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Hypnotically induced somatosensory alterations: Toward a neurophysiological understanding of hypnotic anaesthesia

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ABSTRACT

Whereas numerous studies have investigated hypnotic analgesia, few have investigated hypnotic anaesthesia. Using magnetoencephalography (MEG) we investigated and localized brain responses (event-related fields and oscillatory activity) during sensory processing under hypnotic anaesthesia. Nineteen right handed neurotypical individuals with moderate-to-high hypnotizability received 100 vibrotactile stimuli to right and left index fingers in a random sequence. Thereafter a hypnotic state was induced, in which anaesthetic suggestion was applied to the left hand only. Once anaesthetic suggestion was achieved, a second, identical, session of vibrotactile stimuli was commenced. We found greater brain activity in response to the stimuli delivered to the left (attenuated) hand before hypnotic anaesthesia, than under hypnotic anaesthesia, in both the beta and alpha bands. In the beta band, the reduction of activity under hypnotic anaesthesia was found around 214–413 ms post-stimuli and was located mainly in the right insula. In the alpha band, it was found around 253–500 ms post-stimuli and was located mainly in the left inferior frontal gyrus. In a second experiment, attention modulation per se was ruled out as the underlying cause of the effects found. These findings may suggest that the brain mechanism underlying hypnotic anaesthesia involves top-down somatosensory inhibition and, therefore, a reduction of somatosensory awareness. The result of this mechanism is a mental state in which individuals lose bodily sensation.

1. Introduction

Esdaile (1846) first reported surgical operations performed under hypnotic anaesthesia. Since that time, empirical evidence for the efficacy of both hypnotic anaesthesia and hypnotic analgesia has accumulated, and the medical use of hypnosis has gained increasing acceptance. It is now becoming ever more popular in the treatment of several chronic medical conditions, including fibromyalgia, multiple sclerosis, irritable bowel syndrome, headache, sickle cell disease, spinal cord pain, disability related pain and cancer related pain (for a review see Dillworth and Jensen (2010)), as well as burn injury, heart disease, arthritis, dental problems and eczema (for a meta-analysis see Montgomery et al. (2000)).

Hypnosis typically consists of two processes: *induction* and *suggestion*. Induction comprises of a series of instructions aimed at achieving a focused and absorbed attentional state. Once hypnotic induction is achieved, hypnotic suggestion is applied (for a review on hypnotic suggestion see Oakley and Halligan (2013)). Hypnotic anaesthesia and analgesia occur at the suggestion stage and involve suggestion of the total or partial loss of somatosensory sensitivity. Whereas hypnotic anaesthesia involves suggestions directed at loss of sensory awareness, hypnotic analgesia involves suggestions directed at the reduced experience of pain (McConkey et al., 1999).

Whereas a great number of studies have examined the effects of hypnotic analgesia on pain processing, relatively few have examined the brain mechanisms underlying this effect, and so far as we know only three studies have aimed at investigating the brain mechanisms underlying hypnotic anaesthesia. Thus the current study attempted to shed further light on the brain mechanisms underlying the effects of hypnotic anaesthetic suggestion on sensory processes. Because both hypnotic anaesthesia and analgesia involve suggestion of the reduction in somatosensory sensitivity, and due to the relative paucity of studies examining the brain mechanisms underlying hypnotic anaesthesia, we will begin by reviewing the richer data-set regarding hypnotic analgesia, before

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closing in on hypnotic anaesthesia.

In a meta-analysis evaluating the efficacy of hypnotic analgesia, it was found beneficial in providing pain relief for 75% of participants (Montgomery et al., 2000). The effectiveness of hypnotic analgesia did not differ between clinical participants and neurotypical ones. However, it was found that the level of hypnotizability (the degree to which one is responsive to hypnotic suggestion) does influence the efficacy of hypnotic analgesia in such manner that hypnotic analgesia was only found to help pain-relief in individuals with medium or high levels of hypnotizability.

Despite the empirical evidence of the efficacy of hypnotic analgesia (excluding individuals with low levels of hypnotizability), and the growing popularity of the technique, it is surprising how little is known about its underlying brain mechanisms. A positron emission tomography (PET) study utilizing hypnosis to decrease (hypnotic analgesia) or increase (hyperalgesia) the unpleasantness of thermal pain perception found the neural activity in the anterior cingulate cortex to be modulated in accordance with the changes in thermal pain perception (Rainville et al., 1997). In a follow-up PET study conducted by the same group, hypnotic suggestion was used to decrease or increase the intensity of the perceived pain. It was found that when the intensity of pain increased, neural activity in the primary somatosensory cortex was also increased and when pain was decreased, via hypnotic analgesia, neural activity decreased in the same region. A similar trend was found in the secondary somatosensory cortex. In addition, the anterior cingulate cortex and the insula were found to be involved in the hypnotic suggestions used for the modulation of pain perception (Hofbauer et al., 2001).

Valentini et al. (2013) took a combined approach, examining both the intensity and the unpleasantness of the sensory stimuli in a single hypnosis experiment. The study used EEG to examine how hypnotic analgesia and hypnotic hyperalgesia (suggestion aimed at increasing either the intensity or unpleasantness of the stimuli) affected pain perception in the time and time-frequency domains, in individuals with both high and low levels of hypnotizability. Hypnotic analgesia was found to modulate both the sensory and unpleasantness dimensions of the subjective experience of pain, but only within the high hypnotizability group. Moreover, they did not find differences in response to hypnotic suggestions in early ERP components but rather in the later P2a and P2b components, for which it was found that manipulating the unpleasantness of the sensory stimuli modulated the components' amplitudes. In addition, hyperalgesia resulted in an increase of oscillatory gamma band activity (30–90 Hz), especially in response to the manipulations of unpleasantness which paralleled alterations in participants' subjective experience of pain. The authors concluded that hypnotic modulation of pain, especially of unpleasantness hyperalgesia, involves excitatory and inhibitory fronto-parietal selective attention processes that take place during monitoring and orienting attention toward noxious stimuli (i.e., a top-down modulatory effect).

