Original Article

Experimental study on pressure response to graded spinal canal compromise in an *in vitro* burst fracture mode

ABSTRACT

Background: Spinal cord compression is a known cause of spinal cord injury. The purpose of this study is to measure pressure response during graded spinal cord compression. This information will be important in evaluating the amount of canal compromise that can be tolerated before risking neurological injury secondary to cord compression. To date, there is no published study that has evaluated pressure response to graded canal compromise in the thoracic and lumbar spine.

Materials and Methods: A comparative biomechanical investigation using an *in vitro* burst fracture model of graded spinal canal compromise was performed. Four porcine spines, sectioned into four thoracics and four lumbar segments, were harvested from 30 kg pigs. Graded spinal canal compromise (0.75 mm/30 s) was achieved using a modified 12.7 mm dynamic hip screw. The real-time ventral epidural pressure was measured at each 0.75 mm of canal compromise.

Results: A significant increase in spinal cord pressure was recorded during graded spinal cord compression ($P < 0.0001$), and there were no statistical differences between the increase in pressure measured in the thoracic and lumbar spinal segments ($P = 0.83$). The pressure to degree of canal compromise curve exhibited an initial rapid rise in pressure followed by incrementally smaller increases in pressure as canal compromise increased.

Conclusions: Spinal cord pressure increased with any degree of canal compromise, the most important rise occurring with initial compression. Future studies will evaluate the usefulness of laminectomy *in vivo* to completely restore ventral epidural pressure in the thoracic and lumbar spine.

Keywords: Graded spinal cord pressure, lumbar spine, pig model, spinal canal compromise, thoracic spine

INTRODUCTION

Spinal cord injuries (SCIs) represent a disabling and often irreversible condition that has high socioeconomic costs. Increasing evidence implicates abnormalities in normal spinal cord function in the pathophysiology of acute SCI. There are both primary and secondary mechanisms of injury following SCI - the primary injury being the initial mechanical injury itself. Secondary injury succeeds the initial injury and occurs from a sequence of the biochemical and cellular process that are triggered from the primary injury.[1-5] This is supported by clinical observations that many SCIs occur without actual severance of the spinal cord. In injuries causing cord compression, the ensuing damage is caused from microcirculatory insufficiency to tissues, leading to irreversible tissue damage.[5-7]

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Since the amount of canal compromise following a spinal cord trauma is variable, the temporal evolution for spinal cord dysfunction is dependent on the severity of cord compression. Wolfla *et al*. quantified the effects of graded ventral spinal canal compromise in the cervical spine.[8] In the static condition, this study showed a sinusoidal association between epidural pressures and percent canal compromise, whereby the pressures remained minimal until canal compromised reached about 20%, after which they increased sharply until around 70% where they leveled off again. This suggests that the viscoelastic property of a spinal cord allows it to have some compensatory capacity to prevent acute injury. However, to the best of our knowledge, there has yet to be any study to quantify the pressure response to graded spinal canal compromise in the thoracic and lumbar spine. To better understand pressure response to spinal canal compromise in the thoracic and lumbar spine, we measured changes in ventral epidural pressures during static graded spinal canal compromise.

MATERIALS AND METHODS

Spinal cords were harvested from four mini-pigs weighing 30–35 kg. All animals were euthanized in experiments not related to our work. Spines were collected in their entirety following the animal's death. Thoracic and lumbar cord segments containing six vertebrae were prepared for the experiments. The thecal sac was tied off at each end of the cord segments to assure the integrity of the cord during compression. All cord segments were frozen until used. Bassi *et al*.[9] have shown that freezing and thawing spinal cords has no effect on the intrinsic properties of the cord when compared to fresh spinal cord segments.

