Focused analgesia in waking and hypnosis: Effects on pain, memory, and somatosensory event-related potentials

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Abstract

Somatosensory event-related potentials (SERPs) to painful electric standard stimuli under an odd-ball paradigm were analyzed in 12 high hypnotizable (HH), 12 medium hypnotizable (MH), and 12 low hypnotizable (LH) subjects during waking, hypnosis, and a cued eyes-open posthypnotic condition. In each of these conditions subjects were suggested to produce an obstructive imagery of stimulus perception as a treatment for pain reduction. A No-Analgesia treatment served as a control in waking and hypnosis conditions. The subjects were required to count the number of delivered target stimuli. HH subjects experienced significant pain and distress reductions during posthypnotic analgesia as compared to hypnotic analgesia and between these two analgesic conditions as compared to the two control conditions. Outside of hypnosis, these subjects remembered less pain and distress levels than they reported during hypnotic and posthypnotic analgesia treatments. In contrast, for waking-analgesia treatment, HH subjects remembered similar pain and distress levels to those they reported concurrently with the stimulation. HH subjects, during hypnotic and posthypnotic analgesia treatments, detected a smaller number of target stimuli and displayed a significant amplitude reduction of the midline frontal and central N140 and P200 SERP components. No significant SERP differences were observed for these subjects between treatments in waking condition and between hypnotic and posthypnotic analgesic treatments. For the MH and LH subjects no significant N140 and P200 amplitude changes were observed among analgesic conditions as compared to control conditions. These amplitude findings are seen as indicating that hypnotic analgesia can affect earlier and later stages of stimulus processing.

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1. Introduction

Previous findings have shown that obstructive hallucinations of noxious stimulation in hypnosis reduce pain sensation (for reviews see, [8,30]) and the amplitude of a later P300 component of the SERPs [14,15,48], indicating that the locus of hypnotic influence is not in the initial sensory experience itself, but rather in the cognitive-emotional component of the information processing. However, more recent EEG findings have also

evidenced that focused analgesia, in hypnosis, may reduce a stimulus-locked 40 Hz-EEG synchronization response [12] that is believed to reflect perceptual aspects of stimulation [1]. Therefore, the main aim of the present study was to further evaluate the modulatory effect of hypnotic analgesia on both the earlier N140 and later P300 component of the SERPs, the former believed to be more stimulus orientated and the latter expression of the ongoing cognitive-emotional processing (e.g., [26]). The study was devoted to address a very important and controversial question concerning the impact of hypnosis on the response to suggestions (see [38]). This was carried out by comparing the effects of an analgesia suggestion, administered during a non-hypnotic waking

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condition, with those obtained delivering the same suggestion just after the induction of hypnosis and, later, after a cued posthypnotic condition that is believed to produce a deeper hypnosis [2].

A typical characteristic of the deeply hypnotized individual is the appearance, spontaneously upon emerging from hypnosis, of an apparent amnesia for that which had occurred while in the hypnotic state (posthypnotic amnesia). There seems to be a general agreement that posthypnotic amnesia is not the same as simple forgetting [27,44], and that it is a product of hypnotic suggestion, direct or implicit [32–34,37,52], and also a product of dissociation [21,60]. No reports are known to us evaluating the effect of spontaneous posthypnotic distortion of pain memory. Thus, a further purpose of the present study was to evaluate, upon emergence from hypnosis, the degree in which retrospective pain and distress ratings are spontaneously distorted, and whether these distortions are modulated by individual differences in hypnotizability. The rationale for this evaluation is based on results of previous studies showing that memory distortion of pretreatment pain contributes to an exaggeration of self reports of pain relief [13,22,29,39,40].

Further aim of the present investigation was to evaluate whether: (1) in HH individuals, the analgesic suggestions are more effective following a formal hypnotic induction (hypnotic and posthypnotic conditions) than in the waking condition; (2) pain reduction during hypnotic and posthypnotic analgesia conditions is accompanied by an attenuation of the N140 and P200 peaks of the SERPs.

2. Methods

2.1. Subjects

Thirty-six right-handed undergraduate students (18 women and 18 men; age range 19–28 yr) were selected for high ($N = 12$; 6 women and 6 men), medium ($N = 12$; 6 women and 6 men), and low ($N = 12$; 6 women and 6 men) levels of hypnotic susceptibility. The subjects were tested using the Italian translation by [57] of the Stanford Hypnotic Susceptibility Scale, Form C (SHSS:C; [16,59]). They were categorized as being HH subjects ($N = 12$, $M = 10.5$, SD = 0.7) or LH ones ($N = 12$, $M = 2.9$, SD = 1.5) when their scores on SHSS:C were, respectively, 1 SD above or below the group mean of a larger group of subjects ($N = 105$, $M = 6.8$, SD = 3.9; 65 women and 40 men). The moderately hypnotizable group was formed with subjects who showed hypnotizability scores 1 SD within the group mean ($N = 12$, $M = 6.3$, SD = 0.8). Two female hypnotists carried out the assessment of hypnotic susceptibility about 20 days prior to the EEG recording session. In this session, hypnosis was induced again by one of the two hypnotists who did not know the hypnotizability level of the subject. Subjects were admitted to participate in the experiment only if they reported an absence of medication use or medical conditions that might interfere with pain sensitivity (e.g., diabetes mellitus, high blood pressure, heart diseases, asthma, post trauma to hands, frostbite, arthritis, Raynaud's syndrome). Subjects were not informed of their hypnotic ability and of the relevance of hypnotic ability in the experiment. Women who were in a menstrual period were invited for EEG recordings in another occasion.

2.2. Procedure

The selected subjects were individually invited in the EEG lab and upon arrival they were informed about the nature of the painful electric stimulation. In accordance with the ethical norms of the Italian Association of Psychology (AIP), a written consent was obtained if they agreed to participate in the experiment. In this session, hypnosis was induced for the second time using an Italian translation of the original American protocol of the Stanford Hypnotic Clinical Scale (SHSC; [42]). The subjects were not informed about their hypnotizability level during the EEG recording session and were all naïve volunteers.

