronic three-dimensional-motion measurement system

The specimen was loaded with pure moments in a stepwise fashion in three equal steps up to 1.0 Nm in flexion and extension. The maximum moment of 1.0 Nm was defined as the limit of physiologic loading that did not result in injury by preliminary experiments of repeatability. A 30-s creep was allowed after each load step to minimize the viscoelastic effects of the spine specimens. The specimen was preconditioned, i.e., loaded and unloaded twice. Third cycle load-displacement curves were recorded and used for further analysis (Fig. 2). From the load-displacement curves, flexibility parameters, i.e., range of motion (ROM) and neutral zone (NZ), were determined for flexion, extension, and total (flexion plus extension) motions. Although the elastic zone (EZ) was computed, it was not analyzed, as it is not an independent parameter.

Injury determination

Soft tissue injuries, especially less severe ones, are difficult to detect and quantify. We use the flexibility test to quantify the multidirectional mechanical properties at each spinal level. We define

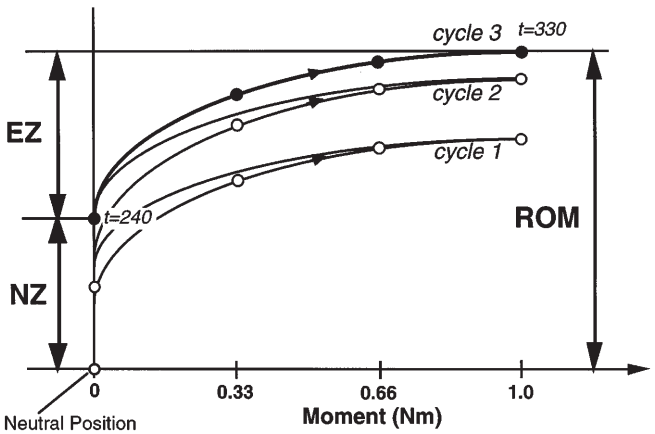


Fig. 2 Schematics of the flexibility test protocol for flexion and extension. Maximum moment of 1.0 Nm was applied in three equal steps and for three load cycles. Motions were measured on the third load cycle at each load step after 30 s wait. From the angle-moment curve, flexibility parameters of range of motion (ROM) and neutral zone (NZ) were determined

Injury Potential as the relative increase in a flexibility parameter after the trauma, when compared to the corresponding intact value. Thus, the flexibility testing was performed when the specimen was intact and after each incremental trama. The Injury Potentials for a flexibility parameter were mathematically given by:

Injury Potential (%) = 100* ($Flex_{Inj}$ – $Flex_{Int}$)/$Flex_{Int}$

where $Flex_{Inj}$ and $Flex_{Int}$ are respectively the flexibilities when injured and intact. Thus, for example, an Injury Potential of zero represents no injury, i.e., intact specimen, and an Injury Potential of 100% indicates that the injured specimen has twice the flexibility of its intact state.

Whiplash trauma model

The apparatus

The whiplash trauma was produced with a specially developed trauma apparatus [29]. The major components of this apparatus are the following (Fig. 3). The trauma sled was mounted on horizontal linear bearings. The acceleration producing system, seen on the left of Fig. 3 consisted of a pneumatic piston, power springs, and an electromagnet release. A brake was positioned at the other end. The lower end of the spine specimen was attached to the sled while the upper end carried a head surrogate.

The surrogate head was made of steel and designed to represent an average human head with mass of 5.5 kg and moment of inertia of 0.035 kg m^2 in the sagittal plane [21]. The weight of the top mount of the specimen and the surrogate head was fully balanced by a low-friction pneumatic suspension system, leaving unaffected the inertial effects of the head surrogate mass. The suspension was such that the head surrogate was completely free to move within its three degrees of freedom in the sagittal plane. Both the balance and the freedom of motion of the spine were fully effective even during the trauma. A head stop was empirically set at a 45° angle so that the natural extension of the head led to a perpendicular contact of the head surrogate and head stop.

