The effect of cerebrospinal fluid on spinal cord deformation in an *in vitro* burst fracture model

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ABSTRACT

Spinal cord injury affects more than 25 people/million annually in the United States and has significant economic, social and personal costs. Current research seeks to investigate the mechanical behavior of the spinal cord during high speed injury events. Knowledge of the column-cord interaction and its relationship to neurological injury could have implications for preventative strategies and clinical interventions as well as aid the development and validation of finite element and experimental models.

The current study extended a previously developed in vitro animal model of the burst fracture process. In particular, this study investigated the effect of cerebrospinal fluid (CSF) on the biomechanics of the cord when subjected to high speed transverse impact. The impact of a propelled bone fragment analogue with the animal cord model was recorded with high speed video and the images analysed to determine the deformation trajectory. Each cord was tested with dura and pseudo-CSF, with dura only and without dura. The trajectories obtained for each condition were compared by means of spatial and temporal descriptors.

Cord deformation was significantly reduced, although not eliminated, in the presence of CSF when compared to the bare state. The time to achieve maximum deformation and the duration of deformation were generally increased in the presence of CSF; however, statistical significance was not indicated. This may suggest a reduction in the cord-fragment interaction force for a given impulse. The dura was found to have no significant effect on deformation behaviour. The difficulty in reconciling data to obtain quantitative measures of cord deformation when surrounded by dura and CSF is discussed. This study indicates that while the protective mechanism of CSF may not fully extend to the high energy impact characteristic of a burst fracture, it may contribute to a lessening of cord deformation and applied force.

INTRODUCTION

Acute spinal cord injury occurs at a rate greater than 25 people/million annually in the United States and has significant associated economic, social and personal consequences (Burke et al., 2001). The biomechanical response of the spinal cord and its interaction with spinal canal components throughout the duration of the acute injury is not fully understood. Of particular interest is the burst fracture event, caused by high load axial impacts of the spinal column such as head-windscreen collisions and shallow diving accidents. It is characterised by propulsion of a bone fragment into the spinal canal with impingement on the spinal cord, and can lead to partial or complete paralysis. Knowledge of the column-cord interaction and its relationship to neurological injury could have implications for preventative strategies and clinical interventions as well as aid the development and validation of finite element and experimental models.

To date, biomechanical investigation of burst fractures has concentrated on the mechanism of vertebral body fracture and the extent of occlusion of the spinal canal in the absence of the spinal cord (Wilcox et al., 2003). More recently the response of the cord and interaction of the posterior longitudinal ligament and dura have been considered (Oakland, 2003). However, the inclusion of cerebrospinal fluid (CSF) in such models has not been reported in the literature.

In the current study, a previously developed *in vitro* animal model of the burst fracture process (Oakland, 2003; also available in Hall et al., 2005) was extended to include CSF. The impact of a propelled bone fragment analogue with an *in vitro* bovine spinal cord was recorded with high speed video and the images analysed to determine spatial and temporal descriptors of deformation.

The objective of this study was to investigate the contributions of the dura and CSF to the response of the spinal cord/dura/CSF construct under conditions of high speed transverse impact simulating a burst fracture event.

METHODS

Bovine spinal cord (age < 3mths) was extracted, prepared and tested within five hours of death. Eight (8) specimens were selected according to dura and cord integrity, length, and diameter and subsequently trimmed to a length of 140 mm. Excess fat was removed and nerve roots cut back to approximately 3 mm from the dural exit. Cyanoacrylate adhesive was applied sparingly to visible dural holes. Specimens were continually hydrated with isotonic saline throughout preparation and testing.

A cryo clamping technique (Oakland, 2003) held the specimens in a tensile testing machine at 5-7% strain (Fig. 1). The transverse impact was achieved by horizontal propulsion of a surrogate bone fragment at a mean velocity of 4.50 m/s (SD 0.52 m/s), using a previously developed device (Oakland, 2003). Posterior spinal elements (i.e. neural arch and ligamentum flavum) were simulated using a flat plastic plate placed against the cord/dura with minimum

force for all tests without pseudo-CSF and approximately 3 mm from the cord in tests with pseudo-CSF, without deformation of the cord. Oakland (2003) found no difference between deflection trajectories obtained with a flat posterior element or that approximating vertebral geometry. No other spinal structures were modeled. Preservation of native CSF was not possible during specimen harvest and preparation. Isotonic saline was manually injected into the subdural space immediately prior and during impact. Both water and CSF are Newtonian fluids with similar viscosity (Bloomfield et al., 1998).