2.3. Pain treatments

During waking and hypnosis conditions, the following five pain treatments were administered to each subject: (1) Waking-Pain (No-Treatment: W-Pain). Subject was required to detect target painful stimuli (eyes-open) without giving suggestions to reduce pain. (2) Waking-Analgesia (W-Analgesia). Suggestion to produce an obstructive imagery of stimulus perception by imagining a glove that was covering the finger and the hand and focusing on sensation in the finger and hand and experiencing that all sensations of the stimulated finger will be attenuated (eyes-open). (3) Hypnosis-Pain (No-Treatment: Hy-Pain). At the end of hypnotic induction and hypnotic testing, painful stimuli were delivered without suggestions to reduce pain (eyes-closed). (4) Hypnosis-Analgesia (Hy-Analgesia). In hypnosis condition was given an analgesia suggestion as in (2) (eyes-closed). (5) Post Hypnosis-Analgesia (P.Hy-Analgesia). Just before getting out from hypnosis, the subject was suggested that he/she will tend to sink deeper and deeper into involvement in the hypnotic state with open eyes, after that the experimenter will have knocked two times on the wall of the sound proof box ('fractionation technique', see Barabasz and Watkins, 2005; p. 193). When the subject was just out from hypnosis, the experimenter knocked two times and suggested to the subject that he/she was going into a deeper hypnosis state and the above-reported analgesia suggestion was administered. At the end of P.Hy-Analgesia treatment, the subject was waked up from hypnosis. Both waking and hypnosis conditions were counterbalanced across subjects in order to avoid possible order effects or habituation. Within each waking or hypnosis condition, the order of the treatments was not varied to prevent for a proactive effect of suggestion. Between waking and hypnosis conditions, a resting period of 12 min was given. In each condition, the subject was asked to count the number of delivered target stimuli.

Each treatment condition lasted about 5 min. Painful stimuli were applied to the subjects middle finger of the right hand and, at the end of each condition, they were asked to rate any

pain and distress experienced for standard stimuli on two separate 10 point numeric rating scales (NRS; [31]). On the left and right sides of the NRS-sensory scale, there were, respectively, the descriptors '0 = no pain sensation' and '10 = the most intense pain sensation imaginable'. Similarly, for the NRS-distress scale, the descriptors ranged from '0 = not at all distressful' to '10 = the most distress imaginable'. An involuntariness measure of pain reduction effect was obtained at the end of Post-Hypnosis-Analgesia condition by requiring participants to rate on the NRS how much pain reduction they experienced as occurring involuntarily (from '0 = quite voluntarily' to '10 = absolutely involuntarily').

Approximately 5 min after the waking or hypnosis session ended, the participants used two 10-point NRS scales to rate remembered pain and distress sensations they experienced for each experimental condition.

2.4. Sensory and pain thresholds

Somatosensory stimuli were delivered by applying two silver–silver chloride cup electrodes to the palmar surfaces of the distal and medial phalanges of the middle finger of the right hand. The electrode cup (1 cm in diameter) was filled with an electro-conductive hypoallergic cream and impedance was kept below 30 kΩ. Stimuli were unipolar electrical pulses (2-ms duration) generated by a constant current stimulator (Digimiter, Mod DS7A).

Sensory and pain thresholds were determined for each participant just before the EEG recording. Participants received first a series of single, unipolar pulses separated by 10 s intervals, starting with an intensity of .05 mA and increasing with steps of .05 mA until the participant reported that the minimum detectable stimulus was reached. The intensity level of the just noticeable pin-prick was taken as the sensory threshold associated with ascending intensity levels. A similar, but reversed procedure, was used to obtain the sensory threshold associated with descending intensity levels, starting from .5 mA and decreasing with steps of .05 mA. The two thresholds were then averaged to obtain the participant's sensory threshold. After the sensory threshold, pain tolerability threshold was determined by delivering stimuli of increasing intensity (steps of 0.5 mA) until a very painful pin-prick was reported. After this level, stimulus intensity was then increased until the subject reported the delivered stimulus as unbearable. This value was defined as the greatest level of pain which a subject is prepared to tolerate. Stimulus intensity used during experimental conditions was 0.5 mA under this tolerability level.

2.5. Electric stimulation

In each experimental condition subjects were engaged in an oddball task consisting of 70 electrical stimuli wherein infrequent targets (14.5%) were interspersed among frequently occurring standard stimuli (85.5%).

Target stimuli were presented in a pseudo-randomized order so that at least 2 standard stimuli preceded a target one. The inter-stimulus interval was set at a constant time of 3 s. Each standard stimulus consisted of one unipolar pulse with a duration of 2 ms. Target stimulus was formed by pairing two standard stimuli with an inter-pulse interval of 25 ms.

2.6. EEG acquisition and processing

The EEG was recorded by using pure tin electrodes mounted on an Electro-cap [5] placed on frontal (F3, F4), parietal (P3, P4) and midline (Fz, Cz, Pz) scalp sites. Linked earlobes served as reference with a forehead ground. Electrode impedance was kept below 3 kΩ and raw EEG signals were recorded using nine amplifiers (amplifier gains set at 10,000 with a band pass of 0.5–75 Hz). Eye movement (EOG) was recorded in a bipolar arrangement, superior orbit referenced to the outer canthus of the left eye. Trials on which the EEG or the EOG exceeded ±100 μV were rejected automatically. The EEG was acquired in digital form, using an IBM-compatible computer, by sampling at 1024 Hz per channel with a 12-bit resolution (Metrabyte Dash-16). For each instruction condition, 70 epochs (60 for standard and 10 for target stimuli) were digitized and stored on hard disk, using a time period of 1000 ms. For each recording epoch, a 100 ms period before stimulus onset was taken as baseline. EEG sweeps with artifacts due to scalp muscle or stimulus contamination, head or electrode movement, slow potential variations, false positive button pressing on standard non-target stimuli were a posteriori eliminated. In this study, only EEG sweeps corresponding to standard stimuli were off-line analyzed. This was done since there is experimental evidence that standard stimuli are more probable to elicit a 'pain-specific' SERP response (a potential which is usually considered of the same family, but with a shorter latency, of the classic P300 wave) that is more dependent from nociceptive component of the stimulus and less influenced by the ongoing cognitive information processing not intrinsic to pain [3,4]. Each EEG epoch was low-pass filtered at 15 Hz (FIR filter 3 dB, 12 dB/octave roll-off) and then averaged.

The most stable and reliable SERP peaks were a negative component peaking at about 140 ms (N140: 142.8 ± 11.2 ms) and a positive component peaking at about 200 ms (P200: 198 ± 10.6 ms). A peak amplitude measure of the N140 component was obtained as the minimum peak amplitude detected within a 70–200 ms time interval. An amplitude measure of the P200 component was obtained as the maximum peak amplitude within a 120–250 ms time window. These peak values were obtained by using algorithms of the ASYST-Keithley programming system for the calculus of local maxima and minima. Time windows used for the measure of N140 and P200 peaks were chosen after visual inspection of the SERPs.

