The Effect of Cerebrospinal Fluid Thickness on Traumatic Spinal Cord Deformation

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A spinal cord injury may lead to loss of motor and sensory function and even death. The biomechanics of the injury process have been found to be important to the neurological damage pattern, and some studies have found a protective effect of the cerebrospinal fluid (CSF). However, the effect of the CSF thickness on the cord deformation and, hence, the resulting injury has not been previously investigated. In this study, the effects of natural variability (in bovine) as well as the difference between bovine and human spinal canal dimensions on spinal cord deformation were studied using a previously validated computational model. Owing to the pronounced effect that the CSF thickness was found to have on the biomechanics of the cord deformation, it can be concluded that results from animal models may be affected by the disparities in the CSF layer thickness as well as by any difference in the biological responses they may have compared with those of humans.

Keywords: spinal cord injury, fluid-structure interaction, transverse impact, biomechanics

Traumatic spinal cord injuries are frequently caused by traffic accidents or falls, in which a vertebral fracture or dislocation may compromise spinal cord function (Sekhon & Fehlings, 2001; Bensch et al., 2006). The mechanical damage to the cord is followed by a physiological cascade of events that may result in a further substantial deterioration of motor and sensory performance (Liverman et al., 2005). Recent studies have found that different mechanical insults to the cord, for instance, owing to lateral or anterior dislocations, give rise to variations in the pattern of cord injury and severity (Fiford et al., 2004; Choo et al., 2007; Clarke et al., 2008). These findings indicate that a more detailed understanding of the mechanics of the injury process can inform future injury prevention strategies, diagnoses, and interventions.

Contusion injury has been frequently simulated in vivo (Allen, 1914; Anderson, 1985; Liu et al., 1997; Kloos et al., 2005; Choo et al., 2007; Maikos & Shreiber, 2007), in vitro (Hall et al., 2006; Jones et al., 2008; Persson et al., 2009), and computationally (Ichihara et al., 2003; Oakland, 2003; Fiford, 2005; Greaves et al., 2008; Maikos et al., 2008; Ouyang et al., 2008; Persson et al., 2011). Recent in vitro studies (Jones et al., 2008; Persson et al., 2009) as well as a computational model (Persson et al., 2011) have found that the cerebrospinal fluid, which surrounds the spinal cord, has an important protective effect on the cord deformation during trauma. However, these aforementioned investigations used a diverse range of animals either in vivo or in the generation of in vitro models, all of which have different relative and absolute cerebrospinal fluid (CSF) layer thicknesses. For instance, whereas there are a number of similarities in terms of anatomical and mechanical data from bovine and human cords (Holsheimer et al., 1994; Bilston & Thibault, 1996; Oakland, 2003; Oakland et al., 2006), the CSF layer appears substantially thicker in humans (Holsheimer et al., 1994; Persson et al., 2009). It is, therefore, important to study the effect of this difference in terms of the traumatic cord insult to ensure that the results from bovine specimens can be effectively translated to humans. Furthermore, similar considerations should be implemented in rodent models, which are frequently used for in vivo studies (Liu et al., 1997; Kloos et al., 2005; Choo et al., 2007; Maikos & Shreiber, 2007), and have a very thin CSF layer (Maikos et al., 2008) when compared with that of humans.

In this study, a previously validated fluid–structure interaction model (Persson et al., 2011) was used to investigate the effect of a change in the CSF layer thickness, in terms of both intra- and interspecies variability on the cord deformation during impact.

Methods

The base model and its validation have been described in detail elsewhere (Persson et al., 2011) but will be summarized in this section. The base computational model is a three-dimensional model built to replicate the transverse impact between a bone fragment and the spinal cord during a vertebral burst fracture. The model was constructed and solved using ADINA (Version 8.5, ADINA R&D Inc., Watertown, MA), which permits the
direct coupling (simultaneous solution) between a solid and a fluid computational (finite element) model as well as the implicit solution of a dynamic fluid–structure interaction (Zhang & Bathe 2001). A simulated bone fragment of a prescribed initial velocity (4.5 m/s) is transversely propelled onto the spinal cord surrounded by CSF and dura mater. The cord and dura ends are fixed and a flat, fixed plate is placed behind them to simulate the posterior elements. Hall et al. (2006) found no significant difference in experimental cord deformation when using a similar flat posterior surface or a more anatomically correct posterior surface. The fluid bottom outlet is closed whereas the top one is open. The material properties used can be found in Table 1.