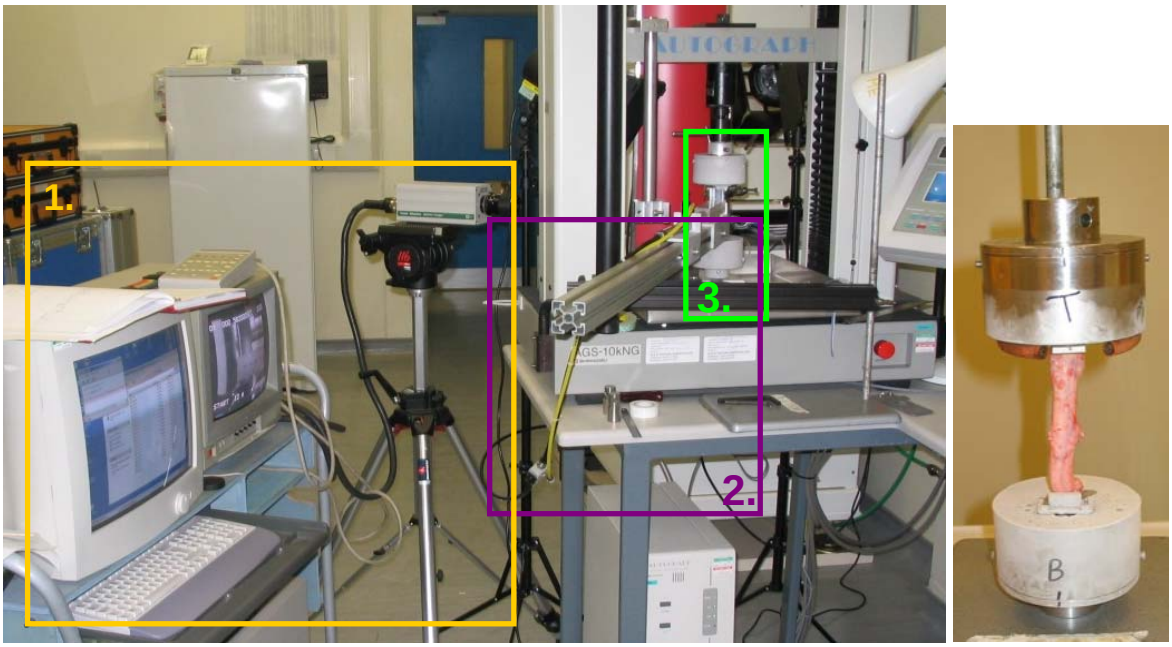

Figure 1: (left) Experimental apparatus: 1. high speed video system, 2. projectile system and 3. cryo clamps and specimen; (right) bovine specimen in cryo clamps.

A high speed video system was used to record the interaction of the bone surrogate and cord construct in the direction normal to impact at 4500 frames per second. Images (256x256 pixels) were analysed using custom software developed in the Matlab programming environment. The positions of the fragment and cord were determined for each frame using a calibration rule attached to the apparatus.

Each specimen was tested according to the following protocol: 1) impact test 1 – specimen intact with dura and pseudo-CSF; 2) drain pseudo-CSF; 3) impact test 2 – specimen intact with dura, without CSF; 4) remove dura *in situ*; 5) impact test 3 – specimen without dura or CSF. (The order of testing was not randomized in order to maintain dura integrity for part 3). The validity of performing multiple impact tests on a single specimen was investigated, and found acceptable for up to 3 tests.

Dura removal (step 4) was carried out with the specimen remaining in the tensile testing machine, with circumferential and longitudinal incisions and careful progressive detachment of dentate ligaments and nerve roots.

A fifth order polynomial was fit to each deformation trajectory and the following parameters determined: maximum deformation, time from first contact to maximum

deformation, duration of top 5% and 25% of deformation. All statistical analysis utilised SPSS (V12.0.1 for Windows).

RESULTS & DISCUSSION

Maximum deformation of the construct tended to be greater in the presence of CSF than with without, though this was not statistically significant (p=0.085, pairwise comparison, Bonferroni corrected). Deformation with CSF was not significantly greater than the bare cord (p=0.122) (Fig. 2).

There was no significant difference between the deformation results for the dura and bare specimens (p=1.000), as shown in Figure 2 (left). This result concurs with the results of Oakland (2003), who concluded, therefore, that the primary mechanical role of the dural sheath is prevention of spinal cord laceration.