2.7. Statistical analyses

A simple split-plot ANOVA with Hypnotizability, serving as between group variables, was used to assess individual differences on subjective threshold measures.

A repeated measure split-plot ANOVA, comprising Hypnotizability (3) and Treatment (5), respectively, as between and within factors, was performed to assess the effect of hypnotizability and treatments on the number of omissions of target stimuli.

To test the effect of experimental treatments on retrospective pain and distress ratings, a repeated measure split-plot ANOVA was carried out, comprising Hypnotizability (3), Recollection (2) and Treatment (5), respectively, as between and

within factors. The Recollection factor included both contingent pain ratings and remembered pain ratings as a within factor with nested Treatment as a repeated measure factor. A similar ANOVA was used to assess the distress and remembered distress ratings.

To test the impact of hypnotic depth on the response to analgesia suggestion change scores of both pain and distress were calculated for each waking and hypnosis condition by subtracting concurrent ratings during the suggestion of analgesia from those obtained in the correspondent control condition. Change scores were analyzed using separate ANOVAs with Hypnotizability (3) serving as between-subjects factor and Condition (3; Waking, Hypnosis, Post-hypnosis) as within-subjects factor.

For each N140 and P200 peak amplitude measure, two separate repeated measures ANOVAs were carried out. One ANOVA was performed for quadrant recording sites (F3, F4, and P3, P4) to assess hemispheric and anterior/posterior effects [46] using Hypnotizability (3) as between-subjects factors, and Treatment (5), Recording Site (2; frontal, parietal), and Hemisphere (2) as within-subjects factors. A separate ANOVA was carried out to assess N140 and P200 changes for midline recording sites (Fz, Cz, and Pz).

Huynh-Feldt epsilon correction of significance levels was used when necessary [58]. Post hoc comparisons were carried out by using a t-test procedure with Bonferroni correction of $\alpha = .05$ [36]. SAS-8.02 was used for all statistical analyses.

3. Results

3.1. Threshold measures, involuntariness ratings, omitted targets, and remembered pain intensity and distress ratings

The ANOVA on sensory threshold, pain, and distress thresholds failed to yield any significant effect (all Fs were not significant).

The ANOVA on involuntariness ratings yielded main effects for Hypnotizability, $F(2, 33) = 6.35$, MSe = 6.92, $P = .005$, indicating that the analgesic effect of the suggestion was experienced as occurring more automatically in HH subjects as compared to medium and LH ones (7.6, 4.7, and 3.8, respectively).

The ANOVA carried out for the number of omitted targets across treatments yielded a main effect for Hypnotizability, $F(2, 33) = 5.12$, MSe = 4.47, $P = .012$, indicating that HH subjects had a greater mean number of omissions as compared to MH and LH subjects (1.75, .93, and .53, respectively). Moreover, the main effect for Treatment was highly significant, $F(3.7, 123.3) = 9.58$, MSe = 2.09, $\varepsilon = .934$, $P < .0001$, as it was the case for the Treatment and Hypnotizability interaction, $F(7.5, 123.3) = 6.75$, MSe = 2.09, $\varepsilon = .934$, $P < .0001$. Post-hoc comparisons of the means indicated that for HH subjects the number of omitted targets was more pronounced during Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain ($t = 3.7$, $P < .01$, and $t = 6.5$, $P < .0001$). The t test also indicated that there

were more omissions during Hy-Analgesia and P.Hy-Analgesia in comparison with Hy-Pain treatment ($t = 4.9$, $P < .001$, $t = 5.6$ $P = 0.001$, respectively). The increase in the number of omitted targets was also significant for the P.Hy-Analgesia as compared to the Hy-Analgesia treatment ($t = 4.3$, $P < .001$). The mean number of omitted targets during treatments for the hypnotizability groups is reported in Table 1.

The ANOVA performed on both contingent and recalled pain ratings with Recollection and Treatment as within factors and Hypnotizability as a between factor yielded a significant main effect for Treatment, $F(2.9, 95.0) = 10.19$, MSe = 2.22, $\varepsilon = .722$, $P < .0001$, and a significant interaction between Hypnotizability and Treatment, $F(5.8, 95.0) = 5.04$, MSe = 2.22, $\varepsilon = .722$, $P = .0002$. Post-hoc comparisons of the overall pain means displayed that in HH participants there were significant reductions of both experienced and remembered pain during Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain and to W-Analgesia treatment (W-Pain vs Hy-Analgesia and P.Hy-Analgesia: $t = 4.6$ and $t = 4.95$, $P < .001$; W-Analgesia vs Hy-Analgesia and P.Hy-Analgesia: $t = 2.4$, $P < .05$ and $t = 3.9$, $P < .01$). The t-tests showed that there was a significant reduction in experienced and remembered pain scores from Hy-Pain to Hy-Analgesia and P.Hy-Analgesia treatments ($t = 4.6$, and $t = 5.1$, $P < .001$), and a significant reduction in pain measures during P.Hy-Analgesia as compared to Hy-Analgesia treatment ($t = 3.2$, $P < .01$). For HH participants, differences in pain ratings between W-Pain and W-Analgesia and between the latter and Hy-Pain treatment were not significant ($t = .9$, and $t = 1.4$, $P > .05$, respectively), while, for these subjects, the reduction of pain during Hy-Pain, with respect to W-Pain treatment, was near the significance level ($t = 2.1$, $P = .06$; see Fig. 1).

Table 1
Mean number and standard deviation (SD) of omitted target stimuli for high, medium, and low hypnotizable participants (HH, MH, and LH) during waking (W) and hypnosis (Hy) treatments

