Hypnotic analgesia, obstructive attentional processes and psychophysiological mechanisms

Vilfredo De Pascalis

This address reviews psychophysiological research on the effects of different hypnotic analgesia suggestions in the reduction of pain perception. Experimental evidence is provided in demonstrating that hypnotic analgesia is the product of an inhibitory processing which involves focused attention and obstructive hallucination. The first mechanism is the product of frontal cortical activity, the second is mainly the product of posterior cortical system which modulates mental imagery. The validity of these statements is provided with traditional EEG rhythms, event-related potentials, and autonomic responses. Findings from our laboratory indicate that different processes are engaged by different suggestions of analgesia, but how the mechanism of hypnotic analgesia is started on by different hypnotic suggestions it remains an unsolved question.

Introduction

Hypnotic approaches for pain control, as for cognitive-behavioral methods, usually involves direct or indirect suggestions to use cognitive strategies devoted to cope with pain such as distraction imagery, obstructive imagery, relaxation, etc. A number of studies have provided experimental evidence that, independently from the treatments used, analgesia suggestions in hypnosis significantly reduce pain perception in highly hypnotizable participants. Therefore, hypnotic analgesia is considered the most reliable of hypnotic phenomena (Hilgard & Hilgard, 1994; Barber, 1996; Chaves & Dworkin, 1997; Zachariae & Bjerring, 1994; De Pascalis et al., 1999; Crawford, 1994). However, despite the great number of reports supporting the marked effect of hypnotic analgesia, the basic psychophysiological mechanisms by which it is achieved still remain obscure. It is known that hypnotic suggested analgesia is a multicomponent process in which a number of different factors as stable individual hypnotizability traits, attentional mechanisms, and expectancies, may modulate individual capacity to control pain. A number of brain imaging studies, using positron emission tomography

(PET) (see e.g., Casey, 1999; Peyron et al., 2000; Willis & Westlund, 1997; Rainville et al., 1997), and functional magnetic resonance imaging (fMRI) (see e.g., Disbrow et al., 1999; Davis, 2000) found multiple cortical and subcortical sites engaged in pain processing. Brain imaging studies using various types of experimental pain in humans, have evidenced reliable patterns of activation in the cortex (see e.g., Jones et al., 1991; Talbot et al., 1991; Craig et al., 1996; Aziz et al., 1997). The regional cerebral blood flow (rCBF) was found to increase in the cortical regions of contralateral primary somatosensory cortex (S1), the secondary somatosensory cortex (S2), anterior and posterior insula and anterior cingulate cortex (ACC). PET studies reveal that hypnosis and attention diversion shared similar cortical and subcortical circuitry by displaying (1) an increased activity in orbitofrontal and subcallosial cingulate cortices, right thalamus, left inferior parietal cortex accompained with a decreased activation in the cingulate cortex (Wik et al., 1999); (2) the activation of left-sided occipital, parietal, precentral, premotor, and ventrolateral prefrontal cortices as well as few right-sided occipital and anterior cingulate cortices (Maquet et al., 1999). In a recent study of Rainville et al. (1999), hypnosis alone produced a significant correlation of occipital rCBF and delta EEG power in the occipital site; the authors reported hypnosis related decreases in rCBF in the right inferior parietal lobule, the left precuneus, and the posterior cingulate gyrus. Hypnosis with suggestions of pain reduction produced additional wide-spread increases in rCBF in the left frontal cortices, while the medial and lateral posterior parietal cortices showed suggestion-related increases overlapping partly with above mentioned regions of hypnosis-related decreases. This wide-spread effect was more extensive than that evidenced in a previous report (Rainville et al., 1997) wherein reductions in ACC activation correlated significantly with reduced perception of distress, whereas primary somatosensory cortex activation was unaltered in hypnosis. These findings provided experimental evidence for frontal limbic lobe activation with emotional-affective component of pain. Suggestions directed at increasing sensory intensity of pain produced parallel modulations of pain intensity and unpleasantness and were accompained by significant larger pain-related activity in S1 with smaller effects in the insula, S2 and ACC. Suggestions in hypnosis devoted to modulate pain affect produced parallel changes in subjective unpleasantness and on ACC activity. Findings by Rainville and colleagues have provided experimental evidence for a partial differentiation of cortical structures involved in pain sensation and pain affect and showed that hypnotic suggestions modulate the activity in cortical areas engaged in the perception of pain.