Data was confounded by the inability to directly visualise the cord within the dura, thus the aforementioned deformation measurements were for the entire construct, not the spinal cord. Thus, the increased deformation is potentially in part due to an increased initial construct diameter. An estimation of actual spinal cord deformation was derived in which the CSF-filled subdural space was considered to be completely deformed prior to deformation of the spinal cord. Thus the spinal cord deformation was defined as follows:

Cord deformation = maximum occlusion - subdural space

Using this estimation, the spinal cord deformations illustrated in Figure 2 (right) for each specimen were obtained. Deformation was significantly lower for the construct including CSF than that excluding CSF (p=0.003) and than the spinal cord alone (p=0.002).

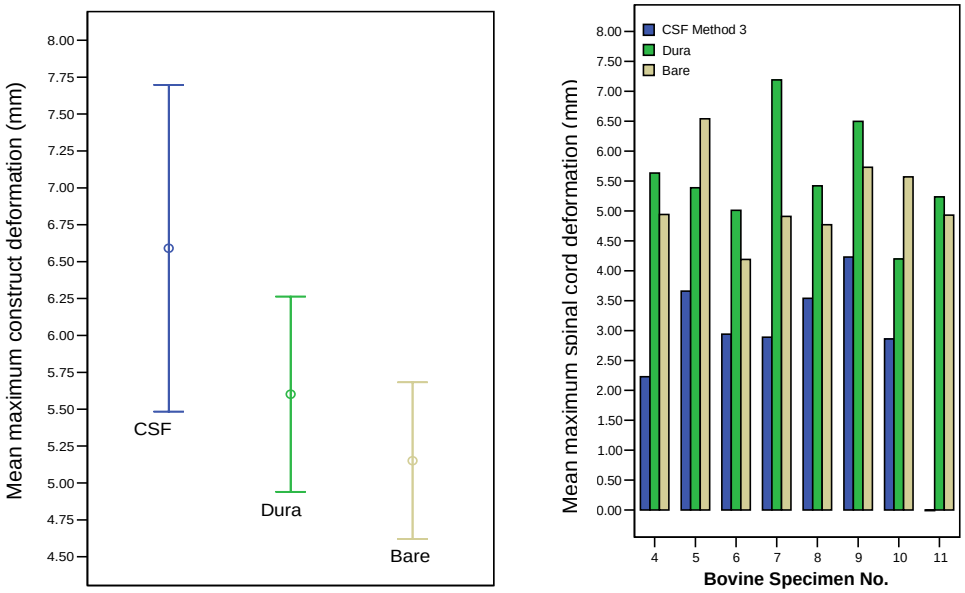

Figure 2. (left) Mean (±1SD) maximum cord construct deformation by construct condition. (right) Estimated ("Method 3") spinal cord deformation by construct condition for each specimen.

The mean estimated cord deformation with CSF was 2.8 mm (SD 1.3 mm), while the mean cord deformation with dura was 5.6 mm (SD 0.9 mm) and without dura was 5.2 mm (SD 0.7 mm). This represents a reduction in mean deformation of 2.8 mm (50%) and 2.4 mm (46%), respectively. For the mean bare cord diameter of 10.1 mm, these deformations correspond to a 55% and 52% spinal cord deformation, respectively. This result indicates that the CSF may partially reduce spinal cord deformation under conditions of high speed transverse impact. However, the clinical significance of this reduction is not known.

An increase in time from initial contact to maximum deformation and duration of deformation (top 5% and 25%) with addition of CSF to the bare spinal cord was observed, but with no statistical significance. Increased duration of contact may indicate a reduction in the cord-fragment interaction force for a given impulse.

The major limitation associated with this study is the inability to directly visualise the spinal cord when encased within the dura and CSF, due to a combination of the semi-opaque nature of the dura and reflection of light off the hydrated dural surface. A variety of back- and front-lighting and contrast dye dosing methods were attempted without considerable gain.

CONCLUSIONS

These results indicate that for a burst fracture situation with vertebral bone retropulsion, cord deformation may be significantly reduced, although not eliminated, in the presence of CSF when compared to the bare state. The significance of this reduction in terms of severity of neurological injury and recovery is not known. The temporal deformation parameters generally increased in the presence of CSF, but did not indicate statistical significance. This may indicate a reduction in the cord-fragment interaction force for a given impulse.

This study has highlighted that further work is required to obtain quantitative measures of spinal cord deformation when encased by dura and CSF. Results indicate that while the protective mechanism of CSF may not fully extend to the high energy impact characteristic of a burst fracture, it may contribute to a lessening of cord deformation and applied force. Future experimental and finite element models of dynamic transverse compression should consider incorporation of CSF layers to more closely approximate the spinal cord biomechanical environment.

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