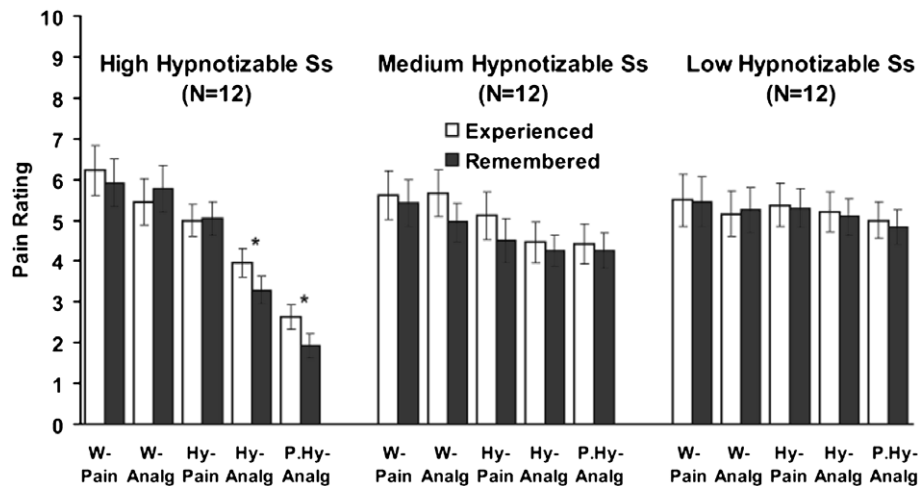| Hypnotizability	N	Treatment	Mean	SD
HH	12	W-Pain	0.33	0.65
		W-Analgesia	0.83	1.40
		Hy-Pain	0.42	1.16
		Hy-Analgesia	2.17	1.27
		P.Hy-Analgesia	5.00	2.59
MH	12	W-Pain	0.60	1.72
		W-Analgesia	0.64	1.62
		Hy-Pain	0.67	1.15
		Hy-Analgesia	1.25	1.96
		P.Hy-Analgesia	1.50	1.93
LH	12	W-Pain	0.50	1.00
		W-Analgesia	0.50	1.45
		Hy-Pain	0.75	2.60
		Hy-Analgesia	0.58	0.99
		P.Hy-Analgesia	0.33	0.89

Fig. 1. Means of Experienced Sensory Pain and Remembered Pain with standard errors to electrical stimulation by hypnosis group and condition (Waking-Pain, Waking-Analgesia; Hypnosis-Pain; Hypnosis-Analgesia; Post-Hypnosis Analgesia). The asterisk (*) indicates significant differences between experienced and remembered pain scores during analgesic treatments in hypnosis (*$P < .05$, t test).

Pain reductions obtained for LH participants did not reach the significance level (all ts < 2, $P > .05$). The MH participants reported smaller pain ratings during Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain ($t = 2.3$, and $t = 2.4$, $P < .05$) and to W-Analgesia treatment ($t = 2.2$, and $t = 2.4$, $P < .05$).

Furthermore, a significant three-way interaction was obtained for the Hypnotizability, Recollection, and Treatment factors, $F(6.2, 101.6) = 2.86$, $MSe = .523$, $\varepsilon = .77$, $P < .05$. This interaction indicated that HH subjects, for hypnotic analgesia and posthypnotic analgesia treatments, recollected significantly lower pain sensations as compared to the pain reported in the concurrent ratings ($t = 2.8$, $P = .020$, and $t = 3.1$ $P = .011$, respectively, for experienced vs concurrent pain ratings of the Hy-Analgesia, and P.Hy-Analgesia conditions). The patterns of experienced and remembered pain ratings across hypnotizability groups are displayed in Fig. 1.

A similar analysis performed on experienced and remembered distress ratings showed that the main effect of Hypnotizability was near to reach the significance level, $F(2, 33) = 2.97$, $MSe = 35.66$, $P = .065$, indicating that HH participants tended to have lower distress levels than MH and LH ones (4.4, 5.5, 6.2, respectively). The Recollection factor was highly significant, $F(1, 33) = 20.73$, $MSe = 1.32$, $P < .0001$, indicating that all the subjects, in the Recollection phase, rated a lower level of distress than they did contingently with treatments. The Treatment effect was also highly significant, $F(2.8, 92.9) = 15.19$, $MSe = 2.53$, $\varepsilon = .704$, $P < .0001$, as well as the interaction between Hypnotizability and Treatment, $F(5.6, 92.9) = 7.18$, $MSe = 2.53$, $\varepsilon = .704$, $P < .0001$. Post-hoc comparisons of the overall pain means displayed that in HH participants there were significant reductions of both experienced and remembered

distress scores during Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain and to W-Analgesia treatment (W-Pain vs Hy-Analgesia and P.Hy-Analgesia: $t = 7.3$ and $t = 6.9$, $P < .0001$; W-Analgesia vs Hy-Analgesia and P.Hy-Analgesia: $t = 4.2$, $P < .01$ and $t = 6.1$, $P < .0001$). These subjects also displayed a significant reduction in experienced and remembered distress scores during Hy-Analgesia and P.Hy-Analgesia treatments as compared to Hy-Pain ($t = 5.9$, and $t = 6.1$, $P < .0001$), and a significant reduction in distress measures during P.Hy-Analgesia as compared to Hy-Analgesia treatment ($t = 3.5$, $P < .01$). In HH subjects distress rating measures did not differ between W-Pain and W-Analgesia ($t = 1.8$, $P > .05$) and between W-Analgesia and Hy-Pain treatment ($t = .36$, $P > .05$), while these measures were significantly reduced during Hy-Pain as compared to W-Pain treatment ($t = 2.9$, $P < .05$; see Fig. 2). Distress reductions, obtained for LH participants, did not reach the significance level (all ts<2, $P > .05$). The MH participants reported smaller distress ratings during Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain ($t = 2.4$, and $t = 2.2$, $P < .05$) and to W-Analgesia treatment ($t = 2.3$, and $t = 2.4$, $P < .05$).

Finally, a significant three-way interaction was obtained for the Hypnotizability, Recollection and Treatment factors, $F(6.9, 114.7) = 2.68$, $MSe = .653$, $\varepsilon = .869$, $P < .05$. This interaction indicated that HH subjects, for hypnotic analgesia and posthypnotic analgesia treatments, recollected significantly lower distress sensations as compared to concurrent painful stimulation ($t = 4.3$, $P < .010$, and $t = 4.5$, $P < .010$, respectively, for experienced vs concurrent pain ratings of the Hy-Analgesia, and P.Hy-Analgesia conditions). The trend of this interaction, observed for the HH subjects, can be derived from the patterns of the experienced and remembered pain ratings displayed in Fig. 2.
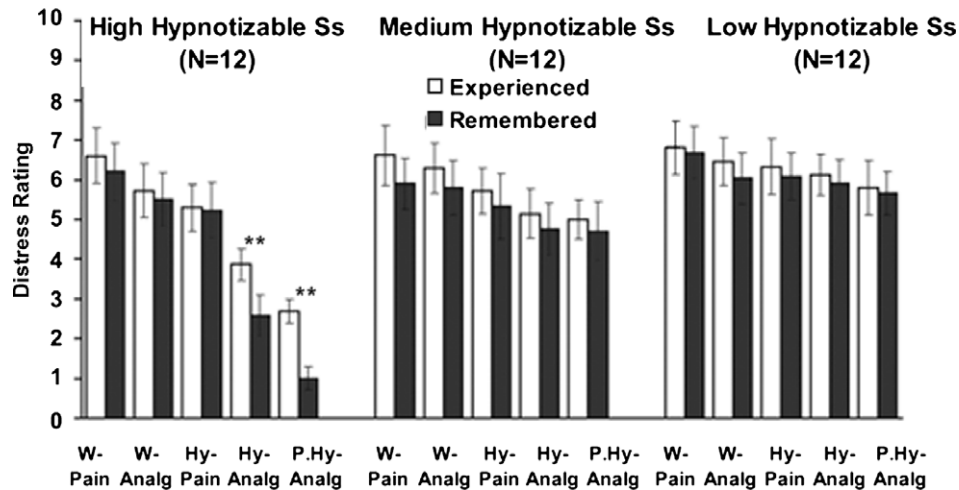
Fig. 2. Means of Experienced Distress and Remembered Distress with standard errors to electrical stimulation by hypnosis group and condition (Waking-Pain, Waking-Analgesia; Hypnosis-Pain; Hypnosis-Analgesia; Post-Hypnosis Analgesia). The two asterisks (**) indicate significant differences between experienced and remembered pain scores during analgesic treatments in hypnosis (**$P < .01$, t test).