For use, cords were thawed in a saline bath at room temperature. A lateral fluoroscopic/computed tomography (CT) image was taken of each segment to measure anterior-posterior canal diameter. This data were later used to calculate the amount of canal compromise. Each segment underwent placement of a ventral compression device. To compress the cord in a graded and controlled manner, a modified 12.7 mm dynamic hip screw (DHS) with a blunt tip was used [Figure 1]. The screw had a 3-mm pitch. A pressure transducer integrated into the DHS screw captured real-time changes in pressure. These signals were transmitted to a previously calibrated pressure monitor and recorded for later use. Before testing, a pilot hole was drilled in the ventral midline of a vertebra. Once the position of the hole was verified under fluoroscopic control, the hole was gradually enlarged with successively larger drill bits until a 10-mm hole was made. Drilling was done under fluoroscopy to ensure we were not drilling beyond the anterior longitudinal ligament. The anterior longitudinal ligament was opened and excised manually. Every segment was verified to ensure the dura mater had remained intact.

Experimental procedure

The DHS screw was first advanced under fluoroscopy so the flat front would be flush with the posterior vertebral body. This would refer to as timestamp 0 - a first pressure was recorded (opening pressure). The screw was then advanced in 0.75 mm increments every 30 s, by turning the screw 90° at a time. The epidural interface pressure was recorded after every 0.75 mm increments of canal compromise. We quantified pressure response to graded spinal canal compromise until approximately 80% of the spinal canal was compromised. A lateral fluoroscopic/CT image was taken when the DHS screw was in its final position, to later quantify the degree of neural compromise [Figure 2].

Data analysis

To be able to make comparisons between animals, pressures were grouped according to spine segment (thoracic vs. lumbar) and then mean, and standard variation was calculated using the distance traveled by the screw after each 90° turn. To determine differences between spine segments, two-way ANOVA with repeated measures was run. Since anatomical differences (primarily canal diameter) could influence resulting variation in pressures, the percentage of canal compromise was determined by dividing the depth of the screw after each 90° turn by the total anterior-to-posterior canal diameter, as measured with fluoroscopy. These measures were used to define the pressure curve produced using nonlinear fit with outlier removal. Since no differences were found between the thoracic and lumbar segments in the ANOVA ($P > 0.05$), all segments were grouped together for the nonlinear fit analysis. One lumbar segment was identified as an outlier and thus not included in the nonlinear fit

Figure 1: Example of the experimental setup. Each spinal segment underwent graded spinal canal compromise with the use of a modified 12.7 mm dynamic hip screw. Part of the vertebral body has been removed for demonstrative purpose

equation produced. All statistical tests were run using Prism 6 (GraphPad Software Inc., La Jolla, CA, USA). All results are displayed as the mean ± standard error and were considered statistically significant at $P < 0.05$.

RESULTS

Opening pressure for all segments was below 5 mmHg. Quite intuitively, as shown in Figure 3a, as canal compromise increased a significant increase in pressure was recorded ($P < 0.0001$, repeated measures ANOVA, main effect). Although slightly higher pressures were observed in the lumbar segments, no statistical differences between the thoracic and lumbar spinal segments were found ($P = 0.83$, repeated measures ANOVA, group effect).

Since the epidural pressures did not differ between lumbar and thoracic regions, all spinal segments were grouped together for the nonlinear fit analysis. Figure 3b shows the curve produced between the percentage of canal compromise (%) and the associated change in pressure. In comparison to Wolfla *et al*.,[8] who reported a sinusoidal increase in pressure, our measures fit a single-phase nonlinear model. This describes an immediate and rapid rise in pressure during the initial advancement of the DHS screw, with pressures showing incrementally smaller increases in pressure as the screw advanced. As shown in Figure 3b, the model produced a curve around which all of our measures from the lumbar and thoracic sections were closely located (gray area denotes 95% confidence interval of the predicted curve).

