fMRI study of hypnosis-induced analgesia

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Introduction

The neural mechanisms underlying the antinociceptive effects of hypnosis remain badly understood. We here used fMRI to study the effect of hypnosis on thulium-YAG laser induced pain in volunteers.

Methods

13 healthy subjects underwent 2 randomized fMRI sessions, one in normal, and one in hypnotic state. During each session, 200 laser stimuli with intensity ranging from 300 to 600 mJ were administered on the left hand. Subjects rated their sensations as P0: nothing perceived, P1: perceived, non painful, P2: mild pain, P3: moderate pain, P4: intense pain. fMRI data were preprocessed and analyzed using statistical parametric mapping (SPM2). Analyses compared activation induced by matched intensity laser stimulation in normal and in hypnotic state. Results were thresholded at small-volume corrected p < 0.05 within the previously identified pain matrix.

Results

A significant difference in sensation was found between normal and hypnotic state for the painful intensity range of stimulation (mean score 1.9 ± 0.3 SD vs. 1.2 ± 0.4, respectively), but not for the non-painful range of intensity (mean score 0.5 ± 0.2 SD vs. 0.4 ± 0.3, respectively). In the normal state, high intensity (painful) compared to low intensity (non-painful) stimuli activated bilateral thalamus, primary somatosensory cortex (S1), insula, and anterior cingulate cortex (i.e., the pain matrix). In the hypnotic state, high intensity compared to low intensity stimuli only identified significant activation in S1. Bilateral thalamus, left insula and bilateral anterior cingulate cortex showed significant less activation in the hypnotic state as compared to the normal state.

Conclusion

Our parametric event-related fMRI study investigating the effects of hypnotic suggestion on pain intensity perception in healthy volunteers demonstrated decreased pain perception during hypnosis, while leaving sensory non-painful perception unaltered. This hypnosis-induced decreased pain perception correlates with a decreased activation in thalamus, insula and anterior cingulate cortex.

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