To test the impact of hypnosis on the response to suggestions, separate ANOVAs on pain and distress change scores were performed. A main effect for Hypnotizability was found for pain change scores, $F(2, 33) = 4.90$, MSe $= 3.06$, $P = .014$, and for distress change scores, $F(2,33) = 4.98$, MSe $= 3.72$, $P = .013$. These effects indicated that HH subjects had more pronounced pain and distress reductions as compared to MH and LH subjects, while there were no significant differences between MH and LH subjects (Pain: 1.39, .43, and .29; Distress: 1.64, .51, and .37, respectively, for HH, MH, and LH subjects). The Condition main effect was also significant for pain change scores, $F(1.9, 64.8) = 5.59$, MSe $= 1.34$, $\varepsilon = .982$, $P = .007$, and distress change scores, $F(1.9, 62.7) = 5.93$, MSe $= .97$, $\varepsilon = .951$, $P = .005$. Post-hoc comparison of the means showed that the effect of pain reduction was significantly stronger during P.Hy-Analgesia as compared with W-Analgesia and Hy-Analgesia, while there were no significant differences between the last two conditions (Pain: 1.15, .34, and .62, respectively; $t = 2.47$, $P = .019$, for P.Hy-Analgesia vs W-Analgesia; $t = 2.17$, $P = .037$, for P.Hy-Analgesia vs Hy-Analgesia; $t = 1.04$, $P > .05$, for Hy-Analgesia vs W-Analgesia). Similarly, distress reduction was significantly stronger during P.Hy-Analgesia as compared with W-Analgesia and Hy-Analgesia, while there were no significant differences between the last two conditions (Distress: 1.27, .54, and .72, respectively; $t = 2.52$, $P = .016$, for P.Hy-Analgesia vs W-Analgesia; $t = 2.73$, $P = .009$, for P.Hy-Analgesia vs Hy-Analgesia; $t = .83$, $P > .05$, for Hy-Analgesia vs W-Analgesia).

The interaction between Hypnotizability and Condition was also significant for pain change scores, $F(3.9, 64.8) = 2.55$, MSe $= 1.34$, $\varepsilon = .982$, $P = .048$, and distress change scores, $F(3.8, 62.7) = 2.67$, MSe $= .97$, $\varepsilon = .951$, $P = .042$. These interactions indicated that in

HH subjects the effect of suggestion was more pronounced and significant during P.Hy-Analgesia as compared to W-Analgesia (Pain: 2.60 vs .77, $t = 3.62$, $P = .004$; Distress: 2.70 vs .92, $t = 4.58$, $P = 0.0008$), and to Hy-Analgesia (Pain: 2.60 vs 1.04, $t = 3.27$, $P = .007$; Distress: 2.70 vs 1.42, $t = 3.15$, $P = 0.009$), while the difference between Hy-Analgesia and W-Analgesia was not significant (Pain: 1.04 vs .77, $t = .46$, $P > .05$; Distress: 1.42 vs .92, $t = 1.73$, $P > 0.05$). Difference scores for MH and LH subjects were all less than 0.75 and all comparisons between conditions were non-significant (all $t < 1.5$, $P > .05$).

3.2. Effects of experimental factors on N140 and P200 peak amplitudes

With the onset of the standard electric stimulus we observed a complex consisting of two components, a N140 peak that displayed a maximum amplitude at fronto-central sites (peak latency of 142 ms at Fz and 139 ms at Cz), and a P200 peak that showed a maximum amplitude at central site (peak latency of 196 ms at Cz).

3.2.1. Quadrant N140 peak amplitude
For quadrant N140 amplitude scores, the ANOVA yielded the following significant effects: (1) Treatment, $F(1.8, 61.4) = 6.19$, MSe $= 10.20$, $\varepsilon = .465$, $P = .004$; (2) Recording Site, $F(1, 33) = 79.21$, MSe $= 64.40$, $P < .0001$; (3) Treatments and Recording Site interaction, $F(2.7, 90.4) = 4.36$, MSe $= 4.40$, $\varepsilon = .684$ $P = .0118$; (4) Hypnotizability and Treatment interaction, $F(3.7, 61.4) = 3.54$, MSe $= 10.2$, $\varepsilon = .465$, $P = .013$. The first effect indicated that there was a more pronounced N140 peak during W-Pain and Hypnosis-Pain as compared to the other three analgesic conditions

(10.2, 9.5, 10.5, 9.3, and 9.2 µV, respectively, for W-Pain W-Analgesia, Hy-Pain Hy-Analgesia, and P.Hy-Analgesia; t values were all significant, $P < .05$). The second effect showed a more pronounced peak over frontal sites as compared to the parietal ones (12.3 vs 7.2 µV). The third effect disclosed that, for Hy-Analgesia and P.Hy-Analgesia, as compared to Hy-Pain treatment, there was a smaller N140 peak over frontal and parietal scalp sites; frontal site: $t = 4.2$, $P < .001$ and $t = 3.8$, $P < .001$; parietal site: $t = 2.2$, $P < .05$ and $t = 3.0$, $P < .01$. In contrast, there were no significant differences between W-Analgesia and W-Pain treatments ($P > .05$). The fourth effect showed that N140 peak reduction was mainly due to the HH participants that produced significant smaller N140 peak amplitudes during Hy-Analgesia and P.Hy-Analgesia as compared to Hy-Pain (8.0 and 7.7 vs 10.9 µV, respectively; t values were 7.7, and 6.7, $P < .0001$) and as compared to W-Pain treatment (8.0 and 7.7 vs 10.2 µV, respectively; t values were 3.1, and 3.3, $P < .01$, respectively). In contrast, for these subjects, the reduction in N140 amplitude during W-Analgesia as compared to W-Pain did not reach the significance level (9.0 vs 10.2 µV, respectively, $t = 2.0$, $P = 0.068$). Similarly, for these subjects, there were no significant differences in N140 amplitude between Hy-Analgesia and P.Hy-Analgesia treatments ($t = 1.1$ $P > .05$).