Notwithstanding the recent substantial progress in the neurophysiology of pain obtained with neuroimaging methods, the basic processes accounting for hypnotic analgesia still remain unknown.

Most of the neurophysiological studies on hypnosis analgesia have attempted to explore basic mechanisms explaining analgesia effect in hypnosis. These studies were mainly focused in evidencing cortical areas linked to an inhibitory mechanism enga-

ged for pain reduction rather than to the relationship between the type of attention-cognitive processes engaged by the subjects for pain relief and the consequent neurophysiological mechanisms. Independently from hypnotic analgesia suggestions used by the subject, studies usually report an inhibitory process as responsible for hypnotic analgesia phenomenon. Since different mental activities may be associated to activations of different cortical areas and/or hemispheres, there are at least two questions of concerns: (1) How different mental activities may prime the same inhibitory process? (2) Which components of the SERPs (or other neural activity responses) are expressions of the ongoing pain specific activity and which components are mostly expression of the ongoing cognitive activity engaged for pain relief? In a recent study, Becker et al. (2000) provided experimental evidence showing that oddball standards protocol is a valid method for detecting an anterior P3 pain-specific peak of the SERPs. Somatosensory event-related potentials (SERPs), evoked by painful phasic stimuli, have been tested as valid electrocortical indicators of pain (Bromm, 1984, 1989, 1995). A number of hypnosis studies among high hypnotizables reports decreases in late SERP positive components in medial frontal, central, temporal, and parietal regions during hypnotic analgesia (Arendt-Nielsen et al., 1990; Crawford, 1994; Crawford et al., 1998; De Pascalis et al., 1992, 1999; Galbraith et al., 1972; Meszaros et al., 1980; Spiegel et al., 1989; Zachariae & Bjerring, 1994). Some other studies of late SERPs during hypnotic analgesia have reported opposing effects between pain perception and late SERP peak amplitudes (see e.g., Halliday & Mason, 1964; Meier et al., 1993). In some previous studies (De Pascalis & Carboni, 1997; Spiegel et al., 1989; Spiegel & Barabasz, 1988), obstructive hallucination under hypnosis, compared to a normal attention condition, showed significant reductions in somatosensory perception and in P3 peak amplitude of the SERPs. In a recent report, Crawford et al. (1998) found that moderate and high hypnotizable patients with low back pain had not only reduced P2 and P3 amplitudes on posterior regions during hypnotic analgesia, but also changes over anterior frontal regions. The anterior frontal changes consisted of an enhanced N140 and a prestimulus positive going contingent variation at the left anterior frontal cortex. The enhanced negative peak during hypnotic analgesia was suggested to indicate a shift in the allocation of attention necessary for inhibitory processing. The frontal inhibition view of hypnosis was supported also by Gruzelier's findings (Gruzelier, 1996). However, findings from the recent report by Kallio et al. (1999) using mismatch negativity (MMN) of the ERPs, did not support the operation of an inhibitory mechanism in hypnosis. Using the amplitude of MMN as an index of cortical excitability, these authors reported significant larger MMNs in hypnosis as compared to a waking control condition.

In short, neurophysiological investigation offers hypnosis research a relatively new perspective in understanding the nature of individual differences in hypnotic susceptibility and hypnosis, including the mechanisms by which different varieties of hypnotic suggestions work.