Once the specimen was mounted in neutral posture on the sled, the springs were compressed by introducing compressed air into the cylinder until a force, calibrated to accelerate the sled to a chosen value, was reached. At time zero, the magnets were released. The sled was struck from the rear, accelerated, reached its maximum velocity, decelerated as it hit the breaks, and finally came to rest. The entire procedure was computer controlled.

The trauma protocol

The initial sled acceleration was 2.5 g. If the specimen did not fail, then the impact acceleration was increased by 2 g. This incremental process continued until 10.5 g. As mentioned, the flexibility was measured before and after each acceleration.

Data anlysis

Data were analyzed with Excel (Microsoft, Redmond, Wash.) and StatView (Abacus Concepts, Berkeley, Calif.). Averages and stan-

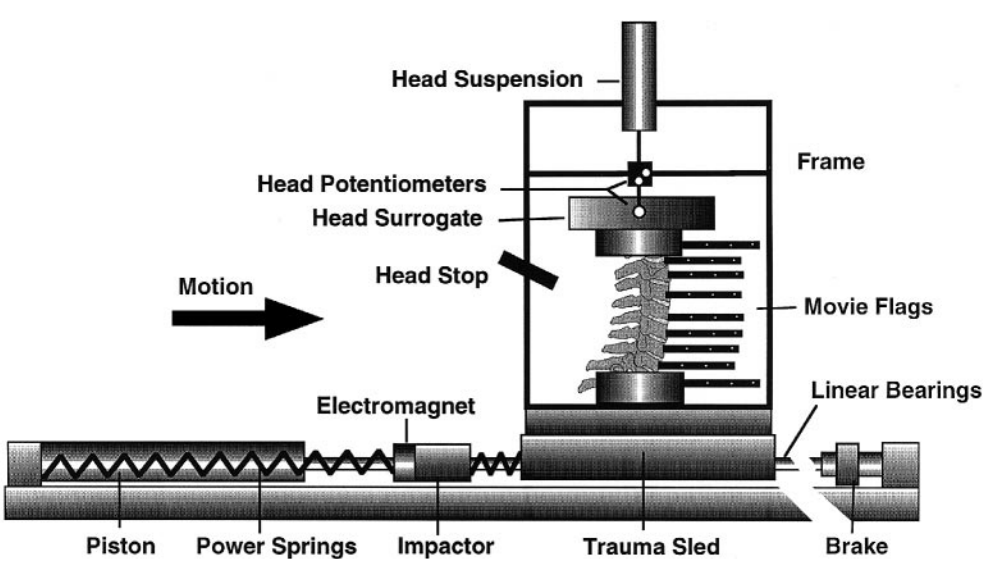


Fig. 3 Schematic diagram of the bench-top whiplash apparatus. The trauma sled was drawn towards the left by the pneumatic piston compressing the power springs. On computer command, the electromagnet released the impactor, striking the sled. At the end of the run the sled was braked. Also seen are the specimen, head surrogate, and head suspension system

dard deviations of ROM and NZ and corresponding Injury Potentials were computed for flexion, extension, and total motion (flexion plus extension) at each spinal level, and for each trauma acceleration. Comparisons were made between the traumatized and intact data to characterize the resulting injuries, i.e., a significant increase in ROM and/or NZ at any spinal level. One-factor ANOVA and Fischer LSD Post-hoc test at 5% and 10% significance levels were used to determine the flexibility increases. Injury threshold was determined as a significant increase ($P < 0.05$) above the intact value in either ROM or NZ.

Results

A total of eight cervical spine specimens were traumatized. Out of the four C0 – T1 specimens, only two survived beyond 2.5 g. Thus, there were too few values at C7 – T1 in most impact classes to result in meaningful statistics for this level. Consequently, the C7 – T1 level was not analyzed. When intact, the largest average ROMs were seen at the upper two levels: 19.5° flexion and 32.3° extension at C0 – C2 (Table 1). Similarly, the intact NZ was the largest in the upper cervical spine: 7.9° flexion and 19.9° extension at C0 – C2 (Table 2).