EEG findings from other studies are inconsistent with these results. While some studies did not detect differences in brain responses to painful stimuli after hypnotic analgesia (Meier et al., 1993), others found various differences, such as decreased ERP amplitudes (De Pascalis et al., 2008;), and increased ERP amplitudes (Friederich et al., 2001; Sommer, 1966) or even both decreased and increased ERP amplitudes (De Pascalis et al., 2001).

De Pascalis et al. (2001) found that hypnotic analgesia administered to highly hypnotizable individuals resulted in increased amplitude in early ERP components at the temporal areas, and decreased amplitude at late ERP components in the frontal and temporal brain areas. However, in moderately hypnotizable individuals, a decreased early component was found at temporal brain areas. Nevertheless, in a follow-up study by the same group, a reduction in amplitude over frontal and central brain areas was also found in early components among highly hypnotizable individuals in response to hypnotic analgesia (De Pascalis et al., 2008).

To summarize these findings, a number of EEG studies report a decrease in early or late ERP components, mainly in frontal, temporal and parietal brain areas during hypnotic analgesia in high hypnotizable individuals, while other studies found an increase or no change. Brain imaging studies have identified the cingulate, primary and secondary somatosensory cortices, as well as the insula to be involved in changes in pain perception due to hypnotic analgesia, such that decreased activity in these areas corresponds to a decrease in perception of the noxious stimuli.

In the first attempt to uncover the brain mechanisms underlying hypnotic anaesthesia, Halliday and Mason (1964) used EEG to record the cortical evoked-related-potentials (ERPs) of nine participants before and during hypnotic anaesthesia. Five of the participants received electrical stimuli to the hand while four participants received mechanical taps (tactile stimuli) to the index finger. Despite participants' reports indicating that they felt no stimulus, for both types of stimuli no changes in ERP amplitudes were found. Similarly, a case study on a 23 year old male participant receiving photic stimulation under hypnotic anaesthesia found no differences in ERP amplitudes (Serafetinides, 1968). In a more recent EEG study, however, it was found that among 10 highly hypnotizable individuals hypnotic anaesthesia resulted in reduced ERP amplitudes throughout the scalp after 100 ms and at electrodes placed above the right frontal, parietal and occipital lobes, after 300 ms (Spiegel et al., 1989).

In light of the paucity of studies on the brain mechanisms underlying hypnotic anaesthesia, and the conflicting results yielded by previous studies on the brain mechanisms underlying hypnotic analgesia, the aim of the present study was to facilitate further understanding of how hypnosis affects brain processing of simple tactile stimuli, by focusing on hypnotic anaesthesia. The study did not focus on pain processing mechanisms but rather on changes in sensory processing mechanisms, administering simple, non-painful, tactile stimuli.

Due to the divergent nature of previous hypnotic analgesia studies and the scarcity of studies on hypnotic anaesthesia we did not formulate precise hypotheses regarding the involvement of specific brain areas in hypnotic anaesthesia, instead the study examined the more global question of whether the changes in sensory processing mechanisms occur at lower levels of sensory processing (sensory cortex), at higher levels of sensory processing (e.g., insula), at more cognitive or executive levels (e.g., prefrontal cortex) or some combination of the aforementioned.

To facilitate the aims of this research Magnetoencephalography (MEG) was used because it offers high temporal resolution together with good spatial resolution and because it enabled us to combine ERF analysis (similar to ERP analysis in EEG) and time-frequency analysis together with source localization. MEG may thus be able to offer new insights into the brain mechanisms underlying hypnotic anaesthesia. It is, however, important to note that source depth limits the sensitivity of MEG source localization analysis, so that it is mainly sensitive to tangential components (i.e., structures located mainly in the sulci) and less sensitive to radial components (i.e., structures located at the crests of the gyri; Hillebrand and Barnes, 2002).

As previous non-hypnosis somatosensory studies have shown, brain spatial-temporal responses to sensory stimuli are frequency dependent, such that different frequencies represent different facets of brain processing (e.g., Bauer et al., 2006; van Ede et al., 2011). It may be that failure to detect changes in brain response to hypnotic anaesthesia and hypnotic analgesia in some previous EEG studies was due to the fact that the root cause of the activity is in

the event-related oscillations and not in the ERPs, we sought to address this in the present study. MEG was also used because it enabled us to bridge between PET source localization findings and EEG findings.

Thus, the study sought to investigate the neurophysiological frequency dependent spatial-temporal changes in somatosensory processing caused by hypnotic anaesthesia. Our hypothesis was that hypnotic anaesthesia modifies spatial-temporal responses to neutral sensory stimuli in the frequency domain. Following on from previous studies that showed that hypnotic analgesia does not affect individuals displaying low levels of hypnotizability, it was decided to test the effect of hypnotic anaesthesia on neurotypical participants displaying moderate to high levels of hypnotizability.

2. Experiment 1

2.1. Method

2.1.1. Participants

Twenty six right-handed neurotypical participants were recruited for the experiment. Exclusion criteria included current or past neurological, physical and mental disorders (including dissociative symptoms), medication or substance abuse, and low hypnotizability assessed using the Hebrew version of the Stanford Hypnotic Susceptibility Scale form C (SHSS-C; Lichtenberg et al., 2009; Weitzenhoffer and Ernest, 1962). The SHSS-C score is the sum of 12 dichotomy tasks (1 point for each successful task), thus, the final score ranges between 0 and 12 with scores between 4 and 8 considered moderate, and 8 or above considered high. The Kuder-Richardson reliability correlation of the Israeli SHSS-C version is .79. It was decided to recruit not only individuals with high hypnotizability but also individuals with moderate hypnotizability for the experiment, in order to increase the study's external validity as, according to the norms of the Israeli version of the SHSS-C, ~80% of the population has medium to high levels of hypnotizability (average score is 5.63, SD=2.61).

Two participants were excluded from participation due to substance abuse, one due to mental disorder and one due to low hypnotizability. Thus, 22 individuals participated in the experiment. However, three participants were excluded from data analysis due to excessive artifacts (heart beat and muscle artifacts). As a result, a total of 19 individuals (eight females and 11 males with an average age of 34.68, SD=9.77) with moderate to high hypnotizability were included in the analysis. Of the remaining participants, five had medium levels of hypnotizability and 14 had high levels of hypnotizability (average score of 9.11, SD =1.1). Participants were recruited from the community and received monetary compensation.