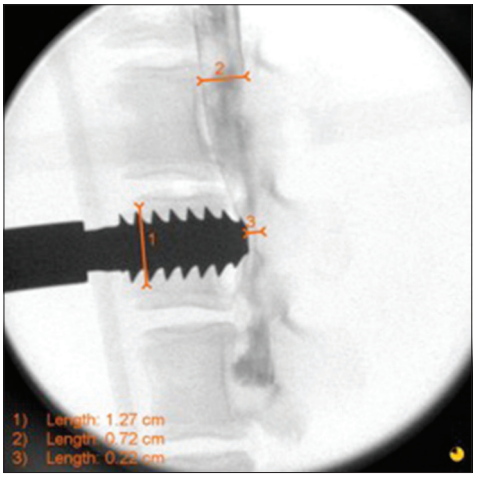

Figure 2: Example of a lateral fluoroscopic/computed tomography image of a spinal segment when the dynamic hip screw reached approximately 70% of spinal cord compression. (1) An example measurement of the dynamic hip screw width, (2) width of the noncompressed spinal cord, and (3) width of the compressed spinal cord. Two lateral computed tomography images were taken during each experiment: the first at timestamp 0 (opening pressure), and a final one following the last compression. Pre- and post-images were used to correlate pressure response to graded degrees of canal compromise

DISCUSSION

This study was the first to measure pressure response during graded canal compromise in the thoracic and lumbar spine. The interpretation of our findings is that both the thoracic and lumbar spine is at risk of important pressure changes with any amount of canal compromise and thus carries important implications in SCI. Spinal cord compression is known to cause biochemical and pathological changes in the cord following injury.[1-5,10]

Early decompression is known to improve neurological symptoms. Despite the seemingly obvious goal of these surgical interventions, the optimal timeframe to undergo decompressive surgery remained unclear, until recently.

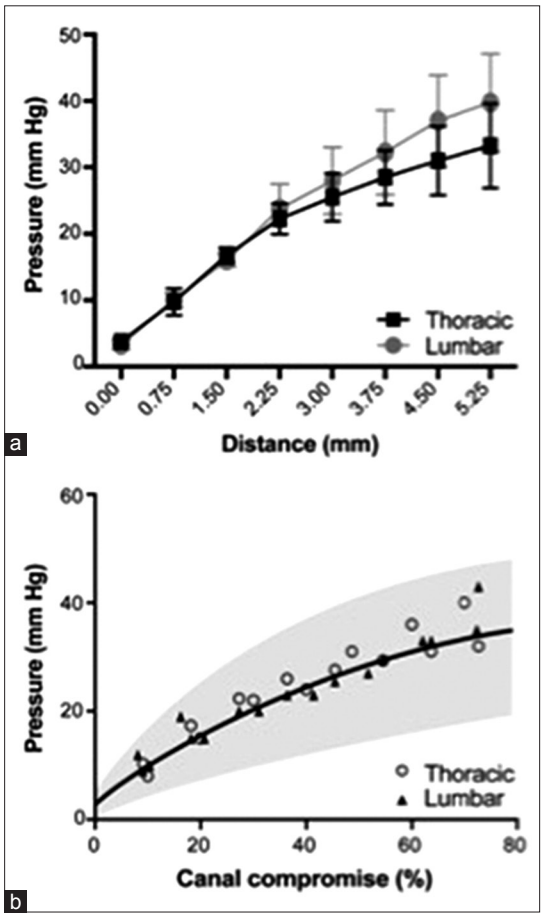

Figure 3: The pressure curves. (a) Ventral epidural pressures recorded after each advancement of the screw; X-axis represents total distance traveled by the screw, Y-axis represents ventral epidural pressure. There were no significant differences between the pressures recorded in the thoracic (black squares) or lumbar (gray circles) spinal segments. (b) Distance traveled by the screw was converted to percent canal compromise (X-axis) and used for nonlinear fit, producing a single-phase curve that demonstrated an immediate rise in pressure with any amount of canal compromise. Gray shaded area represents the 95% confidence interval produced during the nonlinear regression (black, curved line). Our measures are plotted within this 95% confidence interval; thoracic (open circles), lumbar (black triangles)