In general, the flexibilities increased with trauma, as one would expect (Tables 1, 2). Also shown in these tables are statistically significant increases ($P < 0.05$) and tendencies to increase ($P < 0.1$). The first significant increase of 4.8° was seen at C5 – C6 in extension ROM after the second impact of 4.5 g trauma. For the NZ, the first signficant increase (4.0°, extension at C5 – C6) also occurred after the 4.5 g trauma. Thus, 4.5 g is the injury threshold acceleration. There was tendency for the extension NZ to increase after the next impact of 6.5 g trauma at two levels: 13.3° to 20.2° at C0 – C1 and 1.7° to 5.8° at C6 – C7. There were no such increases in the ROM after the 6.5 g trauma.

The relative sensitivity of the two flexibility parameters, i.e., ROM and NZ, are seen more clearly by the corresponding Injury Potentials (Figs. 4, 5). A direct comparison of the ROMs and NZs for flexion (Fig. 4A vs 5A), extension (Fig. 4B vs 5B), and total (Fig. 4C vs 5C), shows that the NZ Injury Potentials are consistently larger than the corresponding ROM Injury Potentials.

Although the initial injuries were detected by the flexibility test at 4.5 g, they could not be visualized at that time. After the final trauma, the injuries were quite severe. An example of the injuries sustained by one specimen after 8.5 g impact is shown in Fig. 6. Note the disruption of the anterior disc fibers at C5 – C6.

Table 1 Averages (SDs) of ranges of motion (ROM) in degrees as measured in *n* specimens by the flexibility test: **A** flexion, **B** extension, and **C** total (flexion plus extension)

A Flexion ROM

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	7.2 (2.5)	12.3 (2.0)	3.7 (1.2)	4.0 (2.4)	4.8 (3.1)	5.5 (2.9)	4.2 (2.6)
2.5 g	8	7.9 (3.0)	11.3 (2.1)	3.7 (0.7)	4.2 (1.8)	5.1 (2.7)	5.4 (1.3)	5.2 (5.0)
4.5 g	5	7.4 (1.4)	13.1 (1.7)	3.6 (0.8)	4.1 (1.6)	4.4 (1.9)	7.9 (2.0)	4.1 (0.7)
6.5 g	5	6.7 (1.0)	11.2 (2.1)	4.0 (0.7)	3.0 (2.4)	5.5 (2.1)	5.9 (2.7)	5.8 (1.3)
8.5 g	5	6.9 (1.2)	13.1 (1.5)	4.1 (1.5)	4.6 (1.3)	5.2 (2.3)	8.6 (2.5)	6.5 (1.8)
10.5 g	2	6.9 (2.4)	17.1# (1.5)	3.0 (0.7)	3.0 (0.0)	4.4 (1.8)	6.4 (1.3)	4.7 (2.2)

B Extension ROM

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	20.2 (4.6)	12.1 (6.5)	3.2 (0.9)	4.2 (2.7)	4.9 (1.8)	4.8 (3.1)	3.8 (2.3)
2.5 g	8	20.8 (4.0)	12.7 (6.4)	3.3 (0.9)	3.8 (3.2)	5.0 (2.5)	5.3 (2.9)	4.3 (2.8)
4.5 g	5	23.3 (3.6)	12.9 (2.8)	3.6 (1.0)	5.8 (3.8)	5.4 (3.6)	9.6* (1.4)	4.4 (1.5)
6.5 g	5	23.4 (2.1)	10.8 (3.1)	3.6 (0.7)	3.5 (2.2)	4.6 (1.9)	10.2* (4.2)	7.9 (4.2)
8.5 g	5	21.4 (2.9)	10.4 (4.8)	3.7 (1.0)	3.7 (0.9)	6.1 (2.2)	11.5* (4.4)	6.6 (2.5)
10.5 g	2	20.0 (0.5)	14.2 (2.6)	3.4 (1.9)	2.6 (0.5)	3.9 (2.5)	13.6* (1.7)	5.2 (3.2)

