Improved sensitivity of spinal fMRI by using physiological recordings in general linear model analysis

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Introduction

Functional magnetic resonance imaging of the spinal cord by means of proton-density weighted spin-echo imaging, has been demonstrated to provide reliable maps of activity, particularly when combined across groups of subjects. Individual maps demonstrate consistent areas of activity as well as some inconsistent activity across repeated studies. Studies of the reproducibility of spinal fMRI results have shown that false negative results (i.e. missed activity) occur more frequently than false positive results, suggesting that there are sources of signal fluctuation that are masking the neuronal-activity-related signal changes. The dominant source of these errors is therefore hypothesized to be motion of the spinal cord, driven by blood and CSF flow, and field variations due to changes in lung volume. Hence the hypothetical sources of error are all related to cardiac and respiratory motion. The purpose of the present study is to test this hypothesis by modelling the motion-related signal changes and using these models as part of the basis set in a general linear model.

Methods

Spinal fMRI studies were carried out in a 3 T Siemens Magnetom Trio using a phased-array spine receiver coil with subjects lying supine. Cold thermal stimulation of the palm of the hand was used to elicit activity in the cervical spinal cord, by means of a Medoc® TSA-II thermal sensory analyzer. Functional image data were acquired with a half-fourier single-shot fast spin-echo sequence (HASTE) with the echo time set at minimum (33 msec) to obtain essentially proton-density weighted images. Signal intensity changes observed upon a change in neuronal activity were the result of signal enhancement by extravascular water protons (SEEP), as described previously.[1,2] Sagittal image data were acquired centered on the C7 vertebra, with an 18 cm x 9 cm FOV, with a 192 x 96 matrix, of 2 mm thick contiguous sagittal slices. Spatial suppression pulses were applied to eliminate signal from anterior to the spine and to eliminate aliasing. The peripheral pulse and respiration were recorded continuously during each study. The resulting three-dimensional image data were analyzed with custom-made software written in MatLab, and physiological recordings were sampled at the time of acquisition of each slice, and at various time lags, and were included in the basis set used for analysis with the general linear model.

Results and Conclusions

Spinal fMRI data consistently demonstrated areas of activity in the spinal gray matter that correspond well with the spinal cord neuroanatomy. Sensory and apparent reflex activity were demonstrated in the cervical spinal cord, and areas of activity were also consistently observed in the medulla, as in previous studies. Comparisons of results obtained when the GLM basis set included 1) the peripheral pulse, 2) respiration, 3) both pulse and respiration, or 4) neither, demonstrated improvements in the consistency of results across subjects when the physiological traces were used. Improvements were assessed qualitatively on the consistency of results with the spinal cord neuroanatomy, and the consistency across subjects. Error results appearing in the CSF or white matter are clearly identifiable. The greatest improvements appeared to be obtained with the use of the peripheral pulse traces alone in the GLM, with little difference noticed with the use of respiration traces either alone or in combination with the pulse traces. These results indicate that movement driven by the heart-beat is likely the dominant source of errors in spinal fMRI, and that its contribution can be reduced as described above. However, characterization of the spinal cord motion will likely improve the effectiveness of this method and significantly improve the reliability of spinal fMRI results. This finding is consistent with the observation that cardiac gating improves the reliability of spinal fMRI results as well.[3]

1. Stroman, P. W. et al. Magn Reson.Med. 48: 122-127, 2002.
2. Stroman, P. W. et al. NeuroImage 17: 1854-1860, 2002.
3. Brooks, J. et al. American Pain Society 23rd Annual Meeting, Vancouver, May 6-9, 2004: 667-2004.