In terms of EEG rhythms, a number of studies have displayed significant changes in

the conventional EEG-rhythms induced by hypnotic analgesia during tonic pain stimulation. The study by Chen et al. (1981) evidenced a reduction in the left-hemisphere of alpha and theta EEG power after hypnotic analgesia during dental surgery. Karlin et al. (1980) evidenced greater total EEG power in the right posterior region to cold pressor pain. Using cold pressor pain, Crawford (1990) reported that high hypnotizable individuals generated more high frequency theta (5.5-7.5 Hz) power than did low ones from mid-frontal to occipital sites. During cold pressor pain, highs had more left-hemisphere high-theta power while they showed, during hypnotic analgesia, a reduction in the left and an increase in the right hemisphere power. De Pascalis and Perrone (1996), during hypnotic analgesia of painful electric stimulations, reported significant reductions of total EEG spectral amplitude (0.50-31.75 Hz), delta (0.50-3.75 Hz) and beta1 (13-15.75 Hz) amplitudes. The amplitude reductions were more pronounced in the right hemisphere, resulting in a more pronounced hemispheric asymmetry in favor of the left hemisphere.

In the present report main pain-hypnosis findings are reported from studies carried out in the psychophysiology laboratory at the department of Psychology of the University of Rome 'La Sapienza'. These studies were carried out in the light of three main models of hypnosis. The first is a neurophysiological model of hypnosis (Gruzelier, 1988, 1990, 1996, 1998; Crawford & Gruzelier, 1992) in which hypnotic analgesia is viewed as the product of an active process engaging the anterior frontal cortex. According to this model, frontal lobe is part of a top-down inhibitory system which modulates thalamo-cortical activity by dissociating pain experience from conscious awareness. This model considers brain processes recruited by the ongoing hypnotic induction as essential for describing hypnotic phenomena. According to this model, hypnotic induction, in highly hypnotic susceptible persons, is conceptualized around three strategies: (1) instructing subject to focus on a small target object and to ignore distracting stimuli involves mainly focal attention and sustained attention, respectively thought as left and right frontal functions; (2) the first stage is replaced by eye closure, deep relaxation and tiredness, indicating that frontal functions are gradually inhibited by deep relaxation suggestions whereby anterior executive functions are suspended and directed by the induction with the suspenction of reality testing and critical evaluation; (3) the third stage involves a cortical activation shift from anterior to posterior and from left to right hemispheric functions through the facilitation of fantasy and dreaming and a relative inhibition of verbal and critical capacity.

The second model is the classic neodissociative model of hypnosis (Hilgard, 1977, 1979) which suggests that hypnotic analgesia is achieved through a dissociation phenomenon, i.e., pain is registered but remains dissociated from conscious awareness. The third model is a derivation of Hilgard's neodissociative model. It concerns the experience of nonvolition that usually accompaines hypnotic suggestions (Bowers, 1981, 1984; Weitzenhoffer, 1978). Since subsystems of control are activated, more or less directly, by hypnotic suggestions without executive initiative, hypnotized subjects

frequently experience suggested analgesia under effortless or nonvolitional personal control (dissociated control).

Experimental evidence is given here that perceptual alterations in hypnosis, as those of positive and negative hallucinations, are some of the most compelling experiences of highly hypnotizable subjects. According to Spiegel (1994), when subjects in hypnosis are fully 'absorpted' in perceptual imaginative or ideational experiences, cognitive resources are fully allocated to the central task, while information out of the attentional focus is dissociated from conscious awareness. Spiegel (1994) has defined hypnosis as a controlled and structured dissociation that provides a model for exploring neurophysiological correlates of dissociative processes.

In some previous studies (Spiegel et al., 1985; Spiegel et al., 1989; Spiegel & Barabasz, 1988; De Pascalis, 1994; De Pascalis & Carboni, 1997; Barabasz et al., 1999), obstructive hallucination of visual and somatosensory stimulation in high hypnotizables under hypnosis produced significant amplitude reductions in the P300 component of the ERPs.

In this report I summarize neurophysiological evidence suggesting that hypnotic analgesia is mainly the product of an inhibitory phenomenon in the brain wherein absorption on obstructive hallucination (rather than distraction) is the main way to have access to it.