C Total (Flexion plus Extension) ROM

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	27.4 (3.7)	24.4 (5.6)	6.8 (1.4)	8.2 (4.7)	9.8 (4.0)	10.4 (5.2)	8.0 (4.3)
2.5 g	8	28.7 (4.5)	24.0 (5.9)	7.0 (1.4)	8.0 (4.6)	10.1 (3.9)	10.7 (3.9)	9.5 (7.4)
4.5 g	5	30.7 (4.9)	25.9 (4.3)	7.3 (1.8)	9.9 (5.4)	9.8 (5.0)	17.4* (1.7)	6.4 (4.6)
6.5 g	5	30.1 (2.8)	22.0 (4.4)	7.5 (1.0)	6.2 (4.2)	10.2 (3.3)	16.8* (6.2)	13.7 (5.4)
8.5 g	5	28.3 (4.0)	23.7 (6.3)	7.8 (2.3)	7.6 (2.7)	11.2 (4.2)	20.1* (6.7)	13.1 (4.2)
10.5 g	2	26.9 (2.8)	31.3 (1.1)	6.3 (2.6)	5.5 (0.5)	8.3 (4.3)	20.0* (3.0)	9.9 (5.3)

* $P < 0.05$; # $P < 0.1$ (with respect to intact values)

Table 2 Averages (SDs) of neutral zones (NZ) in degrees as measured in *n* specimens by the flexibility test: **A** flexion, **B** extension, and **C** total (flexion plus extension)

A Flexion NZ

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	3.3 (1.8)	4.6 (2.4)	1.0 (0.6)	0.9 (0.9)	1.4 (1.2)	1.9 (0.8)	1.1 (0.8)
2.5 g	8	4.1 (2.5)	5.2 (1.9)	1.3 (0.4)	1.7 (1.2)	2.2 (2.3)	2.6 (0.9)	2.7 (4.3)
4.5 g	5	3.5 (1.0)	4.8 (1.4)	1.0 (0.7)	1.5 (1.2)	1.6 (1.6)	3.9 (1.2)	1.2 (0.3)
6.5 g	5	3.3 (1.0)	4.3 (2.0)	1.5 (0.2)	1.2 (2.0)	2.2 (0.4)	2.9 (1.2)	2.6 (0.8)
8.5 g	5	3.6 (1.6)	5.3 (2.7)	0.9 (0.7)	1.3 (1.7)	1.2 (0.7)	3.4 (2.6)	2.7 (1.7)
10.5 g	2	2.6 (3.3)	4.3 (6.2)	0.6 (0.5)	1.0 (1.0)	1.1 (1.3)	2.0 (0.1)	1.8 (1.3)

B Extension NZ

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	13.3 (4.0)	6.6 (3.3)	1.3 (0.8)	2.3 (2.2)	1.8 (2.4)	2.5 (2.3)	1.7 (1.3)
2.5 g	8	13.3 (6.3)	6.7 (2.9)	1.6 (0.5)	2.1 (2.2)	2.6 (2.0)	2.7 (2.3)	2.2 (2.2)
4.5 g	5	13.3 (5.1)	8.6 (2.5)	1.8 (0.8)	3.7 (3.6)	3.1 (3.0)	6.5* (1.7)	1.5 (1.1)
6.5 g	5	20.2# (2.4)	7.2 (3.7)	1.8 (0.7)	2.0 (1.3)	2.6 (1.3)	6.3* (4.1)	5.8# (4.1)
8.5 g	5	17.6# (2.5)	8.9 (7.8)	1.8 (0.9)	1.5 (0.7)	3.2 (1.3)	8.2* (3.3)	4.8# (3.3)
10.5 g	2	18.6# (0.8)	17.0# (10.1)	2.0 (0.8)	1.5 (0.5)	1.8 (0.6)	12.2* (2.5)	3.9 (3.0)