The research was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans, and was approved by the Hadassah Hospital ethics committee. In addition, and according to the Israeli law on hypnosis, a special permission for hypnosis protocol, used in the research, was obtained from the Hypnotic Committee of the Israeli Ministry of Health. Written informed consent was obtained from all subjects.

2.1.2. Stimuli

Vibrotactile stimulation was delivered through plastic tubes connected to a somatosensory generator (Somatosensory Stimulus Generator, 4-D Neuroimaging, San Diego, CA). Pneumatically driven pulses (25 PSI) were applied to the inner tip of the right and left index fingers by a balloon diaphragm attached with a plastic spring clip.

2.1.3. Procedure

Prior to scanning, participants completed a demographic questionnaire followed by the Hebrew version of the Dissociative Experiences Scale (DES; Bernstein and Putnam, 1986) for the purpose of excluding participants with dissociative symptoms, which evidence suggests may be counter-indicative for hypnosis (West, 1965). Participants were then scanned in a supine position. Before the scan, head-shape was digitized using a Polhemus Fastrack digitizer. Scanning commenced with a digital registration of head position. Explanations were then given, emphasizing the need for participants to remain with closed eyes and to refrain from moving the head during the entire experiment. After participants confirmed that they understood the experimental procedure, a session consisting of 200 vibrotactile stimuli was commenced: 100 to the right index finger and 100 to the left index finger in a random order. The stimulus was a rectangular pulse (OFF-ON-OFF) with ON duration of 50 ms and averaged interstimulus interval of 800 ms with a random jitter ranging between −150 ms and +150 ms.

Following the first vibrotactile session, the hypnotic state was induced and deepened by a trained specialist (last author in this paper, EGA) sitting beside the participant in the MEG's shielded room. Hypnosis commenced with simple progressive muscle relaxation, this was deepened by the application of hypnotic pacing to the here-and-now experience of the participant. This was followed by the application of counting, in order to further deepen the hypnotic state. The entire hypnotic induction stage took 5.97 min on average (SD=1.11), with a range of 2.83–7.35 min. Once hypnotic induction was achieved (as adjudged by the hypnotherapist according to changes in participants' breathing pace and facial color), anaesthetic suggestion was initiated by having participants imagine their left hand lying in a bath of cold iced water. To further intensify the experience, suggestion of a total lack of feeling in the hand was applied. Furthermore, participants were asked not only to ignore their left hand (the attenuated hand), but at the same time to concentrate on sensations in their right hand (the non-attenuated hand). The suggestion stage took 2.52 min on average (SD=0.51), with a range of 1.54–3.39 min.

After anaesthetic suggestion was induced, the second session of 200 vibrotactile stimuli (100 to each index finger in a random order) began. At the end of the second vibrotactile session, hypnotic suggestion was used to neutralize the left hand anaesthesia. This was followed by the de-hypnotization and de-induction of all suggestions. A carry-over effect, whereby the effects of hypnosis continues after de-hypnotization, has been observed (e.g., Colby, 1991; Stam and Spanos, 1980). As a result, a counterbalance between conditions was not performed, nor was a third, post de-hypnotization, vibrotactile session incorporated within the experiment.

After completing the experiment, participants were briefed on the possible effects of the hypnosis and were asked to answer a short questionnaire regarding their subjective experience during the experiment. The questionnaire consisted of three open questions and two Likert questions. The open questions asked participants: (a) to describe their subjective experience during the experiment, (b) to recall the stages of the experiment in a chronological sequence and (c) to express their thoughts regarding the experiment.

The Likert questions included two questions aimed at validating the hypnotic induction stage and the suggestion stage. In the first question, participants were asked to quantify the extent to which they felt their attention was focused during the hypnotic induction stage on a scale from 1 to 7 (1 – "I could not focus my attention at all"; 7 – "My attention was extremely focused and I was not disturbed at any point"). The average result was 5.74 (SD=1.15) with 16 participants (out of 19) reporting a score

greater than 4. A one-sample *t*-test against the mid-point of the scale (MP =4) revealed a significant effect, t(18)=6.6, $p < .001$.

In the second Likert question participants were asked to quantify the extent to which they felt a difference between their right and left hands after hypnotic anaesthesia was applied on a scale from 1 to 7 (1 – "I did not experience any difference between the right and left hands"; 7 – "I experienced a significant and unexpected difference between the right and left hands, to the extent that I lost awareness of the left hand"). Average perceived difference between hands was 4.55 (SD=1.54) with 12 participants reporting a score greater than 4. One participant reported experiencing no differences between the two hands and one participant could not recall his experience. A one-sample *t*-test against the mid-point of the scale revealed a significant effect, t(17)=2.9, $p=.01$.

2.1.4. Data acquisition

MEG recordings were conducted using a whole-head, 248-channel magnetometer array (4-D Neuroimaging, Magnes 3600 WH) in a magnetically shielded room. Reference coils located a short distance (~30 cm) away from the 248 channels, and oriented by the x, y and z axes, were used to remove environmental noise. Data were digitized at a sample rate of 1017.23 Hz using a .1–400 Hz online band-pass filter.

2.1.5. Data analysis

2.1.5.1. Pre-processing. Heartbeat and powerline artifacts were removed using an event-synchronous cancellation algorithm (Tal and Abeles, 2013) using MATLAB (Mathworks, Natic, MA). The Fieldtrip toolbox for MATLAB (Oostenveld et al., 2011) was used for data preprocessing and analysis. Data were segmented into epochs, starting 150 ms before vibrotactile stimulus and ending at 500 ms after vibrotactile stimulus. Trials containing power jumps and/or muscle artifacts were then visually rejected. The segmented trials were then low-pass filtered with a cutoff of 40 Hz and the baseline was adjusted by subtracting the mean amplitude of the pre-stimulus period (150 ms) of each trial from all the data points in the segment. Spatial Independent Component Analysis was applied in order to clean eye movements and blinks.

