Spinal Cord Displacement is Increased in Subjects with Cervical Spondylotic Myelopathy Compared to Controls

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Introduction: Cervical spondylotic myelopathy (CSM) is the leading cause of spinal cord (SC) dysfunction in people over 55 years of age in North America. With CSM, cord damage is caused by narrowing ("stenosis") of the spinal canal. Normally during the cardiac cycle, the SC initially moves in a caudal direction and then recovers in the cranial direction [1,2]. However, the relationships between cord motion and the extent and severity of spinal stenosis or CSM symptoms are not well known. The purpose of this study was to compare spinal cord displacement patterns between CSM subjects and controls.

Methods: ***Subjects*** Thirteen subjects (6M, mean age 62, range 50-77y) diagnosed with CSM, as well as fifteen age and gender matched controls (6M, mean age 58, range 50-73) underwent MRI, electrophysiological and clinical evaluations. The severity of CSM was ranked using the Japanese Orthopedic Association (JOA) scoring system. T_2 hyperintensities on the T_2WI were visually evaluated by two radiologists below, above and at the level of stenosis. The compression ratio (AP distance/RL distance) of the cord was also measured at the stenosis for CSM subjects and at C5 for controls.

MR Examinations and Analysis MRI was acquired on a Philips 3.0T Achieva with a phased array spine coil using only the first four channels. Velocity imaging was performed in the cranial-caudal direction using a 3D phase contrast sequence retrospectively gated to the peripheral pulse, with two averages, using an acquired matrix of 256×192×5, FOV 140×140×25 mm^3. The single stack was oriented perpendicular to the SC at the level of the stenosis for CSM subjects, and at the C5 level for controls. Regions of interest (ROIs) were drawn around the SC for each slice. Mean absolute velocity throughout all cardiac time points was determined for each ROI and then averaged over all slices. Cord displacement was calculated as the area under the velocity curve. Two measurements were extracted for each person: maximum cord displacement and cord displacement value at 50% of the heart cycle. CSM subjects and controls were compared using a two-tailed Student's t-test. Displacement was also correlated to clinical scores using a Pearson correlation. Significance was set at $P<0.05$.

Electrophysiological Examinations and Analysis Tibial and ulnar nerve somatosensory evoked potentials (SSEP) were measured in the right and left side for all participants [3]. Changes in amplitude, latency (corrected for body height) and configuration were used to score and classify the recordings as normal or abnormal.

Results: The mean JOA score in CSM subjects was 15.1 (range 9.5-17). All controls had a perfect normal JOA of 17 except for one with 15.5. All CSM subjects but one had T_2 hyperintensities whereas none were found in controls. The mean compression ratio in CSM subjects was 0.34 (range 0.18-0.69) and in controls was 0.45 (range 0.37-0.69). Spinal cord displacement plots are shown for all controls and subjects across the heart cycle separated into those with normal and those with abnormal SSEP in Figure 1. Controls showed a fairly small displacement whereas some CSM subjects had large changes in displacement. Maximum displacement and displacement at 50% of the heart cycle were significantly different between controls and CSM subjects. Furthermore, maximum displacement and displacement at 50% of the heart cycle were able to separate controls with normal SSEP from CSM subjects with normal and abnormal SSEP. Displacement at 50% of the heart cycle was able to further separate controls with normal SSEP from controls with abnormal SSEP (Figure 2). No significant correlations were found between maximum displacement or displacement at 50% of the heart cycle and JOA scores, compression ratio or the presence of T_2 hyperintensities.

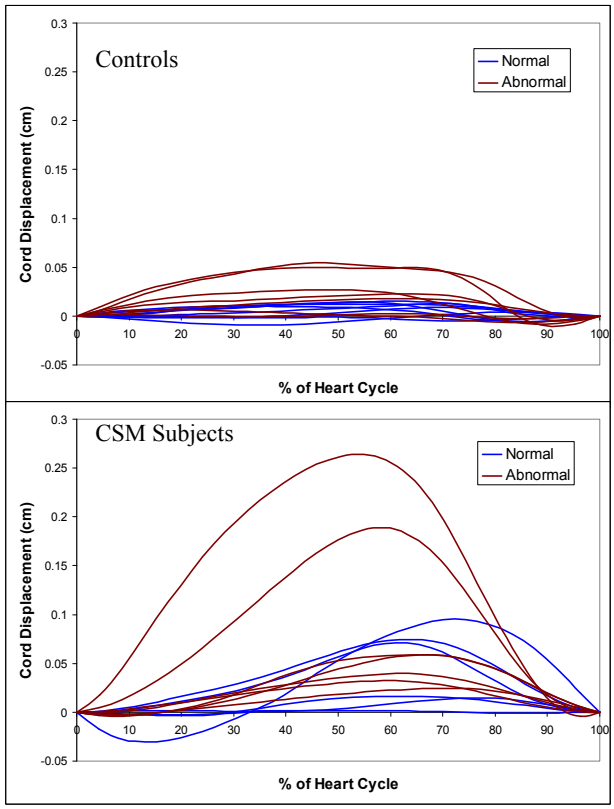

Figure 1: Plots of spinal cord displacement over the heart cycle for control subjects (top) and subjects with CSM (bottom). The subjects are further subdivided into those with normal and those with abnormal SSEP values.

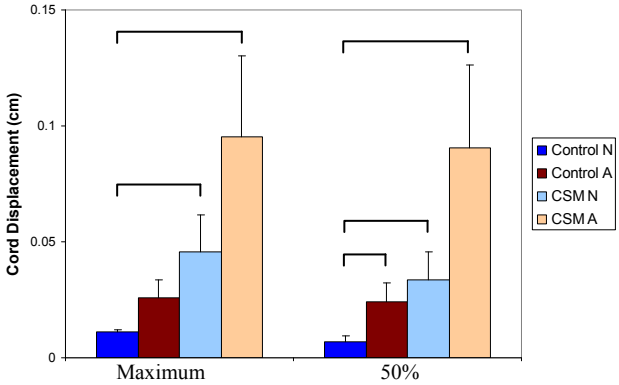

Figure 2: Maximum cord displacement and cord displacement at 50% of the heart cycle for control subjects and subject with CSM. The subjects are subdivided into those with normal (N) and abnormal (A) SSEP values. Significant differences are shown ($P<0.05$). Error bars indicate standard error.

Discussion and Conclusion: The results confirm that in controls the spinal cord is undergoing a limited cranio-caudal movement (here measured as cord displacement) that is closely related to the heart cycle and is considered to be induced by the CSF flow [4]. The unexpected increase in cord displacement in patients with CSM could be explained by changes in CSF flow characteristics caused by the spinal canal stenosis. Interestingly, abnormal SSEP was associated with an increase in SC movement while clinical measures of functional impairment were less related. Increased SC movements may relate to underlying pathophysiological mechanisms in patients suffering from CSM that contribute to the deterioration of SC function. Further research into the reason for the increase in cord displacement and whether the increase in some CSM subjects could be used as a predictor of clinical course or of response to treatment is warranted.

References: [1] Levy, Radiology, 1988. [2] Mikulis, Radiology, 1994. [3] Hausmann, European Spine Journal, 2003. [4] Enzmann, Radiology, 1992.

Acknowledgements: Thanks to the volunteers and technologists at the UBC MRI Research Centre and Juliet Batke at ICORD. We thank Philips Healthcare for their continued research support. Funding was provided by the Cervical Spine Research Society and the Michael Smith Foundation for Health Research.