C Total (Flexion plus Extension) NZ

	n	C0-C1	C1-C2	C2-C3	C3-C4	C4-C5	C5-C6	C6-C7
Intact	8	17.0 (4.3)	10.9 (3.2)	2.2 (0.9)	3.1 (2.9)	3.2 (2.8)	4.4 (2.9)	2.8 (2.1)
2.5 g	8	19.6 (4.6)	11.9 (4.7)	2.8 (0.7)	3.6 (3.0)	4.9 (3.6)	5.3 (2.4)	4.9 (6.2)
4.5 g	5	16.7 (5.9)	13.4 (3.2)	2.8 (1.5)	5.2 (4.8)	4.8 (3.9)	10.4* (1.5)	2.0 (1.5)
6.5 g	5	23.7 (3.0)	11.5 (4.4)	3.3 (0.9)	3.2 (2.8)	4.4 (1.9)	9.2* (5.2)	8.4 (4.7)
8.5 g	5	21.2 (3.7)	14.6 (7.8)	2.8 (1.2)	2.5 (1.0)	4.4 (1.8)	11.6* (6.2)	7.5 (3.6)
10.5 g	2	21.3 (4.1)	21.8# (3.8)	2.3 (1.3)	2.6 (1.4)	2.9 (2.0)	13.6* (2.4)	5.0 (4.2)

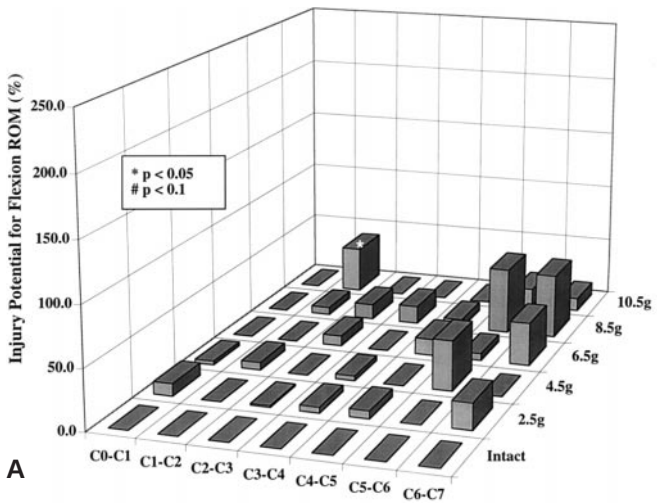
*$P < 0.05$; #$P < 0.1$ (with respect to the intact values)

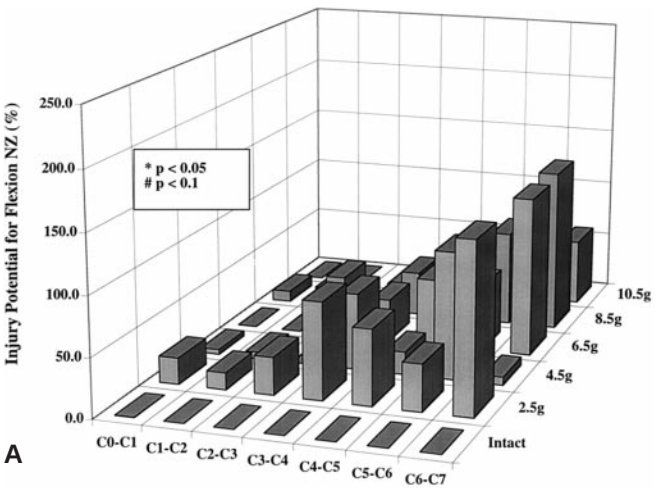
Discussion

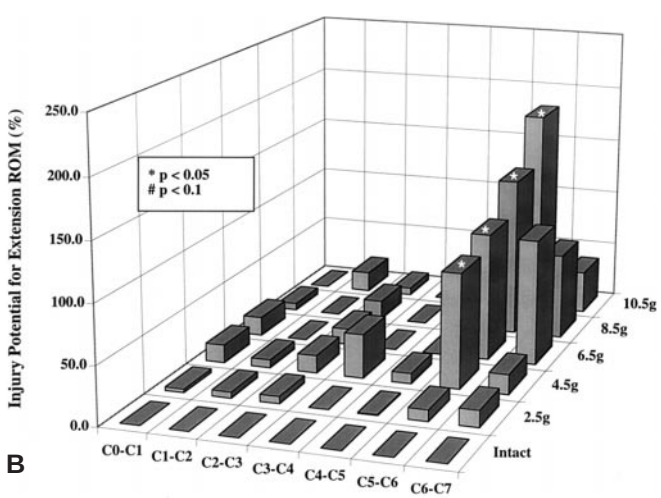
The major findings of our whiplash simulation study are three. First, the increases in flexibility occurred after 4.5 g at the C5 – C6 level, both in ROM and NZ. Additionally, there was a tendency to increase in NZ at C0 – C1 and C6 – C7 levels at the next higher trauma, i.e., 6.5 g. Second, significant increases in ROM and NZ occurred only in extension, although there was a tendency for flexion ROM at C1 – C2 to increase after the 10.5 g trauma. Third, as we had expected, the NZ was found to be a more sensitive parameter than the ROM in determining the physical changes in the spine due to the trauma. For example, the average Injury Potential, i.e., increase above the intact value, after 4.5 g trauma at C5 – C6 level was 98% for ROM versus 160% for NZ. Also, NZ predicted flexibility increases earlier, i.e., at a lower g-value, than did the ROM. For example, after the 6.5 g trauma, ROM increased ($P < 0.05$) above the intact value at C5 – C6 level, as did the NZ. However, in addition, NZ, but not the ROM, tended to increase ($P < 0.1$) at C0 – C1 and C6 – C7 levels. This higher sensitivity of the NZ versus ROM is consistent with findings from previous studies [25].

