Cerebrospinal fluid pressures during dynamic contusion-type spinal cord injury in a pig model

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Introduction and Objective
Despite early recognition of a potential role of cerebrospinal fluid (CSF) in spinal cord injury (SCI), the interactions between CSF, spinal cord and dura are poorly understood. Fluid impulses in the CSF may cause neural tissue damage at sites remote to the injury site. The objective of this study was to develop a pig model of SCI featuring human-like dimensions and injury parameters, and to measure CSF pressure at several locations along the spinal cord length, at the instant of SCI.

Methods
CSF pressure was measured at four spinal levels in anaesthetized pigs (n=12, mean 20.9 kg) at the instant of injury, using injury parameters that simulate a medium and high severity human burst fracture. A multilevel laminectomy was performed to expose the thoracolumbar dura and spinal cord. Four miniature fibre-optic pressure sensors were inserted into the intrathecal space, 30 and 100 mm cranial and caudal to the injury site. Using a custom designed pedicle mounted device, a 20 g load-cell instrumented weight was released from 32 cm (low severity) and 125 cm (high severity) onto the exposed cord. Displacement of the weight tip was monitored with high speed video. Animals were euthanized 14 hours post-injury and the spinal cord was harvested for histology. The protocol was approved by the UBC Animal Care Committee.

Results
Preliminary analysis of data for the high severity injury group has been completed (n=6) thus far. Mean impact velocity was 4.73 ± 0.15 m/s, mean peak spinal cord compression was 7.66 ± 1.3 mm and peak impact load was 64.8 ± 12.5 N. The peak pressures at 30 mm from the epicenter ranged from 615.6 to 1430.4 mmHg (mean 957.2 mmHg). At 100 mm from the epicenter, peak CSF pressures ranged from 18.5 to 93.2 mmHg (mean 48.7 mmHg).
Conclusions
Currently, there is no established pressure injury tolerance for neural tissue; however, the CSF pressures measured in this study are of the same order of magnitude, or higher than, those shown to cause functional deficits and cell damage in rat brains subjected to blast overpressures. This suggests that animal, cadaver and computational models that seek to elucidate the biomechanics of SCI should consider including the effects of transient CSF pressure impulses.