Cerebrospinal Fluid Pressure in an *In Vivo* Porcine Model During Simulated Whiplash Loading of Different Severities

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I. INTRODUCTION

Whiplash injury is a common automotive injury that can lead to chronic pain in some patients [1-2]. The whiplash injury mechanism remains poorly understood, and no lesion is yet accepted as the primary source of all symptoms [3]. One theory proposes that the rapid retraction and extension of the neck during a whiplash perturbation causes transient pressure gradients in the cervical spinal canal [4]. These pressure gradients are theorised to generate shear stresses and strains on the afferent nerve cells in the dorsal root ganglia (DRG) of the cervical spine. Others have observed pressure transients in the cervical spine and evidence of cell membrane damage in the DRG using an *in vivo* porcine whiplash model [5-6]; however, how these pressure transients vary with the head and neck kinematics remains unknown. The aim of this study was to investigate the relationship between whiplash head/neck kinematics and the cervical cerebrospinal fluid (CSF) pressure.

II. METHODS

Ethics approval was obtained from the University of British Columbia Animal Care Committee (A19-0290). Four female Yorkshire pigs (22.6 ± 1.6 kg) were anaesthetised, intubated and mechanically ventilated. To measure CSF pressures, three fiber-optic pressure transducers (FOP-LS-2FR-20, FISO, Quebec, Canada) were inserted through a spinal catheter at T11 or T12, advanced cranially in the subarachnoid space, and positioned at the levels of the C2, C5 and C7 vertebral bodies (Fig. 1A). To measure head kinematics, three orthogonal linear accelerometers (7265A, Endevco, Irvine, USA) and an angular rate sensor (ARS Pro-1500, DTS, Seal Beach, USA) oriented to measure flexion/extension were rigidly mounted to the frontal bone (Fig. 1B). Signals were hardware filtered (CFC-1000), recorded at 10 kHz, and digitally low-pass filtered (4-pole Butterworth) at 100 Hz.
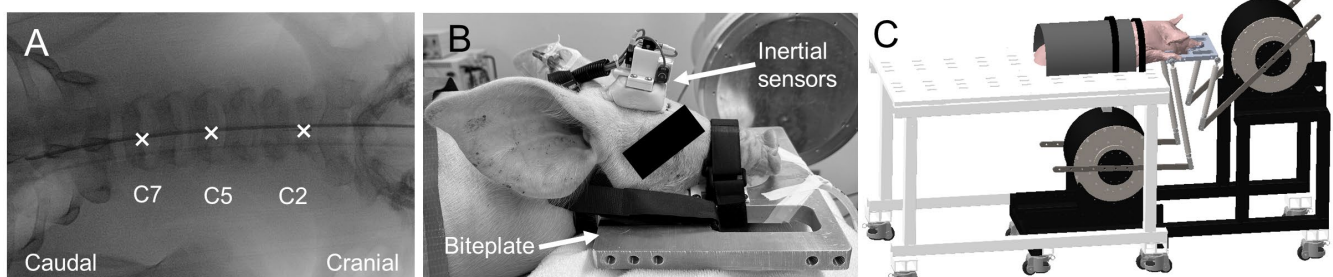

Fig. 1. A: Dorso-ventral fluoroscope of cervical spine with locations of the C2, C5, and C7 pressure transducers indicated. B: Photo of biteplate and head instrumentation. C: Schematic of whiplash device. The animal was secured to the table and positioned with its head cantilevered off the table edge and secured to the biteplate.

The animals were placed on a test table with the torso secured to the table (Fig. 1C) and a biteplate was adhered to the upper and lower dentition using polymethyl methacrylate and fastened to the head using straps (Fig. 1B). The whiplash device consisted of two rotary servomotors and a series of mechanical linkages attached to the biteplate. The motors could be programmed to generate different combinations of translation and rotation of the biteplate to simulate different combinations of head/neck retraction and extension. In this study, whiplash exposures consisted of 60° head extension with no initial retraction. Each animal experienced 10 whiplash exposures of increasing severity by incrementally reducing the time to reach maximum extension from

800 ms to 100 ms. In addition, an intermediate severity whiplash exposure (time to reach maximum extension = 130 ms) was repeated three times to characterise the repeatability of the kinematic and pressure measurements. The animals were humanely euthanised with intravenous sodium pentobarbital following testing.