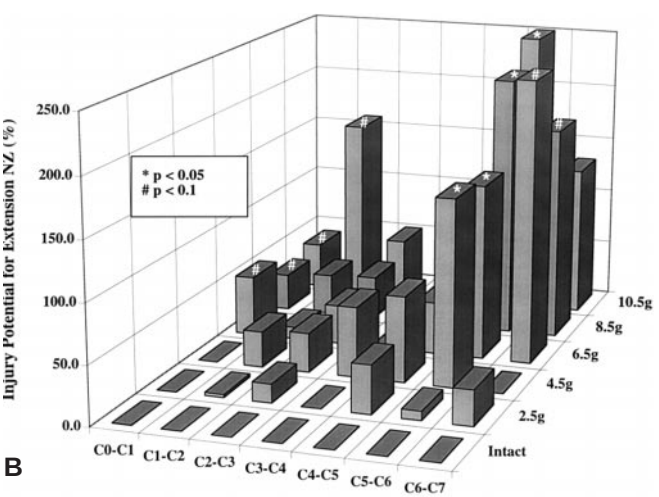
The flexibility method used here is well suited to quantify the soft tissue injuries sustained during simulated whiplash trauma. Complete disruptions of the various anatomic elements in severe whiplash trauma is sometimes identified at the time of surgery [1, 14, 16]. However, a significantly larger number of victims are subjected to less severe whiplash. They presumably have incomplete soft tissue injuries, which are not readily visualized even by MRI. These patients do not undergo surgery. Therefore, these soft tissue injuries go undetected and unidentified. Whiplash patients suffer pain and disability [9] and degenerative changes [14] for many years. We speculate that the clinical symptoms, especially long-term symptoms, may be explained on the basis of incomplete soft tissue injuries that may not easily heal. In these subfailure injuries, the soft tissues are not completely torn, but become stretched beyond their elastic limit [27]. Subsequent increases in flexibility have been correlated to soft tissue injuries of the spine [7, 22–24, 27]. Thus, increased flexibility implies yielding of the soft tissue structure, and constitutes functional injury to the spine.

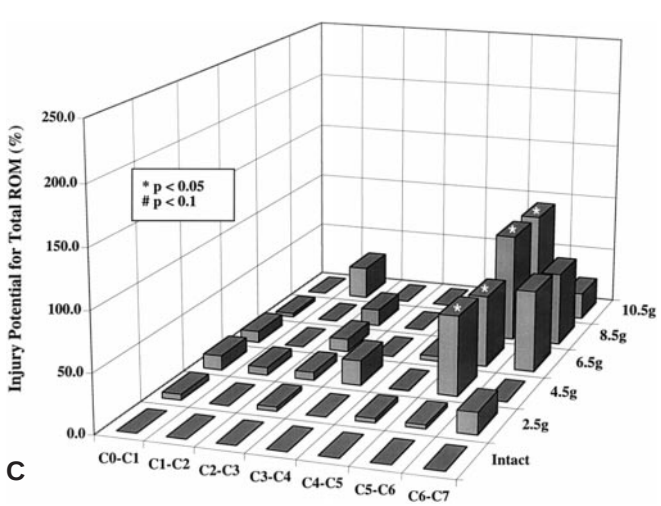
We used the incremental trauma method to produce injuries of increasing severity. There is another approach to experimental trauma, i.e., single trauma. There are important differences between the two approaches, and some advantages to the incremental trauma. Incremental trauma, as compared to single trauma, may injure the specimen