2.1.5.2. Analysis. Time-frequency analysis was performed across conditions and subjects (grand average) in order to inspect the overall response to the stimuli along the time-frequency domains. Tapers were applied to each time window and time-frequency representations of power were calculated using a Fast Fourier Transform applied to a short sliding time window. This process was conducted twice: once to produce the image of the induced (non-phase-locked to stimuli onset) and evoked (phase-locked to stimuli onset) activity for each trial, and once to produce the image of the evoked activity only, for the average across all trials. We used a Hanning taper and applied a fixed time window of 250 ms with zero-padding on each side, resulting in a spectral resolution of 1 Hz ranging from 4 Hz to 40 Hz.

In order to estimate the neural sources of the components detected in the time-frequency images and to statistically compare between conditions, Synthetic Aperture Magnetometry (SAM) beamforming analysis was used (Robinson, 1999). Beamformer was used to construct a spatial filter for each point in a predefined three-dimensional grid, with 5 mm spacing between adjacent grid points, filling the volume of the brain. The head model was constructed using multiple spheres in a single conductance volume. The weights of the spatial filters were computed separately for each frequency band (alpha, beta and low-gamma) using a covariance matrix calculated on the entire segment of the cleaned, unaveraged data from all trials (for the evoked component, a frequency band of 1–40 Hz was used), in order to avoid a situation in which differences between conditions may result from different weights. MEG data were then projected through these spatial filters to give an estimation of neural activity at the target grid points for each time point. In order to avoid *center-of-head-bias* due to noise (a common problem with beamformer source localization), neural activity estimation was normalized by dividing the power of activity in each voxel by the square of the weight norm (Sekihara et al., 2004).

Based on the time-frequency images (see, Fig. 1), a 200 ms sliding time window was chosen for the source localization analysis. 200 ms was chosen because it maintains a balance between being sufficiently long enough for finding results in the alpha band (which peaked at 12 Hz for the attenuated hand under hypnosis, as can be seen in Fig. 1-D) and sufficiently short to filter out the evoked response from the lower frequencies. Thus, for each voxel in the cortex, power was calculated over a 200 ms time window with zero-padding on each side, that slid in 10 ms steps across the time course of activation of each trial for the induced component, and of the average of all trials for the evoked component (total of 47 time windows starting at 150 ms pre stimulus and ending at 500 ms post stimulus). Then two paired *t*-tests were conducted (one for each hand) between the before and under hypnosis conditions at each time window, using the AFNI 3dttest++ function (Cox, 1996) with a threshold *t*-value of ± 3. No interaction between Hand and Hypnosis was sought at the source level due to expected differences in Hand, resulting from the asymmetric nature of the stimuli.

In order to account for multiple comparisons, a permutation test was conducted (Nichols and Holmes, 2002). For each time window, a paired *t*-test was repeated 400 times (200 for each hand). In each permutation, condition (before vs. under) was randomly assigned for each subject. The largest cluster of adjacent voxels with $|t| > 3$ was recorded from each permutation, generating a probability density distribution of the maximal significant clusters. Only significant clusters (with $|t| > 3$) in the real data that exceeded the 95th percentile of the maximal cluster size distribution were considered significant.

Coregistration of the functional MEG data to the individual subject's MRI was performed using three anatomical landmarks and was manually corrected using headshape digitization. Because MRI scans for five participants were not available, a template MRI (Collin27) was modified to fit each of these subject's digitized headshapes using SPM8 (Wellcome Department of Imaging Neuroscience, University College London, www.fil.ion.ucl.ac.uk). The modified template was then used in order to convert the beamforming functional images to Talairach space.

2.2. Results

Time-locked brain responses to stimuli consisted of two main components (Fig. 1): an early evoked component, manifested as a relative increase at low frequencies, and an induced component, manifested as desynchronization peaking in the beta band (13–25 Hz) and extending into the alpha band (8–12 Hz) and low-gamma band (26–40 Hz).

At the source level, a before-under hypnosis comparison for the attenuated hand yielded two significant clusters: one in the beta band (13–25 Hz) and the other in the alpha band (8–12 Hz). The beta band cluster, indicating more brain activity before hypnosis than under hypnosis, was found around the time window of 214–413 ms post stimuli. The cluster was comprised of 80 voxels located in the right hemisphere, mainly in the insula (for full details, see Table 1 in appendix A, and Fig. 2). The alpha band cluster, indicating more brain activity before hypnosis than under hypnosis, was significant at three different time windows: 253–453 ms, 263–463 ms and 301–500 ms post stimuli. At its peak