3.2.2. Midline N140 peak amplitude

The ANOVA on midline N140 amplitudes yielded the same significant effects as those evidenced for quadrant data: Treatment, $F(1.8, 59.0) = 5.07$, MSe = 11.15, $\varepsilon = .447$, $P = .017$; Recording Site, $F(2, 66) = 101.04$, MSe = 48.29, $\varepsilon = 1.101$, $P < .0001$; Treatment and Recording Site interaction, $F(4.0, 130.9) = 4.37$, MSe = 3.28, $\varepsilon = .496$, $P = .0025$; Hypnotizability and Treatment interaction, $F(3.6, 59.0) = 5.38$, MSe = 11.15, $\varepsilon = .447$, $P = .0015$. Moreover, this analysis evidenced a significant triple interaction between Hypnotizability, Treatments, and Recording Site, $F(7.9, 131.5) = 2.53$, MSe = 3.23, $\varepsilon = .498$, $P = .013$. This effect indicated that, across Fz and Cz recording sites, HH participants had more pronounced N140 amplitude reductions during Hy-Analgesia and P.Hy-Analgesia as compared to Hy-Pain (Fz: 11.4 and 11.7 vs 16.7 µV, respectively; t values were 6.6, and 6.0, $P < .0001$; Cz: 12.2 and 12.6 vs 17.4 µV, respectively; t values were 7.4, and 6.9, $P < .0001$) and as compared to W-Pain treatment (Fz: 11.4 and 11.7 vs 15.6 µV, respectively; t values were 3.5, and 3.2, $P < .01$, respectively; Cz: 12.2 and 12.6 vs 15.9 µV, respectively; t values were 2.8, and 2.5, $P < .05$, respectively). For HH subjects, there were no significant differences on midline N140 amplitude between W-Analgesia and W-Pain (all $t < 2$, $P > .05$) and between Hy-Analgesia and P.Hy-Analgesia treatments (all $t s < 1$, $P > .05$). SERP

responses recorded at midline locations are displayed in Fig. 3.

3.2.3. Quadrant P200 peak amplitude

Quadrant P200 amplitude scores were found to be modulated by the same significant effects as those evidenced for the N140 peak: (1) Treatment, $F(2.5, 82.2) = 6.02$, MSe = 9.19, $\varepsilon = .623$, $P = .0018$; (2) Recording Site, $F(1, 33) = 19.89$, MSe = 36.32, $P < .0001$; (3) Treatments and Recording Site interaction, $F(3.7, 123.7) = 3.76$, MSe = 3.38, $\varepsilon = .937$ $P = .008$; (4) Hypnotizability and Treatment interaction, $F(5.0, 82.2) = 2.63$, MSe = 9.19, $\varepsilon = .623$, $P = .052$. The Treatment effect indicated that this positive peak was smaller during Hy-Analgesia and P.Hy-Analgesia as compared to Hy-Pain treatment (6.1, 6.5, and 7.6 µV, respectively; all $t s > 3.5$, $P < .001$), while the difference in peak amplitude between W-Pain and W-Analgesia did not reach the significance ($t = 1.1$, $P > .05$). The Recording Site effect showed a higher peak over frontal region as compared to the parietal one (7.9 vs 5.9 µV). The Treatment and Recording Site interaction effect disclosed that over the frontal region, during Hy-Analgesia and P.Hy-Analgesia, there were smaller peaks as compared to Hy-Pain condition, while these differences were not significant over parietal one (frontal region: $t = 5.0$, $P < .001$ and $t = 3.11$, $P < .01$; parietal region: $t = 1.7$, $P > .051$ and $t = 1.4$, $P > .05$). The Hypnotizability and Treatment interaction showed that HH participants (but not MH and LH participants) produced significant smaller P200 peak amplitudes during Hy-Analgesia and P.Hy-Analgesia treatments as compared to Hy-Pain (5.6 and 6.1 vs 8.1 µV, respectively; t values were 9.7, $P < .0001$, and 3.0, $P < .01$) and as compared to W-Pain treatment (5.6 and 6.1 vs 8.1 µV; t values were 3.2, $P < .01$, and 2.3, $P < .05$, respectively). For these subjects, there were no significant differences on P200 amplitude between W-Analgesia and W-Pain (8.0 vs 8.1 µV; $t < 1$, $P > .05$) and between Hy-Analgesia and P.Hy-Analgesia treatments (5.6 vs 6.1; $t < 1$, $P > .05$).