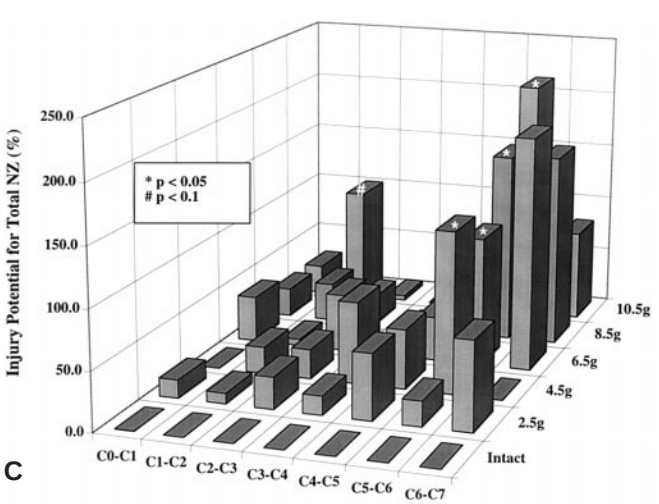


Fig. 4 A–C Injury Potentials for ROM at C0–C1 to C6–C7 levels due to whiplash trauma from 2.5 g to 10.5 g. These are percentage increases above the corresponding intact values. **A** Flexion, **B** extension, **C** total (flexion plus extension). Statistical significances are shown by * ($P < 0.05$ and # ($P < 0.1$).

Fig. 5 Injury Potentials for NZ at C0-C1 to C6-C7 spinal levels due to whiplash trauma from 2.5 g to 10.5 g. These are percentage increases above the corresponding intact values. **A** Flexion, **B** extension, **C** total (flexion plus extension). Statistical significances are shown by * ($P < 0.05$) and # ($P < 0.1$)

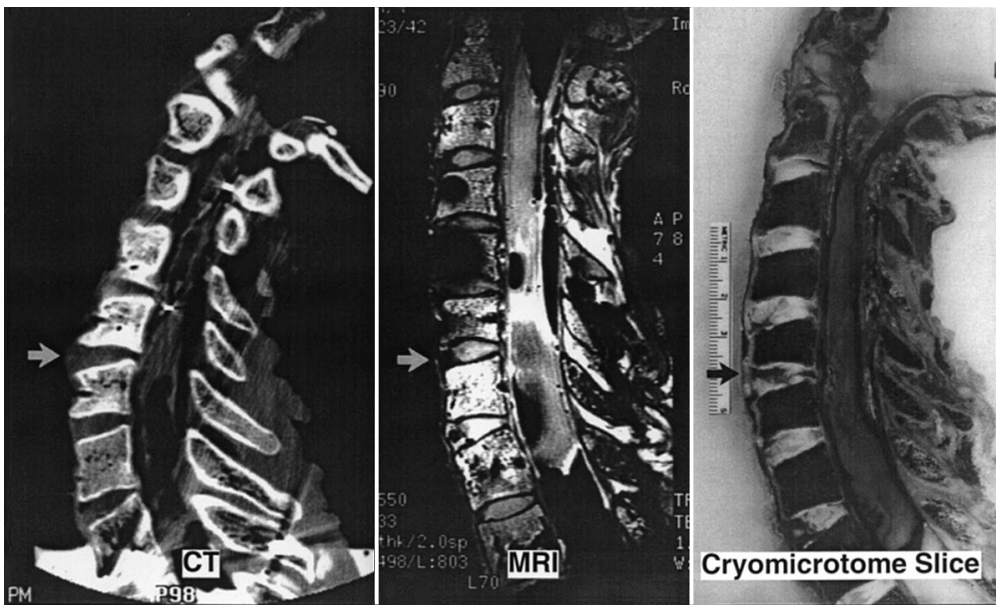


Fig. 6 Example of injury sustained in simulated whiplash trauma after 8.5 g impact. Note the C5-C6 anterior disc injury as seen in CT, MR, and cryomicrotome images (courtesy Dr. VM Haughton)