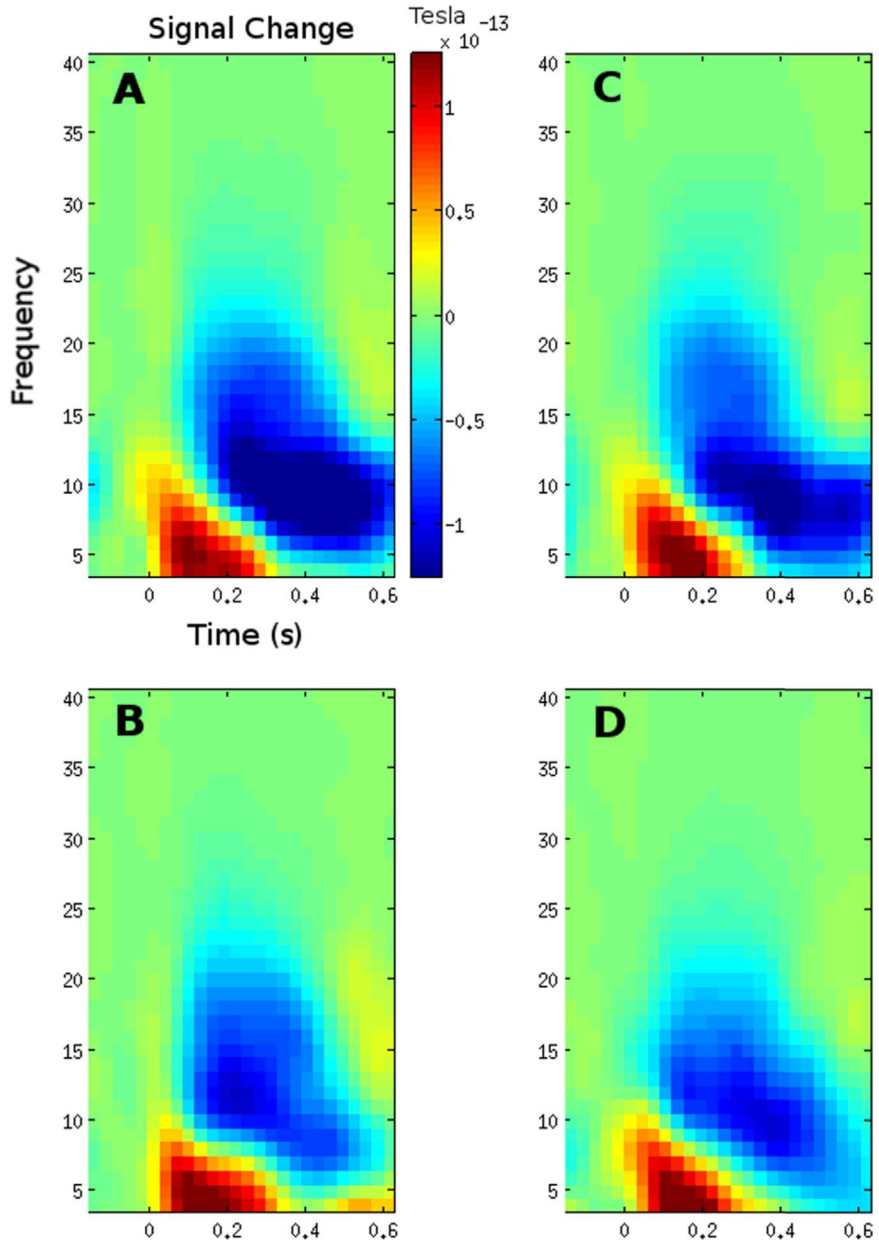

Fig. 1. Grand average time-frequency images across sensors. Figs. A–D show the total time-locked brain response (induced and evoked) to stimuli for each condition, colors represent signal change relative to baseline, where cold colors indicate activity suppression and warm colors indicate activity enhancement. A – attenuated hand before hypnosis; B – attenuated hand under hypnosis; C – non-attenuated hand before hypnosis; D – non-attenuated hand under hypnosis. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

(301–500 ms), the cluster included 183 voxels located in the left hemisphere, predominantly in the inferior frontal gyrus (for full details, see Table 1 in appendix A, and Fig. 3).

No significant clusters of differential activity were found for the gamma band or for the evoked response. No significant differences between states were found for stimuli applied to the non-attenuated hand.

In order to rule out habituation as an alternative explanation for the results for the attenuated hand, the before hypnosis trials for that hand were divided into two blocks (beginning and end of the before hypnosis condition). Two separate *t*-tests were then conducted between the two blocks: for the alpha band at the time window of 214–413 ms post stimuli and for the beta band at the time window of 301–500 ms. Results were then masked with the significant voxels from the previous analysis (see Figs. 2 and 3). No significant clusters were found for either the alpha band or the beta band.

2.3. Discussion

The study sought to identify the neuropsychological spatial-temporal mechanisms underlying somatosensory alterations caused by hypnotic anaesthesia. Using MEG, brain activity was recorded during the direction of sensory stimuli to the right and left index fingers in two separate sessions, one prior to hypnosis and one after anaesthetic suggestion had been hypnotically induced in the left hand (attenuated hand).

Differences were found for the attenuated hand only in both the alpha band and the beta band, indicating greater brain activity before hypnosis than under hypnotic anaesthesia. In the alpha band, the significant cluster was found around 253–500 ms, and activity predominated in the left inferior frontal gyrus. In the beta

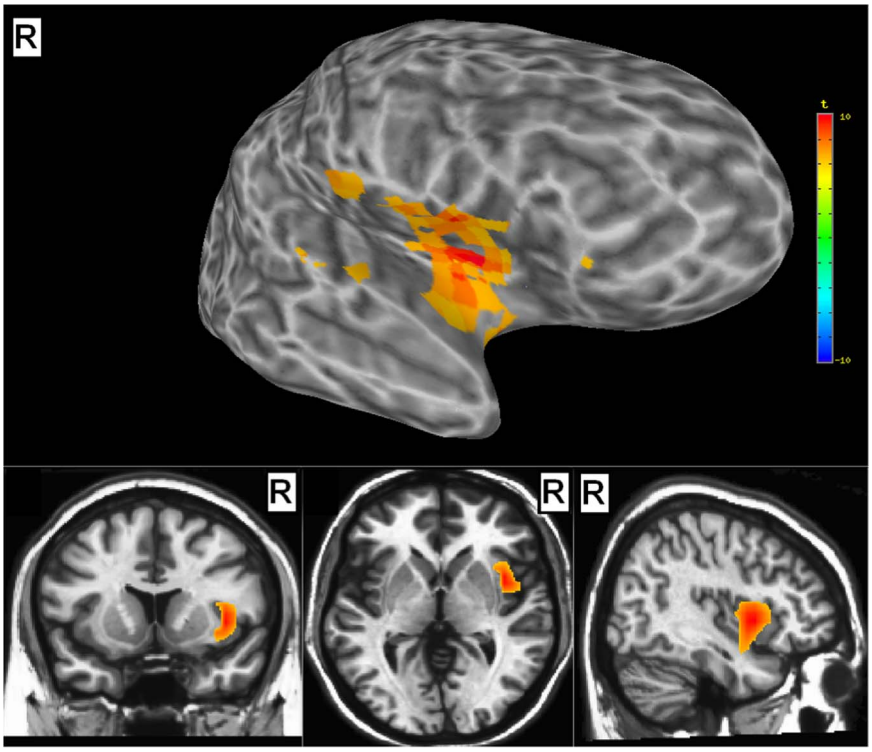

Fig. 2. Significant cluster for the before and under hypnosis comparison in the beta band, for the attenuated hand, in the time window of 214–413 ms. Colors represent significant *t* values. In transverse and coronal sections, left side image corresponds to left hemisphere. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