3.2.4. Midline P200 peak amplitude

The ANOVA on P200 amplitude over midline recordings yielded the same significant effects as those evidenced for quadrant data: Treatment, $F(3.0, 99.1) = 7.58$, MSe = 8.87, $\varepsilon = .751$, $P < .0001$; Recording Site, $F(2, 66) = 50.71$, MSe = 51.51, $\varepsilon = 1.099$, $P < .0001$; Treatment and Recording Site interaction, $F(4.5, 147.6) = 2.29$, MSe = 2.44, $\varepsilon = .559$, $P < .0001$; Hypnotizability and Treatment interaction, $F(6.0, 99.1) = 3.92$, MSe = 8.87, $\varepsilon = .751$, $P = .0015$. Moreover, a significant triple interaction between Hypnotizability, Treatments, and Recording Site was also obtained, $F(8.9, 147.6) = 2.59$, MSe = 2.44, $\varepsilon = .559$, $P = .0085$. This effect indicated that, across Fz and Cz
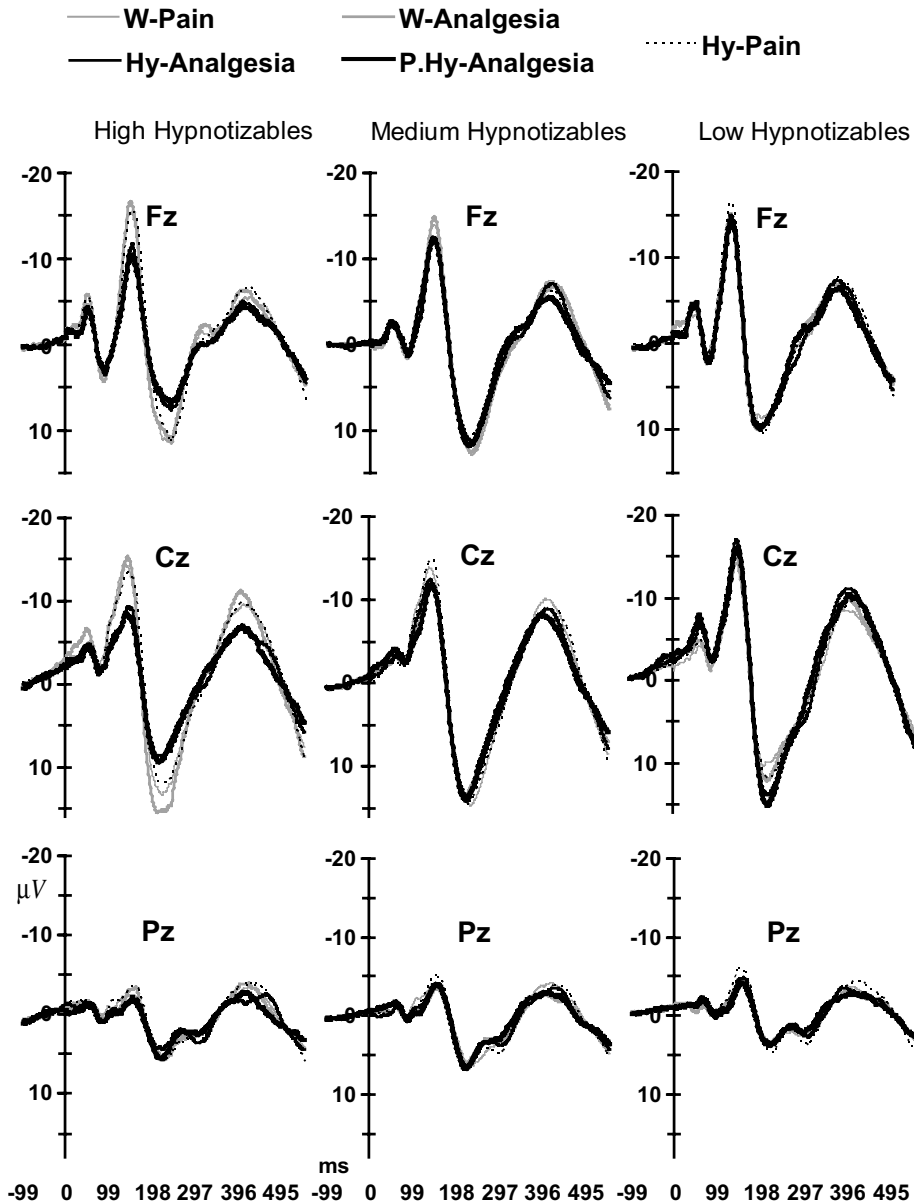
Fig. 3. Somatosensory event-related potentials (ERPs) to painful standard stimuli averaged across frontal, central, and parietal midline sites in high, medium and low hypnotizable subjects during Waking, Hypnosis and Post-Hypnosis conditions for Pain and Analgestia treatments.

recording sites, HH participants had more pronounced P200 amplitude reductions during Hy-Analgesia and P.Hy-Analgesia as compared to Hy-Pain (Fz: 8.0 and 8.4 vs 12.6 µV, respectively; t values were 4.5, and 4.6, $P < .001$; Cz: 9.6 and 10.2 vs 14.4 µV, respectively; t values were 4.5, and 4.2, $P < .0001$) and as compared to W-Pain treatment (Fz: 8.0 and 8.4 vs 12.8 µV, respectively; t values were 4.2, and 3.2, $P < .01$, respectively; Cz: 9.6 and 10.1 vs 15.4 µV, respectively; t values were 4.5, and 4.5, $P < .001$, respectively). For HH subjects, there were no significant differences, across midline locations, for P200 amplitude between W-Analgesia and W-Pain (all $ts < 2$, $P > .05$) and between Hy-Analgesia and P.Hy-Analgesia treatments (all $ts < 1$, $P > .05$). SERP

responses recorded at midline locations are displayed in Fig. 3.

3.3. Relation between pain and distress ratings and ERP responses

Correlation coefficients, obtained between rating measures and N140 and P200 peak amplitudes, did not yield any significant relationship, except for a significant correlation found between P200 amplitude over left-parietal scalp location (P3) and pain ratings obtained during P.Hy-Analgesia treatment ($r = .34$, $P < .05$). However, in order to evaluate how changes in experienced pain and distress scores, as induced by

analgesic treatments, were related with changes in N140 and P200 amplitudes, Pearson's correlation coefficients were calculated. Change scores were calculated by subtracting scores obtained during W-Analgesia from those obtained during W-pain, and scores obtained during Hy-Analgesia and P.Hy-Analgesia from those during Hy-Pain. The reduction of experienced pain was significantly correlated with the reduction of midline frontal N140 amplitude during Hy-Analgesia and P.Hy-Analgesia treatments ($r = .40$, $P < .05$ and $r = .43$, $P < .01$, respectively). Similarly, the reduction of experienced distress was significantly correlated with the reduction of midline frontal N140 amplitude during Hy-Analgesia and P.Hy-Analgesia treatments ($r = .43$, $P < .01$ and $r = .44$, $P < .01$, respectively). No significant correlations were found between pain or distress reductions and P200 amplitudes of the ERPs.

4. Discussion

Self-report data displayed differential patterns of responding with MH individuals showing a moderately lower level of pain during hypnosis as compared to waking condition, and LH individuals showing little differentiation between the waking, hypnosis, and post-hypnosis conditions. In contrast, HH individuals displayed significantly lower pain and distress scores under Hy-Analgesia and P.Hy-Analgesia as compared to W-Pain and Hy-Pain conditions. These results are consistent with a number of studies showing that HH individuals are more responsive to hypnotic analgesia suggestions [2,10,14,47,48,50,51,61]. Moreover, the finding that HH subjects experienced significant pain and distress reductions during P.Hy-Analgesia as compared to Hy-Analgesia condition supports the assumption that the magnitude of responses to suggestions is enhanced during a more intense individual's hypnotic involvement [28,53]. The findings that pain and distress ratings did not differ between W-Pain and Hy-Pain treatments among hypnotizability groups (see Figs. 1 and 2) suggest that differential respondings to painful stimuli were due to the influence of the hypnotic suggestions rather than to an effect of the hypnotic induction per se. In fact, in the present study, MH and LH individuals were less responsive to the hypnotic suggestions than the HH individuals, although the former during Hy-Analgesia and P.Hy-Analgesia reported smaller pain and distress ratings than during W-Pain and W-Analgesia treatments (see white columns on Figs. 1 and 2).

