FMRI during spinal cord stimulation in chronic pain patients

J. Tintera[1], J. Kozak[2], I. Vrba[3], J. Vrana[4], H. Polacek[4], R. Rokyta[4], I. Ibrahim[1], A. Stancak[4]
[1]Radiology, IKEM, Prague, CZ, Czech Republic, [2]Faculty Hospital Motol, Prague, CZ, Czech Republic, [3]Hospital Na Homolce, Prague, CZ, Czech Republic, [4]Charles University Prague, Prague, CZ, Czech Republic

Introduction

High-frequency electrical stimulation (40–100 Hz) of the dorsal spinal cord is used to alleviate intractable neuropathic pain. Since central mechanisms of analgesia during spinal cord stimulation (SCS) and the cerebral response to switching on the stimulator are not known, we performed fMRI study in 8 patients during SCS.

Material and methods

Study was conducted in 8 patients (3 women, 5 men, 39–52 years) suffering from intractable pain in one or both lower extremities after failed back surgery. Stimulation electrodes (Itrel, Medtronic, USA) were implanted in epidural space of the lumbar spinal cord. FMRI was performed using the trial period in the block design comprising three conditions each lasting 32 s: a) SCS, b) heat painful stimulation (HPS) applied to the leg ipsilateral to the positions of SCS, and c) simultaneous HPS and SCS.

Functional MRI was performed on 1.5T scanner (Siemens Vision) using GE EPI sequence (TR=4.5s, TE=54ms flip angle 90°, 40 slices of 3 mm thickness each, isotropic voxel size 3x3x3 mm^3). Functional data were analyzed in SPM2 using fixed-effect model and also using simple correlation statistic on individual subject level. Signal time curves in activated areas were analyzed.

Results

SCS-related activations in individual subjects indicated weak BOLD-signal change and large variability between subsequent stimulation periods and subjects. Large variability of activation changes might be related to latency effects and/or interactions with pain. One patient had to be excluded because of technical failure.

Nevertheless, statistically significant (uncorrected P=0.001) activation during SCS was found in dorsal medial primary motor cortex (M1, Brodmann area 4) corresponding to foot motor area in all patients (Fig. 1a). In group analysis, the left and right putamen were also activated in addition to M1. However, statistically significant de-activation (stronger BOLD signal during rest than during SCS) was also found in all patients. The de-activation clusters were found in the primary somatosensory cortex somatotopically corresponding to representation of the tight or hip (Fig. 1b).

De-activation in these cortical regions may correspond to decrease of pain during SCS, and could serve as an objective indicator of an effective SCS. The group statistics for activation and de-activation is demonstrated in Fig. 2.

Although pain-related activations were small in our group of patients, possibly due to post-operative antalgic treatment and the underlying neuropathy, the SCS-related activation in the foot motor area increased during heat pain stimulation (fig. 3).

Conclusions

Results indicate that analgesic effect of SCS may be related to heightened activation of the cerebral motor system involving primary motor cortex and striatum, and deactivation in primary sensory cortex. Interestingly, direct electrical stimulation or transcranial magnetic stimulation of primary motor cortex alleviates chronic pain suggesting a shared neuronal network underlying different neurostimulation methods.

Supported by IGA (NR-8232) and Research Goals (0021620816).

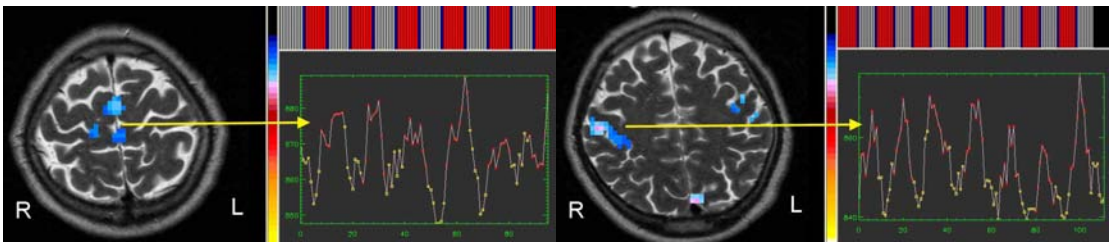

Fig. 1. a) Activation, b) de-activation and time dependency of signal in M1 and sensory cortex of subject 5.

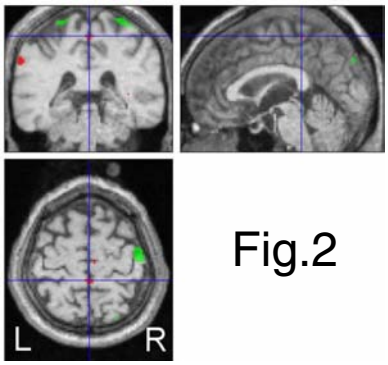

Fig.2

Fig. 2. Activation (red) and de-activation (green) during SCS for all patients.

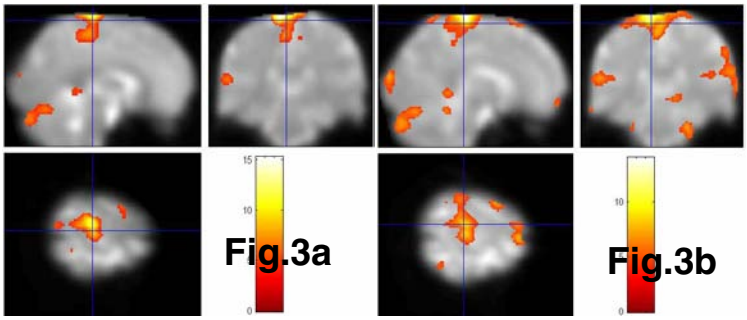

Fig. 3. a) Activation during SCS and b) combined SCS+HPS.