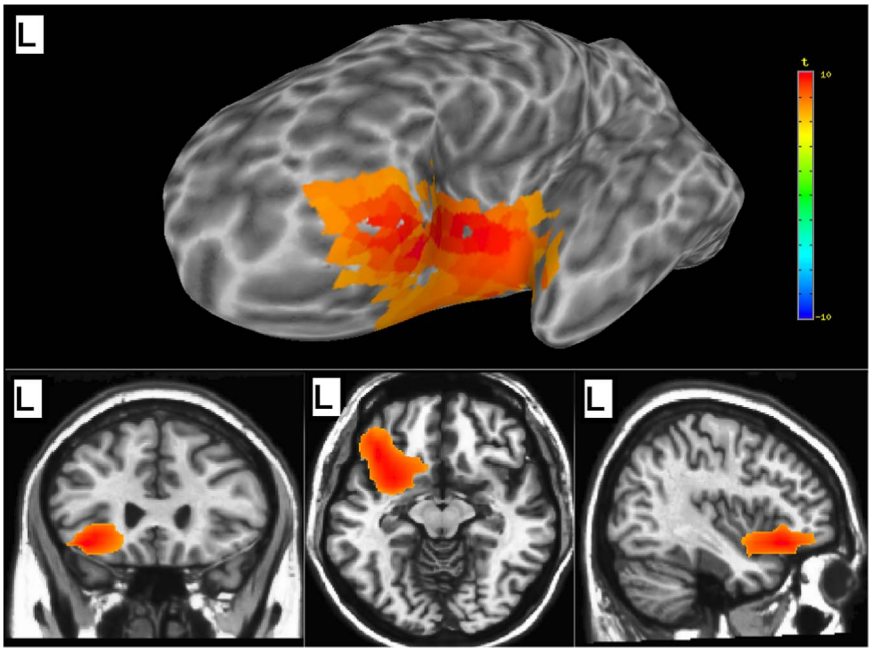

Fig. 3. Significant cluster for the before and under hypnosis comparison in the alpha band, for the attenuated hand, in the time window of 301–500 ms. Colors represent significant *t* values. In transverse and coronal sections, right side image corresponds to right hemisphere. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

band, the significant cluster was found at 214–413 ms, and activity predominated in the right insula.

Although these results support our hypothesis of altered somatosensory brain processing in response to hypnotic anaesthesia, it is possible that attention modulations *per se* are the underlying cause of the differences found. In other words, it is conceivable that hypnotic anaesthesia simply shifted participants' attention from the left hand to the right hand (Vuilleumier, 2005). In order to account for this alternative explanation we conducted a second experiment. This time, in the second session of vibrotactile stimuli, instead of hypnotically inducing reduced sensation to the left hand, we manipulated participants' attention, shifting it from the left hand to the right hand by asking them to count the number of stimuli received to the right index finger and ignore those to the

left. Our main hypothesis was that the pattern of results found in the attention manipulation experiment would differ from those found in the hypnotic anaesthesia experiment.

3. Experiment 2

3.1. Method

3.1.1. Participants

Sixteen right handed participants with moderate to high hypnotizability as assessed using the Israeli norms for the Stanford Hypnotic Susceptibility Scale (Lichtenberg et al., 2009; Weitzenhoffer and Ernest, 1962) were recruited for the second experiment. Two participants were excluded due to technical problems with recording the stimuli timings. Thus, six females and eight males with an average age of 28.86 (SD=5.17) were included in the experiment. Participants were pre-screened for neurological, physical and mental disorders (including dissociative symptoms). Participants were recruited from the community.

3.1.2. Stimuli and procedure

The procedure for applying vibrotactile stimulation was the same as in the first experiment, however, in the second session, rather than apply hypnotic induction and anaesthetic suggestion, participants were instructed to ignore their left hand and to count the number of stimuli administered to their right hand (Counting condition). In order to validate the manipulation, once the second vibrotactile session ended, participants were asked how many stimuli they received to each hand. The average of participants' reports of the number of stimuli directed to the right hand was 85.79 (SD=10.43), and only 24.43 (SD=12) to the left hand. A paired sample *t*-test revealed that this discrepancy is significant, $t(13)=1.8$, $p<.001$, validating the experimental manipulation.

