Phase-Contrast MRI of Pulsatile Cerebrospinal Fluid Flow in Patients with Cervical Spondylitic Myelopathy.

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Introduction

Cervical Spondylotic Myelopathy (CSM) is one of the most common spinal cord disorders particularly in elderly patients, however the natural history of this condition is poorly understood with only weak correlation between spinal cord compression seen in MRI scan and presence or severity of clinical myelopathy. Early diagnosis is very important as once neurological deficits have developed they are unlikely to improve. The precise pathophysiology of CSM is not very clear however narrow spinal canal, presence of compressing spondylotic spurs and spinal cord ischemia are believed to be important factors. Previous investigations with MRI have shown a strong correlation between spinal cerebrospinal fluid (CSF) area and presence of clinical myelopathy (1) and severity of myelopathy and disturbance of pulsatile CSF flow (2). A suspicion that CSF flow abnormalities and pressure fluctuations may be a contributing factor towards development or progression of CSM led us to investigate the relationship between clinical and subclinical myelopathy (subjective myelopathy) and CSF flow in the cervical spinal canal as detected with phase contrast MRI.

Method

Three subject groups consisting of 9 patients with cervical spondylitic myelopathy, 9 patients with subjective myelopathy and 30 normal volunteers were subjected to phase-contrast MRI to study the dynamics of pulsatile cerebrospinal fluid (CSF) flow. All subjects were imaged on a 1.5T Phillips MR system. A retrospectively gated phase-contrast sequence (TR 25.5 msec, TE 10.6 msec, flip angle15°, velocity encoding profile 5 cm.s-1, field of view 160 mm, matrix 256 x 256, slice thickness 6 mm) was used to measure flow velocity for 15 time points in the cardiac cycle. For the group with clinical myelopathy flow measurements were performed above, at and below the level of stenosis. In the remaining subjects measurements were performed above, at and below the C4-C5 interface. Definition of the CSF space within the images was performed by manual selection of a small rectangular region of interest (ROI) in the centre of an area of high flow. The mean grey level (and hence flow velocity) for this ROI was measured for each time point in the cardiac cycle. The resultant time curve of mean velocity was then correlated with the corresponding time curve for every pixel in the image set. This resulted in a correlation image, where each pixel represented the correlation coefficient r of the waveform for that pixel location, with the waveform of the selected ROI. This image was then thresholded at an empirically determined value of 0.8 to determine the boundaries of measurable CSF space. Flow through this space was calculated from heart rate, mean velocity and cross sectional area.

Results and Discussion

A CSF waveform for each scan was extracted by plotting the mean flow velocity against timepoint (fig. 1). Waveforms were successfully extracted for normals (n = 29), subjective myelopaths (SM) (n = 8) and clinical myelopaths (CM) (pre-stenosis n = 7, mid and post stenosis n = 6). The procedure failed in 2 CM due to absence of measurable flow. The amplitude of this waveform represented the range between maximum caudal (+ve) and maximum cranial flow. Inter-group comparison showed no significant difference between groups in the uppermost image (pre-stenosis for CM). However there were significant intergroup differences at the stenotic and post stenotic levels (fig 2). A posteriori tests for intergroup differences (Tukey's honestly significant difference criterion) showed significant differences between normals and CM at both levels (p = 0.001, mid, p = 0.005, post). SM patients showed a similar trend to CM but with less pronounced flow reductions and this reduction was significantly different from normals only at the stenotic level (p = 0.05). Although mid and post stenotic flow was substantially reduced in the CM group flow above the stenotic segments does not appear to be reduced which would suggest an increase in CSF pressure just prior to the stenosis. The proportion of the cardiac cycle in which CSF flow was in the caudal direction showed no significant difference between any group or image, however for the first pre-stenosis image, proportional time from commencement of caudal flow to peak flow was significantly shorter (p = 0.05, Kruskal-Wallis) in the CM group than in the other groups. This shortening of the time period could be indicative of a build up of pressure due to the limited passage of CSF through the stenotic section.

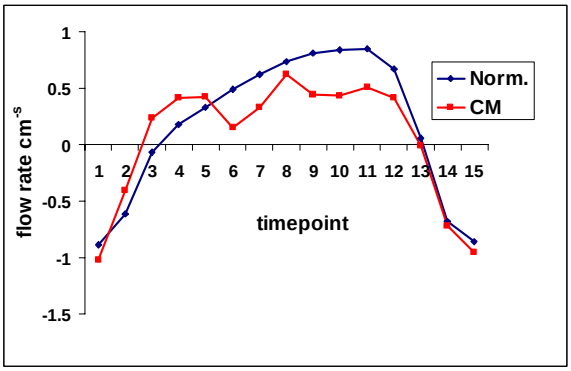

Figure 1. Mean CSF waveform for all subjects in groups normals and CM

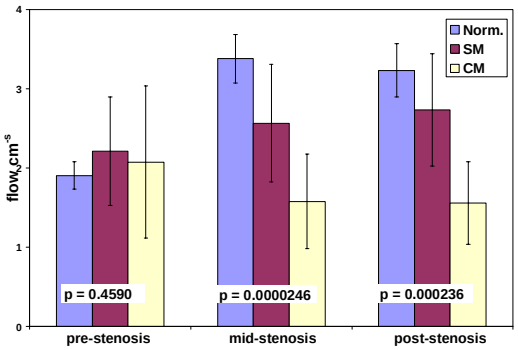

Figure 2. Mean flow range between different groups error bars are 95% confidence levels for mean.

Conclusions

The demonstration of reduced CSF flow at and below stenotic levels in patients with CM could represent an epiphenomena secondary to stenosis. The identification of similar changes in patients with minimal stenosis and subjective myelopathy supports the hypothesis that disturbance of CSF flow has a mechanistic involvement in the aetiology of myelopathic symptoms and signs. The study also shows that measurements of CSF flow can have clinical value in the assessment of patients with apparent myelopathic symptomatology in the absence of solid supportive physical signs.

References

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