The geometry for the base model was based on the average bovine dimensions found in a parallel, experimental study (Persson et al., 2009), which was used to validate the computational model (Persson et al., 2011). The average thickness of the CSF layer found in the experiments (1.5 mm) was used in the base model. However, the thickness of the CSF layer in the experiments, that is, within the bovine specimens, varied between 0.6 and 2.8 mm, and, in the current study, models with layers of these two thicknesses were also produced (Figure 1).

Table 1: Material properties of all components of the model

<table>
<thead>
<tr>
<th>Material</th>
<th>Stress/Strain Relationship</th>
<th>Density (kg/m³)</th>
<th>Poisson’s Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal Cord</td>
<td>Ogden model, a $\sigma = \mu(\lambda^{\alpha-1} - \lambda^{-0.5\alpha-1})$; $\mu = 2$ kPa, $\alpha = 9$</td>
<td>1050</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>(Hung et al., 1982)</td>
<td></td>
<td>(density of brain; Nelson et al., 1971)</td>
</tr>
<tr>
<td></td>
<td>Dura Mater: Linear elastic: $E = 80$ MPa</td>
<td>1000</td>
<td>0.49</td>
</tr>
<tr>
<td></td>
<td>(Tunturi, 1977; Tencer et al., 1985; Runza et al., 1999; Persson et al., 2010)</td>
<td></td>
<td>(as quoted in Oakland, 2003)</td>
</tr>
<tr>
<td>Bone Fragment (Tufnol)b</td>
<td>Linear elastic: $E = 6.5$ GPa</td>
<td>1360</td>
<td>0.3</td>
</tr>
<tr>
<td>Posterior Element (Stainless Steel)b</td>
<td>Linear elastic: $E = 193$ GPa</td>
<td>8000</td>
<td>0.3</td>
</tr>
<tr>
<td>Cerebrospinal Fluid (Saline Solution)</td>
<td>Newtonian: $\eta = 0.001$ Pa s</td>
<td>1000</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>(Brydon et al., 1995)</td>
<td></td>
<td>(Richardson &amp; Wissler, 1996)</td>
</tr>
</tbody>
</table>

aOgden model, where $\sigma$ is stress and $\lambda$ is the stretch ratio.

bManufacturer’s data: Tufnol Grade 6F/45 (Tufnol Composites Ltd, Birmingham, U.K.) and stainless steel Grade 304 (Aalco Metals Ltd, Surrey, U.K.).

Figure 1 — Cross-sections of a representative animal cord and the geometries produced to represent the natural variability of the bovine spinal cord and the surrounding cerebrospinal fluid. The base model in this figure is the same as the bovine model depicted in Figure 2.

Figure 2 — Cross-sections of the geometries produced to represent the human spinal cord in comparison with the bovine base model.
Results

The trajectory of the simulated bone fragment impacting on the base model is shown in Figure 3. The cord started to deform immediately after impact between the fragment and the construct, and maximum cord deformation was reached when the inertia was overcome and the whole construct started to move toward the posterior plate (the posterior plate was placed 0.5 mm away from the construct to replicate the experiments).

The fragment trajectories of the other models are shown in Figure 4. The trajectory into the model of a human T12 is also illustrated in Figure 5. Whereas all bovine models gave similar maximum deformations of the whole construct, the duration of the impact and maximum cord displacement were substantially different. The larger CSF layer was found to give a longer impact period but a smaller deformation of the cord. The converse was true for the smaller CSF layer in which a shorter period of deformation was observed. The correlation of the behavior between the cord and dura in terms of the deformation was greatest when the CSF layer was thinnest. This small CSF thickness model also gave rise to higher stresses and strains in the cord at maximum deformation (Table 2).

Contrary to the bovine model, in the human models there was only one peak of cord deformation (before the cord moved toward the back plate) since the pellet recoiled before the neural tissue could impinge on the posterior elements owing to the thicker CSF layer. Smaller amounts of cord compression were found for the human dimensions, but higher stresses at maximum cord compression because of the smaller cross-sections of these cords (Table 2). The percentage compression was also similar for human models and the average bovine model. The longitudinal strain was lower in the human models compared with the base model owing to the lower amount of displacement of these cords. At maximum compression of the whole construct, the strain was higher in the C6 model than in the T12 model, as this cord was still deforming at that point. The C6 model failed to give a converged solution after 9 ms as a result of an excessive distortion of the fluid (finite element) mesh at the sides (at the level of impact).

Figure 3 — The fragment trajectory into the cord/dura/CSF construct of the bovine base model.