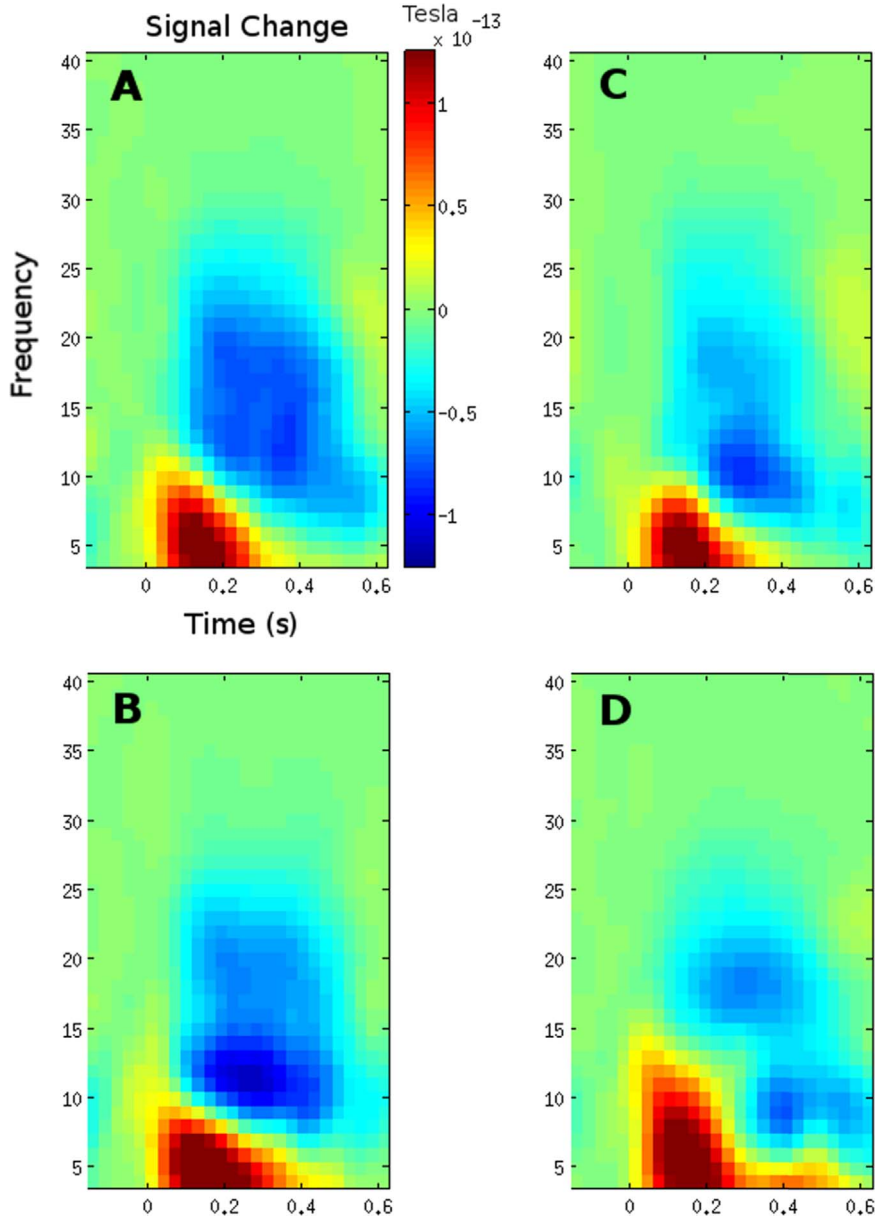

Fig. 4. Grand average time-frequency images across sensors. Figs. A–D show the total time-locked brain response (induced and evoked) to stimuli for each condition, colors represent signal change relative to baseline, where cold colors indicate activity suppression and warm colors indicate activity enhancement. A – attenuated hand before counting; B – attenuated hand during counting; C – non-attenuated hand before counting; D – non-attenuated hand during counting. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Contrary to the first experiment, participants were not required to complete either the Hebrew version of the Dissociative Experiences Scale (DES; Bernstein and Putnam, 1986) or the questionnaire regarding their subjective experience during the experiment.

3.1.3. Data acquisition and analysis

Data acquisition and analysis proceeded in the same manner as in the first experiment. As in the first experiment, time-frequency plots showed two separate components; an evoked component, and an induced component that peaked in the beta band and extended into the alpha and low-gamma bands (see Fig. 4).

3.2. Results

For both the attenuated and non-attenuated hands, a before-during counting comparison yielded significant results for the induced response. For the attenuated hand, two significant clusters were found in the beta band, indicating more brain activity occurring before counting than during counting. The first cluster was found in seven consecutive time windows, ranging from 7 ms to 266 ms post stimuli, and was located in the left hemisphere. At its peak (37–237 ms), the cluster included 337 voxels, the majority of which were located in the middle and superior temporal gyrus, the parietal lobule, and the insula (for full details, see Table 2 in appendix A). The second cluster was found in the time window from 115 to 315 ms post stimuli, and incorporated areas in both hemispheres. In the left hemisphere, most activity was found in the cuneus and the lingual gyrus, whereas activity in the right hemisphere was concentrated mostly in the parietal lobule, the temporal gyrus and the supramarginal gyrus (for full details, see Table 2 in appendix A).

For the non-attenuated hand, a significant cluster, indicating greater brain activity before counting than during counting, was found in the low-gamma band. It commenced at 135 ms and ended at 443 ms (with a peak between 175–357 ms). The cluster was comprised of 760 voxels, mainly in the left hemisphere, and included the right and left cingulate gyrus, the right and left insula, left temporal gyrus, left parietal lobule and the left pre- and post-central gyri (for full details, see Table 2 in appendix A).

No significant clusters of differential activity were found for the alpha band or for the evoked response.

3.3. Discussion

In order to rule out simple attention modulation as an alternative explanation for hypnotic anaesthesia, Experiment 2 was conducted. However, in the second vibrotactile stimulation session in Experiment 2, rather than using hypnotic anaesthesia to induce reduced sensation, participants were instructed to count the number of stimuli administered to their right hand (non-attenuated hand) while ignoring their left hand (attenuated hand). Differences were found both for the attenuated and non-attenuated hands. In both cases, more activity was found before counting than during counting.

In the beta band, similar to the results in the previous experiment, two significant clusters were found for the attenuated hand. Nevertheless, differences appeared earlier (7–266 ms; 115–315 ms) than in the hypnosis experiment (214–413 ms). More importantly, the reductions in activity were located in different brain areas. Whereas in the hypnosis experiment decreased activity was primarily located in the right insula, in the attention modulation experiment activity decreases occurred in the left middle and superior temporal gyrus, insula and inferior parietal lobule, and in the left cuneus, left lingual gyrus, right parietal lobule and right temporal and supramarginal gyri. Additionally, and in contrast with the hypnosis experiment, a significant cluster was found for the non-attenuated hand in the low-gamma band. This is similar to previous findings of a reduction of activity in the low gamma band in response to hyperalgesia, which, according to the authors' conclusion, represents the process of orienting attention toward a stimuli (Valentini et al., 2013).

The different pattern of results between the two experiments rules out the possibility that the anaesthetic suggestion applied during hypnosis merely shifted participants' attention from the left hand to the right hand. This is not to claim that attention modulation processes do not take place in hypnotic anaesthesia; on the contrary, we assume that some sort of attention modulation must be involved in hypnotic anaesthesia. Nevertheless, if attention modulation alone was sufficient to explain the results of the hypnosis experiment, then the pattern of results found in both experiments would have shown greater overlap.