with each impact and thus bias each subsequent impact. On the other hand, the single trauma method requires, a priori, determination of the sled acceleration needed to produce a given injury. Given the variability of the human cervical spines (age, gender, size, bone density, degeneration, etc.), this task is extremely difficult, if not impossible to accomplish. The choice of too small an acceleration may not produce any injury, while a too large acceleration may fracture the specimen, eliminating if from the whiplash trauma simulation. In contrast, the incremental trauma method allows for a precise monitoring of the injury after each trauma. The experiment can be stopped when the required injury is achieved. Additionally, in the incremental trauma approach, to document the injury statistically, the specimen serves as its own control, thus requiring a smaller sample size. The single trauma approach, on the other hand, requires separate groups of specimens for intact and for different injury types. Given the variability of human specimens, the single trauma approach will need a much larger number of specimens. The question of equivalency of the final injuries produced by the two approaches was recently investigated. The injuries produced by single and incremental traumas were directly compared, using paired anterior cruciate ligament (ACL) preparations [15]. The single trauma consisted of stretching the ACL preparation by 88% of the failure deformation. In the incremental trauma, the paired ACL preparation was stretched incrementally to 55, 66, 77, and 88% of the failure deformation. Finally, both ligament groups were stretched to failure. The viscoelastic results of the 88% stretches and the failure load-deformation curves were compared. None of the 11 parameters used for this purpose showed any significant differences between the two trauma types. Therefore, we conclude that to establish injury threshold the incremental trauma model is the better approach. Note that the threshold of 4.5 g determined in our study was achieved after the second trauma only. We believe the first trauma of 2.5 g was too mild to have produced any injury. Therefore, 4.5 g is a good estimate of the injury threshold acceleration of the cervical spine.

There are some limitations to our studies. Being an in vitro study, there are no muscles, which may serve several roles in accident victims. First, muscles stabilize the neck and thus help carry the weight of the head. This function was simulated in our model by suspending the head from its center of gravity. The low-friction suspension system was carefully designed to balance the weight of the head during the entire trauma period, without compromising the inertial effects of the head mass or constraining the spinal motions. Second, muscles can act in response to trauma to produce forces to limit the head and neck motions. However, the reaction time for an unwarned victim to develop sufficient muscle force to brace the spine is approximately 200 ms [12, 33]. This is more than twice as long as the time to the peak whiplash trauma, i.e., when there is maximum risk for injury [13]. Finally, muscles may assist in passively tethering the spine. This was not simulated in our model, and its consequences are presently unknown. It should be noted, however, that several important findings from our study closely match the observations made by recent in vivo studies using volunteers [22]. The maximum head rotation, which varied between 18° for 10.5 g to 31° for 2.5 g in our study [6] is within the range (18°–51°) observed by others [21]. Finally, the intervertebral kinematics during the trauma was closely matched by cineradiography measurements in volunteers during simulated whiplash [17].

We hope that the results of the present study will help the clinician to direct their attention to the soft tissue injuries in whiplash trauma patients. Our results point to the lower levels of the cervical spine as the potential injury sites. This is especially true for relatively low-energy rear-end impacts. At higher impact energies, the upper levels of the cervical spine are also prone to injury. Further, our findings suggest that these injuries at the lower levels of the cervical spine will most likely tear the anterior structures, e.g., anterior longitudinal ligament and disc, and damage the posterior elements, e.g., facet joints. This is consistent with clinical observations of disc disruptions [1, 3, 16, 30] and facet joint pain [4] seen in whiplash patients. The present whiplash model, incorporating a whole fresh human cadaveric cervical spine specimen, can be used to study other types of trauma, i.e., frontal and side impacts. It can also be used to study the effects of important parameters for the prevention of whiplash injuries, e.g., head rest position and head-neck posture at the time of rear-end impact.

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