ASSESSMENT OF CSF FLOW AND SPINAL CORD MOTION IN CERVICAL SPONDYLOSIS USING PHASE CONTRAST MRI

S. M. Meyers\textsuperscript{1,2}, E. L. MacMillan\textsuperscript{3}, B. Mädler\textsuperscript{4}, I. M. Vavasour\textsuperscript{5}, D. K. Li\textsuperscript{5}, M. F. Dvorak\textsuperscript{6,7}, A. Curt\textsuperscript{6,8}, and A. L. MacKay\textsuperscript{5,9}

\textsuperscript{1}University of British Columbia, Vancouver, BC, Canada, \textsuperscript{2}Physics, University of Alberta, Edmonton, Alberta, Canada, \textsuperscript{3}Dept. of Clinical Research, University of Bern, Bern, Switzerland, \textsuperscript{4}Philips Healthcare, Vancouver, BC, Canada, \textsuperscript{5}Dept. of Radiology, University of British Columbia, Vancouver, BC, Canada, \textsuperscript{6}International Collaboration on Repair Discoveries, Vancouver, BC, Canada, \textsuperscript{7}Dept. of Orthopaedics, University of British Columbia, Vancouver, BC, Canada, \textsuperscript{8}Dept. of Neurology, University of British Columbia, Vancouver, BC, Canada, \textsuperscript{9}Dept. of Physics & Astronomy, University of British Columbia, Vancouver, BC, Canada

Introduction

Cervical spondylotic myelopathy (CSM) is the leading cause of spinal cord dysfunction in people over 55 years of age in North America. With CSM, cord damage is caused by narrowing of the spinal canal, called a stenosis, which can also alter the flow of cerebrospinal fluid (CSF) through the canal \cite{1,2}. However, the relationships between CSF flow, cord motion and degree of compression or CSM symptoms are not well known. The purpose of the study was to compare velocity patterns of CSF and cord between CSM subjects and controls.

Methods

MRI Experiment: 14 subjects diagnosed with CSM as characterized by established clinical deficits (6 female, 8 male, mean age 63.1 years, range 50-77 years) and age-matched controls (11 female, 7 male, mean age 59, range 51-75) were recruited in accordance with the local institutional review board. They were scanned on a 3.0T MRI system (Philips Healthcare, Best, The Netherlands) 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, and an acquired matrix of 256×192×5, FOV 140×140×25 mm\textsuperscript{3}. Total scan time averaged 5 minutes, depending on an individual’s heart rate. The single stack was oriented perpendicular to the spinal cord at the level of the stenosis for CSM subjects, and at the C5 level for controls.

Data Analysis: Maps of absolute value of velocity, maximum velocity, and standard deviation of velocity over all cardiac time points were calculated for each pixel. Regions of interest (ROIs) were drawn around canal and cord for each slice on magnitude images, with reference to standard deviation maps in order to help distinguish structures. Mean absolute velocity and maximum velocity throughout all cardiac time points were calculated within each ROI, and were then compared between CSM subjects and controls using a two-tailed Student’s t-test. As well, the mean velocity within a volume of interest (VOI, created by combining each ROI along 5 slices) was plotted along the heart cycle for each CSM and control subject.

Results

Mean and max CSF velocity were significantly higher for controls than CSM subjects, whereas mean cord velocity was significantly lower for controls (p-values < 0.0001). No significant difference was found for max cord velocity. Velocity plots along the heart cycle support t-test results: plots of CSF velocity were much shallower for CSM subjects than controls (Fig. 1). The cord velocity in CSM subjects generally had higher peaks than those of controls, and most exhibited the same flow pattern as regular CSF flow (Fig. 2). The cord velocity in control subjects was irregular and shallow, with velocity changing direction more frequently.

Conclusions

This study marks the first time that both CSF flow and cord motion have been measured in CSM by phase contrast imaging. These results clearly indicate a decrease in CSF flow and an increase in spinal cord motion in CSM subjects, which may suggest that spinal cord motion is a consequence of the lack of CSF flow.

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References