It is important to note that, in the present study, in line with the recommendation of [38], the impact of hypnosis on the response to suggestion was tested by delivering the exact same suggestion to subjects in and out of hypnosis. The findings indicate that in a condition of deep hypnosis (as it is assumed to be the case of post-hypnosis condition), the analgesic effect of suggestions

was amplified in comparison with both the waking non-hypnotic condition and the standard hypnotic condition.

In terms of retrospective pain and distress ratings, the present results indicate that HH subjects, five minutes after the conclusion of hypnosis, over-evaluated the effect of pain reduction they experienced during Hy-Analgesia and P.Hy-Analgesia treatments. In contrast, for W-Pain, W-Analgesia, and Hy-Pain conditions, these subjects remembered similar pain and distress levels to those they concurrently experienced with stimulation.

It is known that negative affect increases acute pain report and has been implicated with the level of maximum pain that is recalled and in the extent to which the memory of pain becomes distorted across time [24,25,56]. The more pronounced pain and distress ratings, as recollected for waking and hypnosis-pain conditions, provide an experiential context through which the painful stimulus is processed and later better recalled. This interpretation is consistent with research suggesting that emotionally arousing or stressful experiences are generally well remembered, and that pain processing and recall are highly dependent upon contextual cues associated with the painful experience [45]. It may be that the more pronounced memory distortions of pain and distress, as observed during hypnotic analgesic treatments, can be explained as a proactive effect of the reduced emotional distress during these hypnosis treatments.

In terms of SERP's changes, the most striking feature is a drop in amplitude for both the N140 and P200 peak amplitudes, observed for the HH individuals, over frontal and central recordings during both Hy-Analgesia and P.Hy-Analgesia treatments. The expected more pronounced peak amplitude reduction during P.Hy-Analgesia, as compared to Hy-Analgesia treatment, was not observed. This apparent lacking difference may be due to a floor effect since the drop in amplitude for N140 and P200 components, during analgesic treatments in hypnosis, was quite large (about 5 μV, see Fig. 3). For the MH and LH subjects, no significant amplitude changes were observed during analgesic treatments in hypnosis as compared to painful treatments.

There is experimental evidence that the somatosensory N140 peak increases with selective attention [18,19,23,41,43]. However, the drop in amplitude, that we observed for the N140 component during Hy-Analgesia and P.Hy-Analgesia treatments in HH subjects, cannot be univocally seen as a reflection of reduced arousal. This is because no effect of a hypnotic induction alone, usually associated with a reduced level of arousal, was observed on N140 amplitude (i.e., the N140 amplitude during Hy-Pain was not smaller than during W-Pain). Considering that during Hy-Analgesia and P.Hy-Analgesia pain and distress reductions were found

significantly correlated with the reductions of frontal N140 amplitude, it can be suggested that, during hypnotic analgesic treatments, the drop in N140 amplitude is the product of the modulation of pain experience associated with the obstructive nature of the "glove analgesia" suggestion. This suggestion implies a modification or reinterpretation of the sensation as non-painful rather than a distraction from the pain sensation since the oddball paradigm requires the subject to attend the stimulus.

A variety of studies suggest that the later positive components of the SERPs are related to the painfulness of the stimuli and that these components may even be lacking when the stimuli are not experienced as painful [4,26]. Thus, assuming that the P200 component is a pain specific component of the SERPs [4], the reduced P200 peak, observed in the present study during hypnotic and posthypnotic analgesia treatments, can be seen as reflecting the operation of an inhibitory mechanism in the pain processing system. This inhibitory process may be more of a global than specific nature since it involves attentional and pain-specific components of the SERPs. A similar inhibitory effect, involving negative and positive ERP components, has been also reported in previous hypnosis studies using obstructive imagery of incoming visual [11,54,55] and somatosensory stimuli [14,15,48].

In the present study, both N140 and P200 components displayed a more fronto-central focus with a greater modulation of their peak amplitudes as a function of hypnotic involvement. This observation parallels the findings reported by [48] who reported two negative components of the ERPs maximal at vertex (N140 and P250) in response to electric stimulation and suggested to reflect the activity of cingulate and subcortical areas. Thus, the present findings appear in agreement with the hypothesis that hypnotic suggestions, in HH individuals, influence the somatosensory areas of the cortex. The statement that primary somatosensory cortex plays a leading role in pain perception and is important for the discrimination and modulation of various aspects of pain is derived by animal and human studies (see [6,20]). This modulation consists in the inhibition of the sensory areas through cortico-thalamic descending projections.

In conclusion, our findings that obstructive suggestions reduced both the early N140 and the later P200 peaks in high susceptible subjects under hypnosis support the hypothesis that hypnosis procedure can affect earlier and later stages of stimulus processing (see Fig. 3). The early effect (N140) may be related to differences in arousal in the relevant circuitry that may be mediated by structures such as the parts of the ascending colinergic reticular arousal system [49] or the intralaminar nuclei of the thalamus [35]. The later effects (i.e., the reduced P200 peak amplitude for the hypnotic analgesic

conditions) can be seen as a reflection of the reduced activity in the anterior cingulate or prefrontal cortex, known to be activated as parts of the aversive response [7,9,17].

Although this study provides experimental evidence that hypnosis is a valid tool for pain reduction in HH subjects, the interpretation of the present finding is limited by the potential emotional effect of the oddball stimuli involving painful shocks. The parameters used in this study for the oddball target stimuli suggest that they were likely to be perceived as quite painful (i.e. a pair of standard stimuli which were adjusted to be close to, or at, tolerance level). This implies that the response to standard stimuli may actually involve an anticipatory component that may induce some anxiety associated with the risk of receiving a more painful shock. The analgesic effect reported may, therefore, reflect both a direct effect on the response to standard stimuli as well as an indirect effect due to the reduction in the anxiety associated to the very painful target stimuli. Therefore, to avoid this potential emotional effect, future oddball ERP studies on pain modulation in hypnosis should use standard and target stimuli with similar intensity and randomized interstimulus intervals.

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