4. General discussion

Induced activity, in response to vibrotactile stimuli, was found in both experiments, across conditions, peaking at the beta band and extending into both the alpha and low-gamma bands. Activity started shortly after the stimulus was administered (~100 ms) and faded out at around 500 ms post stimulus administration. In addition, evoked activity was found in low frequencies (delta and theta bands), commencing immediately after the stimulus (~0 ms) and continuing for about 300 ms. These general patterns of induced and evoked activity in response to vibrotactile stimuli (see Fig. 1) are in alignment with findings from previous somatosensory MEG studies employing vibrotactile stimuli (e.g., Bauer et al., 2006; Nierhaus et al., 2015).

Further, in the hypnotic anaesthesia experiment significant differences were found between conditions for the attenuated hand (left hand), such that at the peak of activity (the beta band), less brain activity was found in the right insula during the administration of anaesthetic suggestion than prior to it. This result aligns with Hofbauer et al. (2001) finding that the contralateral insula is involved in pain modulation during hypnotic analgesia.

Situated at the intersection of the temporal, parietal and frontal lobes, the insula is believed to serve as an integrative center for own-body representation and awareness, and thus to be highly responsive to somatosensory stimuli (e.g., Bottini et al., 1995; Olausson et al., 2008; Ritter et al., 2009; Simmons et al., 2013; Straube and Miltner, 2011; Tsakiris et al., 2007; for a review see, Blanke, 2012; Craig, 2011). Indeed, in a pioneering extracellular recording study in rhesus monkeys aimed at differentiating the response properties of neurons in the insula to somatic, auditory, visual and gustatory stimuli, it was found that a major portion of the neurons in the insula are dedicated exclusively to somatic processing (Schneider et al., 1993).

The insula receives large quantities of information from ipsilateral somatosensory areas and projects information into the amygdala and the hippocampus. It thus serves as a corticolimbic pathway through which tactual recognition emerges contralaterally (e.g., Craig, 2011; Schneider et al., 1993). As such, it is unsurprising that the functioning of the insula was found to be correlated with attention to stimuli, such that greater activity in the insula represents greater attention to the stimuli (Dosenbach et al., 2007; Duncan and Owen, 2000; Menon and Uddin, 2010; Nelson et al., 2004; Seeley et al., 2007; Sterzer and Kleinschmidt, 2010).

In light of the above, our finding of a reduction in beta band brain activity in the right insula during hypnotic anaesthesia directed to the left hand would appear to represent a reduction in awareness of the left hand, and of the tactual stimuli administered

to it. As the different pattern of results found in the attention modulation experiment indicates, this effect is not merely due to participants' attention shifting from the left hand to the right hand. This experiment was designed retrospectively, to rule out the alternative explanation of attention shifting from the left hand (attenuated hand) to the right hand (non-attenuated hand) as the cause of the differences found. It thus arose from a limitation in the initial experimental design, as it would have been better to include an attentional control condition in the hypnosis experiment to begin with.

The interpretation of the beta band results is indirectly supported by alpha band results indicating greater activity in the alpha band in the left inferior frontal lobe before hypnosis than under anaesthetic suggestion. Alpha oscillations are known to be related to selective attention mechanisms, such that alpha levels are typically higher after the presentation of attenuated stimuli compared with non-attenuated ones (for a review see Foxe and Snyder (2011)). In other words, the relative absence of activity in the alpha band is suggestive of greater cognitive engagement. Thus, our finding of lower levels of activity in the alpha band in the left inferior frontal gyrus under anaesthetic suggestion implies that this area was more engaged in response to tactile stimuli when under hypnotic anaesthesia than prior to it.

There is a growing body of evidence indicating that the left inferior frontal gyrus plays a crucial role in top-down inhibitory control. For example, in working memory (Thompson-Schill et al., 2002), in semantic processing (Thompson-Schill et al., 1998), in resolving conflicts that arise from incompatible representations (Novick et al., 2005), and in motor inhibition (Swick et al., 2008). Our findings further support this, suggesting that the left inferior frontal gyrus is also involved in top-down somatosensory inhibition in hypnotic anaesthesia; functioning as a top-down inhibitory mechanism suppressing irrelevant sensory input.

Taken together, these results may suggest that the brain mechanism underlying hypnotic anaesthesia is decreased involvement of the insula (contralateral to the affected area of the body) coupled with increased involvement of the left inferior frontal gyrus, where the left inferior frontal gyrus is engaged in top-down somatosensory inhibition and the insula in somatosensory awareness. These two complementary mechanisms may work together to induce mental states in which individuals lose bodily sensation, for example, as seen in the first experiment, to the left hand. However, due to the limitations of the analysis (lack of connectivity analysis), this conclusion should be taken with caution and further research should address this directly.

During the anaesthetic suggestion procedure, participants were asked to visualize their left hand (attenuated hand) lying in a bath of cold iced water. Previous studies have shown that viewing one's body can produce an analgesic effect (e.g., Longo et al., 2009, 2012). Thus, it is possible that participants visualizing their attenuated hand during the hypnosis experiment played a mediating role in reducing somatosensory awareness.

The study has a number of significant limitations which should be the focus of future studies. Firstly, and as mentioned in the introduction, the MEG is mainly sensitive to tangential sources of activity and less sensitive to radial sources of activity. Thus, it is possible that other brain areas are also involved in changes in sensory processing in response to hypnotic anaesthesia, but because of the source localization limitations of MEG we were unable to detect them. In the future it would be valuable to run an experiment simultaneously combining MEG with EEG in order to give greater focus to radial sources of activity as well. Secondly, in order to rule out attention as an alternative explanation, future studies should include an attentional control condition to allow direct statistical comparison between the hypnotic conditions and the attentional conditions. Thirdly, the absence of a low hypnotizability participant group limits the scope of generalization of our findings and their validity. In future studies a low-hypnotizability group should be included as a control group. It is to be hypothesized that this group would be less susceptible to hypnotic anaesthesia, and would therefore report less of a perceived difference in feeling between the two hands and show a reduced brain effect for hypnotic anaesthesia.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.neuropsychologia.2016.05.020.

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