

Imaging Cerebral Blood Flow Changes Due to Pain with Arterial Spin Labeling

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Introduction: To date, functional magnetic resonance imaging (fMRI) studies involving pain have used blood oxygen level-dependent (BOLD) contrast to measure the neural activity generated in response to nociceptive stimuli. However, BOLD has certain limitations with respect to pain imaging. It cannot image chronic pain, and it is not suited for low-frequency tasks [1], such as changes in pain caused by medication. Arterial spin labeling (ASL) does not suffer from these limitations, but the reduced sensitivity limits its use in pain imaging as BOLD studies have demonstrated that the signal changes with respect to pain are significantly smaller than with sensorimotor or visual tasks. However, this limitation can be overcome by using recent technical developments, such as higher field magnets and phased array coils. The purpose of this study was to determine if ASL has the sensitivity to image neural activity associated with acute pain.

Materials and Methods: A multi-slice, spiral ASL sequence was used to collect CBF images on a 3T magnet equipped with an 8-channel, receive only, phased array coil (GE medical systems) [2]. Ten axial perfusion-weighted images were acquired every 6 s with an in-plane resolution of 3.75 mm and a slice thickness of 6 mm. Noxious thermal pain (task) and innocuous warmth (rest) were delivered to the palm of the left hand in alternating periods of one minute, for a total of 11 minutes per session. Each subject underwent either 2 or 3 sessions. Image processing and statistical analysis was performed with SPM2. Six normal, healthy, male volunteers aged 24-27 years took part in this study.

Results: Table 1 shows which of the brain regions typically associated with pain were activated in this study. Activation was seen consistently in the insular and secondary somatosensory cortices, predominantly on the side contralateral to the stimulus. Activation of the primary motor cortex is attributed to the fact that each subject rated their pain continuously during each session by moving a sliding scale with their right hand. Figure 1 shows the activation map for one subject. Visible on the sagittal image are activations in the thalamus, cingulate cortex, and primary motor cortex, while cingulate and bilateral insular cortex activations can be seen on the coronal image.

Discussion: Due to the results of numerous positron emission tomography (PET) and BOLD studies involving painful stimuli, certain brain areas have become associated with the experience of pain. These include the insular regions, anterior cingulate cortex, thalamus, as well as the primary and secondary somatosensory cortices [3]. The work presented here demonstrates that ASL is able to detect activations in most of these pain-related areas in response to an acute stimulus. The next part of this study is to compare the activation from ASL with that from BOLD. The ability of ASL to detect pain activation offers the possibility of studying chronic pain conditions, which is an area of pain research that has not been studied with fMRI due to the above-mentioned limitations with BOLD imaging

| Subject | Insula/S2 | Thalamus | M1 | S1 | Cingulate |
|---------|----------------------|----------------------|-----------|----------|-------------------------------------|
| 1 | R: (11.8) | B: (12.7) | L: (12.7) | R: (9) | Anterior: (11.5) Central: (15.2) |
| 2 | L: (5.7) | | L: (5.2) | | |
| 3 | R: (4.9) | R: (5.0) L: (4.4) | L: (7.4) | | |
| 4 | R: (4.9) | R: (4.8) L: (5.5) | L: (6.7) | R: (7.8) | |
| 5 | R: (4.6) | | | R: (8.0) | |
| 6 | R: (5.8) L: (8.0) | | L: (7.2) | R: (8.8) | Central: (6.5) |

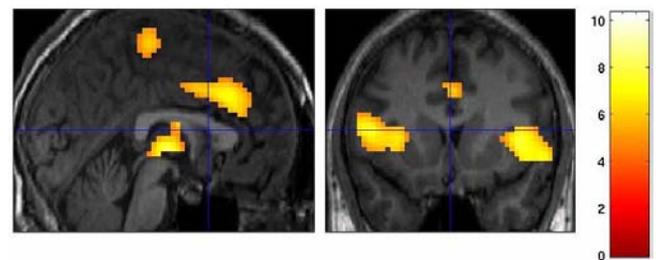


Fig. 1 Statistical parametric map (SPM) of pain activation for one subject. Activated pixels are suprathreshold t-statistics, corrected for multiple comparisons.

Table 1 Locations of activation in response to pain. M1: primary motor, S1/S2: primary/secondary somatosensory. R: Right, L: Left, B: Bilateral. Numbers in brackets are percent CBF change.

References

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- (2) St. Lawrence et al., *MRM* 2005;53(3):735-738
- (3) Peyron et al., *Neurophysiol Clin* 2000;30:263-288