Figure 4 — The fragment trajectory into the cord/dura/CSF construct of all models. The filled lines represent the whole construct (cord/dura/CSF) deformation and the dashed lines represent the cord deformation.
Discussion

In this work, a previously validated fluid–structure interaction model (Persson et al., 2011) was used to assess the effect of a variation in CSF thickness on the cord deformation during a contusion injury. This was done both in terms of natural variability (bovine, 0.6–2.8 mm layer thickness) and in terms of the geometrical variation between species (bovine and human [human CSF thickness 3.35 and 4.5 mm for C6 and T12, respectively]).

Evaluating the effect of intraspecimen variability within a bovine model, it was found that a larger CSF layer gave rise to smaller deformations and lower stresses and strains within the cord. Furthermore, the human models that had thicker CSF layers also gave rise to lower absolute deformations and strains within the cord. However, the percentage compressions were similar to those of the average bovine model. In addition, higher stresses were found within the human cords at maximum cord compression owing to the smaller cross-sections of these cords. Although the exact quantitative effects are difficult to establish, an increase in cord compression (Anderson, 1985; Kloos et al., 2005) and longitudinal strain (Galbraith et al., 1993; Bain & Meaney, 2000; Fiford, 2005; Shi & Whitebone, 2006) would result in an increase in neurological damage. A decrease in CSF layer thickness would therefore tend to lead to an aggravation of the neurological injury that is more focused at a particular level.

The results also indicate that there may be differences in neurological damage from trauma because of morphological, as well as physiological, differences between species. The majority of recent in vivo models have used rodents (Liu et al., 1997; Jakeman et al., 2000; Fiford et al., 2004; Fiford, 2005; Kloos et al., 2005; Choo et al., 2007; Maikos & Shreiber, 2007; Clarke & Bilston, 2008; Clarke et al., 2008), which have a thinner CSF layer than both bovine and human subjects (Maikos et al., 2008). Furthermore, the impacts in in vivo animal models are usually performed on the posterior part of the cord with the animal in prone position. A prone position has been found to reduce the thickness of the posterior fluid layer (and increase that of the anterior layer) in humans (Holshheimer et al., 1994), indicating an additional source of error in relation to CSF layer thickness. These differences in anatomy and injury setup may contribute to some of the differences found between rodent and human neurological results in terms of the biological response to trauma (Hagg & Oudega, 2006).

There are some limitations of the present model, such as its geometrical simplicity and the use of a linear material model as opposed to a nonlinear one for the dura mater. However, previous studies have used a similar level of detail (Oakland, 2003; Greaves et al., 2008; Maikos et al., 2008), and a parallel study (Persson et al., 2011) found no substantial effect on the cord compression (a difference of 2%) owing to a division of the cord into gray and white matter.

In conclusion, the use of different animal models with dissimilar CSF layer thicknesses may give different results in terms of final neurological deficit. The CSF thickness has a substantial effect on the spinal cord deformation during trauma and should be taken into account in future models.

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Table 2: Summary of the results for the computational models

<table>
<thead>
<tr>
<th>Model</th>
<th>Maximum Cord Compression in the Anteroposterior Direction</th>
<th>( \sigma_{\text{Von Mises}} ) (kPa) at Max Compression*</th>
<th>Longitudinal Strain, ( \varepsilon_{zz} ) at Max Compression*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bovine base model</td>
<td>41% / 4.1 mm</td>
<td>15.3 (13.5)</td>
<td>0.039 (0.110)</td>
</tr>
<tr>
<td>Bovine small layer</td>
<td>53% / 5.3 mm</td>
<td>31.8**</td>
<td>0.139**</td>
</tr>
<tr>
<td>Bovine large layer</td>
<td>37% / 3.7 mm</td>
<td>13.4 (6.4)</td>
<td>0.042 (0.079)</td>
</tr>
<tr>
<td>Human geometry—C6</td>
<td>43% / 2.9 mm</td>
<td>32.3 (8.4)</td>
<td>0.027 (0.065)</td>
</tr>
<tr>
<td>Human geometry—T12</td>
<td>40% / 2.8 mm</td>
<td>36.6 (4.0)</td>
<td>0.020 (0.010)</td>
</tr>
</tbody>
</table>

*The values at maximum compression of the whole construct are shown in parentheses.

**Here the maximum compression of the cord coincided with the maximum compression of the whole construct.

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Figure 5 — Cross-section of the impact between the simulated bone fragment and the human T12 cord/dura/CSF construct.
Acknowledgments
This work was made possible through funding obtained from the European Community, Contract MEST-CT-2005-020599.

References