III.   INITIAL FINDINGS

Exemplar head kinematics and CSF pressure for one animal are presented here (Fig. 2). The CSF pressures at all three spinal levels followed a similar pattern throughout the head-neck motion (Fig. 2A). Peak CSF pressure occurred shortly after maximum extension and increased with increasing whiplash severity (i.e. shorter times to maximum extension). Peak CSF pressure was consistently higher at the C5 level compared to the C2 and C7 levels across different whiplash exposure severities (Fig. 2B). Qualitatively, there was excellent repeatability in the head kinematics and CSF pressure across the three repeated tests (Fig. 2C).
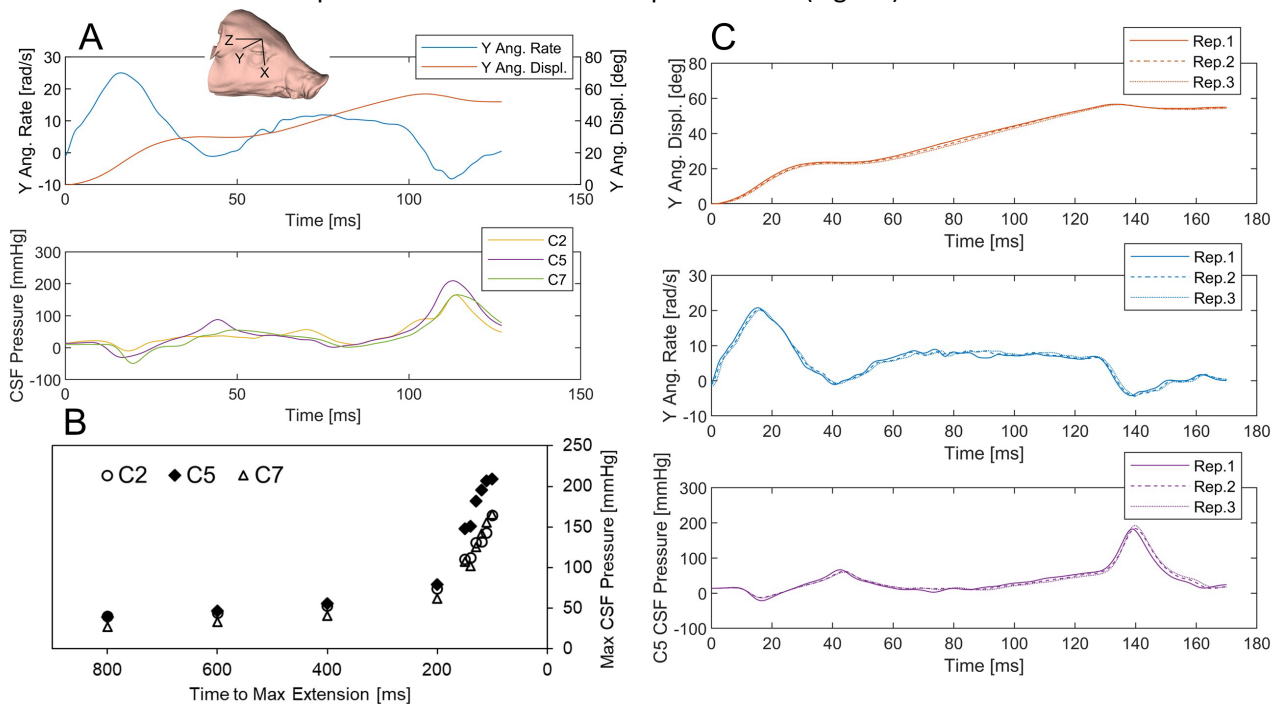

Fig. 2. A: Angular rate and displacement of the head and CSF pressure traces for one severe whiplash exposure. B: Maximum CSF pressure for whiplash exposures increased with increasing severity (represented here by shorter times to reach maximum extension). Note the horizontal axis is reversed. C: Head angular displacements, angular rates and C5 pressures for three repeated trials at one intermediate whiplash severity.

IV.   DISCUSSION

These preliminary results show that this whiplash injury model can produce repeatable head kinematics and CSF pressure mechanics, and can be used to investigate the effect of kinematic parameters on spinal canal pressure. Peak CSF pressure increased non-linearly as the time to reach maximum extension was decreased (surrogate for increasing whiplash severity). These observations are limited to a single animal and future work is needed to determine if these patterns hold across more subjects and to explore how different combinations of retraction and extension contribute to the CSF pressures at the different spinal levels. These data will improve our understanding of the DRG whiplash injury mechanism, provide information to validate computational models, and ultimately help inform the design of interventions for whiplash injury prevention in humans.

V.   ACKNOWLEDGEMENTS

VI. REFERENCES

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