The Surgical Timing in Acute Spinal Cord Injury Study was published showing that traumatic cervical SCI patients who underwent early (<24 h after injury) decompressive surgery had a significantly more favourable neurological recovery, defined as at least 2 grade American Spinal Injury Association Impairment scale improvement at 6-month follow-up, as compared to those who underwent surgery >24 h after injury.[10]

Graded spinal cord compression has been useful in studying changes in spinal cord electrophysiology and blood flow in the acute SCI model.[7,11] Carlson *et al.* recognized that 20% canal compromise was needed to produce a 50% reduction in spinal cord evoked potential amplitude.[11] In an attempt to elucidate both circulatory and conduction disturbances during cord compression, Griffiths *et al.* assessed spinal cord blood flow, and dorsal column conduction during subacute spinal cord compression in dogs. Strikingly, their study showed that blood flow stopped at cord pressures above 55–60 mmHg. The dorsal column evoked potential amplitude was, however, significantly decreased at much lower pressures. Conduction failure occurred at perfusion pressures of 20–30 mmHg.[6]

As seen in our results, there is an immediate rise in cord pressure with any degree of canal compromise. Our experiments also generated the unanticipated finding that the thoracic and lumbar spine responds similarly to graded spinal cord compression. This may be attributed to the fact that pig spinal cord is not anatomically similar to those of humans. The spinal cord of pigs generally terminates at the level of the second sacral vertebra. Therefore, the cauda equina begins more caudal in the pig than it does in the human. The similarity between the gross anatomy of the thoracic and lumbar spinal cords between these levels likely influences their similar response to graded canal compromise.[12]

In the present study, the epidural pressure was measured to quantify pressure response that occurs as the result of graded spinal canal compromise. Mini-pig spines were chosen as a model for our study given their size to fit our DHS screw. Moreover, pigs have been previously used in many spine studies.[13-16]

Finally, as described in Figure 3b, one goal of our study was to create a predictive model that could describe the pressure curve produced using a nonlinear fit analysis. This model successfully predicted, within its 95% confidence interval, all of our measures from the lumbar and thoracic sections.

Results of the present study show our measures fit a single-phase nonlinear model. Interestingly, our results show a different pressure-to-degree of canal compromise curve in the thoracic and lumbar spine than those reported in the cervical spine.[8] The work from Wolfla *et al*. in the cat, showed a sinusoidal association between cervical ventral epidural pressure and canal compromise, where the spinal cord was able to compensate, with the little appreciable rise in epidural pressure, for the first 20% of canal compromise. With further compression, they reported a steep rise in cord pressure. Differences between our findings and Wolfla *et al*. could be due to anatomical differences between animal models.

Our study shows that ventral epidural pressure rises more rapidly during the initial spinal canal compromise, with pressures incrementally showing smaller increases in pressure as the screw advanced. Furthermore, our absolute pressures were also much less than previously reported. This may be associated to the model used in different studies. Yet, the most important finding in this study remains that any amount of canal compromise will affect epidural cord pressure.

Future directions

This study used a modified DHS screw to produce controlled graded spinal cord compression. The authors plan future experiments to evaluate the effects of laminectomy following acute and sub-acute SCIs, in the anesthetized animal. In particular to investigate if a single level laminectomy could completely restore ventral epidural pressure in the thoracic and lumbar spine. In addition, it would be further beneficial to explore tissue damage following spinal cord compression.

CONCLUSIONS

Graded spinal cord compression of *in vitro* mini-pig spinal columns demonstrates a rapid rise in spinal cord pressures with even small spinal canal incursions. The pressures generated could injure the spinal cord *in vivo*, but *in vivo* experiments should be carried out to further validate this model of cord injury.

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Conflicts of interest

There are no conflicts of interest.

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