IDENTIFICATION OF SOFT TISSUE WHIPLASH INJURIES THROUGH BIOMECHANICAL TESTING OF THE CERVICAL SPINE BEFORE AND AFTER SIMULATED TRAUMA

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INTRODUCTION:

Injury mechanisms associated with soft tissue injuries of the cervical spine during low-speed, rear-impact collisions are complex and are not well understood. It is not known which soft tissue structures are damaged during the trauma. Injury prevention strategies formulated without a clear understanding of injury mechanisms have had little effect on overall occupant protection. The severity of whiplash related disorders most likely depends upon the acceleration magnitude and occupant posture at the time of impact. Panjabi et al. (1998) have simulated whiplash trauma using isolated osteoligamentous cervical spine specimens within a bench-top whiplash apparatus. The incremental trauma approach was used and biomechanical testing was performed on the specimens intact and following each trauma. Using this approach, insight into injury mechanisms and specific injury locations may be determined. The purpose of the current study was to use biomechanical testing of the cervical spine before and after simulated whiplash trauma as a tool to identify soft tissue injury sites, severity, onsets and progressions.

METHODS:

Six fresh-frozen human cervical specimens (occiput-T1) were prepared for trauma by carefully dissecting all non-osteoligamentous soft tissues. The occiput and T1 were rigidly secured in resin mounts. Motion measuring flags were secured to each vertebra and the occiput mount was provided with a loading jig. Following intact flexion-extension flexibility testing, a surrogate head was fixed to the occiput mount. The whiplash apparatus consisted of a bench-top sled upon which the T1 mount was rigidly fixed, high speed cameras (500 f/s) and custom designed transducers. Rear-end, head-forward impact simulations were performed using the incremental trauma approach at five impact accelerations: 2, 3.5, 5, 6.5 and 8 g. Muscle force replication was used during the whiplash simulations and was removed prior to each flexibility test. Following each trauma, the surrogate head was removed and flexibility testing was repeated. Flexibility testing was performed by applying pure moments to the occiput. A maximum load of 1.5 Nm was reached in four equal steps. At each moment step, the loading was held constant for 30 seconds to allow for viscoelastic creep. Two preconditioning cycles were applied in which no data were collected. Kinematic data were recorded during the third loading cycle. The flexibility parameters of neutral zone (NZ) and range of motion (ROM) were determined for total (flexion plus extension) motions. The injury potential was defined as the relative percentage increase in a flexibility parameter when compared to the corresponding intact value. Single factor, repeated measures ANOVA and Bonferonni post-hoc tests were used to determine significant increases ($p<0.05$) in NZ and ROM.

RESULTS:

In general, the injury potentials increased with increasing sled acceleration (Figures 1 and 2). Although some changes were observed at nominal acceleration levels, the major changes occurred at the lower cervical spine at 5 g and above, as evidenced both by the NZ and ROM injury potentials. Following the 6.5 g and 8 g impacts, both the NZ and ROM injury potentials indicated progression of injury severities at the lower cervical spine and the spread of injury potential to the middle cervical levels. At the upper cervical spine, the C0-C1 NZ injury potential increased at 5 g and above, while both the NZ and ROM injury potentials increased at C1-C2 following the 6.5 g impact.

DISCUSSION:

Although much previous attention been given in the literature to better understanding whiplash associated disorders, few studies provide comprehensive biomechanical evidence to support hypotheses regarding injury mechanisms, precise injury sites and severities. The results of the current study have potential to strengthen our understanding of injury mechanisms, however the assumptions of the model must be considered when interpreting the results clinically. Although muscle force replication was used to enhance the biofidelity of the model during trauma, no attempt was made to simulate the neural-muscular control system. In addition, the T1 mount was rigidly fixed to the trauma sled. Volunteer experiments have shown that T1 moves during whiplash. The effects of these assumptions on the mechanical properties of the spine following trauma are unknown. The current study demonstrated that significant increases in both NZ and ROM injury potentials occurred at four intervetebral levels following the 5 g impact. Above 5 g, the NZ and ROM injury potentials progressed and began spreading to the mid cervical spine. The injury potentials at the upper cervical spine increased at the higher acceleration levels. Simulated whiplash experimentation using the incremental trauma approach allows the mechanical properties of the cervical spine to be evaluated before and after the impacts, and compared to corresponding intact values. This approach provides a much greater data set as compared to whole cadaver, artificial dummy or volunteer experiments. The data generated from the current model can be used to better understand whiplash injury.

REFERENCES:

Panjabi et al. Whiplash injuries and the potential for mechanical instability. *Eur Spine J*. **7**:484-492, 1998.

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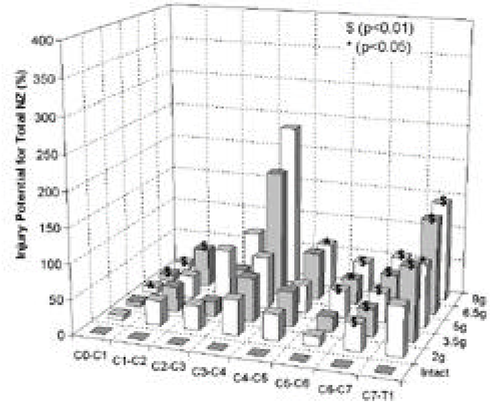

Figure 1: Total (flexion plus extension) neutral zone percentage increases from corresponding intact values.

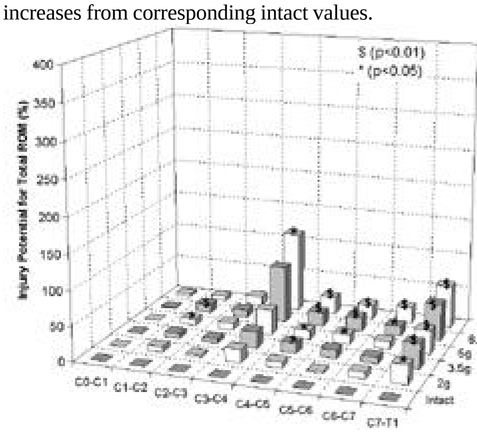

Figure 2: Total (flexion plus extension) range of motion percentage increases from corresponding intact values.