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Hypercapnia and Hypoxia in the Human Brain: Effects on Resting and Activation - Induced MRI Signal

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PURPOSE:

Susceptibility - weighted MRI signal in the human brain is observed during combinations of task activation, hypercapnia, and hypoxia. The purpose is to further characterize the hemodynamic mechanisms which contribute to activation - induced MR signal changes.

A method, stemming from these studies, which may enable more accurate localization of neuronal activity from functional MR images, is demonstrated.

INTRODUCTION:

Previous studies (1-7) suggest that global hemodynamic stresses of ischemia, apnea, hypoxia, and hypercapnia cause changes in MR signal, under non - flow sensitized conditions, primarily through changes in hemoglobin susceptibility. These signal changes differ regionally due to differences in blood volume, oxygenation, and/or vascular patency. Our hypothesis is that task-induced signal changes are transduced through the same hemodynamic mechanisms as global hemodynamic stresses such as hypercapnia. We test this hypothesis by observing the effects of hypercapnia and hypoxia on resting brain signal and on activation-induced signal changes. In addition, we compare, voxel by voxel, the changes in signal caused by the stresses with the changes caused by task activation.

One difficulty in interpretation of neuro-functional MR images is that variations in vessel architecture (eg: large draining veins) lead to uncertainty of true foci locations and distributions. We demonstrate a method by which regional signal differences due to differences in vessel architecture are removed from neuro-functional MR images by normalization to a mask created by a global hypercapnic stress.

METHODS and RESULTS

Sequential single shot gradient-echo echo planar images containing the motor cortex were obtained (resolution = 3.75 x 3.75 x 5 mm^3, TE = 40 ms, TR = 2 sec) from 3 healthy subjects using a Signa 1.5 T scanner, a 30.5 cm i.d. three-axis local gradient coil, and a whole brain endcapped quadrature birdcage rf coil. First, cyclic finger tapping alone was performed. Pixels demonstrating activation were used for comparison. Hypercapnia (breathing 5% CO_2) and hypoxia (breathing 12% O_2) were induced in subsequent time course series during which the subjects either rested or performed cyclic finger tapping.

Figure 1, shows that while the overall signal is increased by hypercapnia, a strong vasodilator, the activation - induced amplitude is strongly damped. While the overall signal is decreased by hypoxia, the activation-induced signal change is only slightly damped.

A voxel-wise comparison between activation - induced signal changes and hemodynamic stress - induced changes, shown in Figure 2, demonstrate that they are roughly proportional to one another, suggesting that they share the same contrast mechanism.

Assuming that hypercapnia and neuronal activation cause similar hemodynamic events; one global and the other local, then, division of a "percent change during brain activation" image by a "percent change during hypercapnia" image would give a ratio map of task-induced signal activation which is normalized to the signal change accompanying global vasodilation. Because perfusion changes accompanying hypercapnia can be quantified, there exists the exciting possibility of creating quantitative maps of perfusion changes accompanying task activation.

In this study, division of the activation image by the hypercapnia image caused the largest signal changes to move from the sagittal sinus to a region in cortex.

CONCLUSIONS:

We observed that hypoxia and hypercapnia caused opposite global signal changes, likely to be proportional to blood volume, and that hypercapnia significantly damped activation - induced signal changes, due to a competing vasodilation effect. Task induced signal changes appear to be coming from similar contrast mechanisms as signal changes caused by global hemodynamic stresses. Normalization of neuronal activation images to a hypercapnia mask removed signal variations caused by regional differences in blood volumes and caused a shift in activation foci from regions of apparently large blood volume to cortical regions.

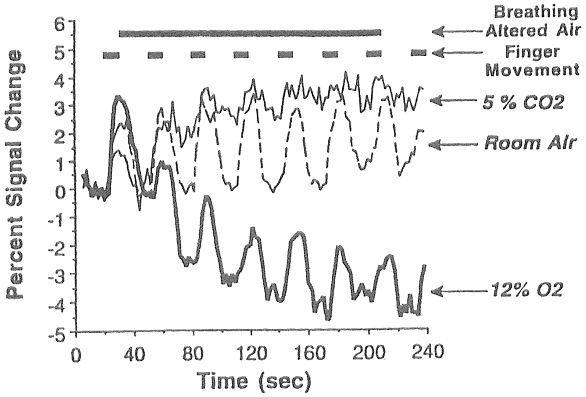

Figure 1: Plots from a 15 voxel region in motor cortex. Cyclic finger tapping was performed during breathing of either room air, air with 5% CO_2, or air with 12% O_2. Horizontal bars indicate when gasses were administered and when finger tapping took place.

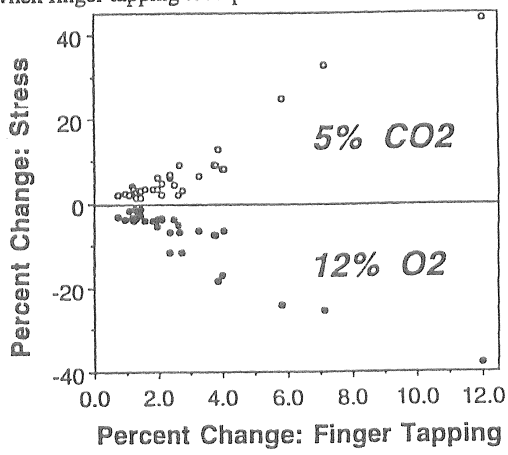

Figure 2: Voxel-wise comparison from 32 voxels in motor cortex during separate trials of finger tapping (room air), hypoxia (no tapping), and hypercapnia (no tapping).

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