Obstructive hallucination

In our first hypnosis-obstructive hallucination study (De Pascalis, 1994), highly hypnotizable persons displayed a decrease in P1, N1, and P3 peak amplitudes of the ERPs as a consequence of obstructive hallucination in hypnosis. Obstructive hallucination was induced with suggestions to imagine a cardboard that was blocking the view of a circular light spot. Results from this study confirmed those previously reported by Spiegel et al. (1985), Spiegel and Barabasz (1988), and Barabasz et al. (1995) by demonstrating that obstructive hallucination in hypnosis is the product of an inhibitory top-down process that involves the early preattentive stages of visual information processing. In a later study (De Pascalis & Carboni, 1997), the effects of obstructive hallucination previously found on visual ERPs was extended to somatosensory event-related potentials (SERPs). Target and standard electric stimuli of mild intensity were delivered on the right wrist to 10 high and 10 low hypnotizable women engaged in a somatosensory target detection task. Aim of the study was to evaluate the effects of hypnotic alterations of somatosensory perception on SERP peaks and evoked cardiac deceleration response. Results from this study evidenced that in high hypnotizable persons the suggestion to imagine a glove that was covering the stimulated wrist was effective in reducing stimulus perception. Highly hypnotizable persons, during obstructive hallucination in hypnosis, displayed reduced P300 peak amplitudes and pronounced heart rate (HR) decelerations in response to target stimuli.

These results confirmed the validity of the assumption that a top-down inhibitory process is responsible for hypnotic analgesia and suggested that hypnotic obstruction

is the product of the modulatory thalamocortical activity operated by frontal lobe activity.

Hypnotic analgesia

In our first study on hypnotic analgesia (De Pascalis & Perrone, 1996), we provided experimental evidence supporting the validity of dissociated control view of hypnosis (Bowers, 1992, 1994). This model suggests that hypnotic analgesia is the product of a dissociated control mechanism and mental imagery is a concomitant rather than a mediator of suggested analgesia. EEG spectral amplitude and HR variability (normalized spectral power density for mid frequency peak in the 0.06-0.14 Hz range, and high frequency peak in the 0.15-0.32 Hz range, respectively) were recorded. EEG activity was obtained from frontal and central scalp sites, and from the middle of temporo-parietal-occipital scalp junction. Highly hypnotizable subjects significantly reduced pain and distress levels during hypnotic analgesia. Hypnotic analgesia produced significant amplitude reductions in the total (0.5-31.75 Hz), delta (0.5-3.75 Hz), and beta1 (13-15.75 Hz) EEG bands over left and, to a greater extent, over the right posterior recording areas. These effects during hypnotic analgesia were paralleled by significant reductions in sympathetic activity. Since pain reductions, during hypnotic analgesia, were significantly paralleled by amplitude reductions within the total EEG spectral band in the right hemisphere, it was deduced that the inhibition of the right hemisphere plays an important role in the relief of pain. This deduction is in agreement with the idea that the right hemisphere activity modulates sustained attention and negative emotional state (Tucker & Williamson, 1984; Pribram & McGuinness, 1992).

In a more recent study (De Pascalis et al., 1999), the effects of different hypnotic analgesia suggestions on pain reduction and concurrent changes on cognitive and physiological responses were evaluated. Aim of this study was to validate and extend Zachariae and Bjerring (1994) findings and to provide an answer to the hypothesis that, independently from the strategy used, hypnotic analgesia is the product of a single phenomenon in the brain and a successful hypnotic suggestion is one of the possible keys to have access to it. Somatosensory event-related potential (SERP) and skin conductance response (SCR) changes during hypnotic suggestions of Deep Relaxation (suggestion that relaxed body would not feel any pain), Dissociated Imagery (suggestion to divert attention out of the body and to imagine oneself floating out of the body and 'up in the air'), Focused Analgesia (suggestion to focus on sensation in the stimulated arm and to experience as if a glove was covering the hand and the wrist), and Placebo (subjects had their hand dampened with a colored mixture of water and alcohol that was presented as a tipical anesthetic), as compared to a Waking baseline condition (no suggestion to reduce pain), were evaluated. SERPs were recorded from frontal, temporal, central, and parietal scalp sites. 10 high, 9 mid, and 10 low hypnotizable right-handed women participated in the experiment. The following measures were obtained: (a) pain and distress tolerance ratings; (b) sensory and pain thresholds to biphasic electrical stimulation delivered to the right wrist; (c) N280 and P400 peak amplitudes of SERPs to

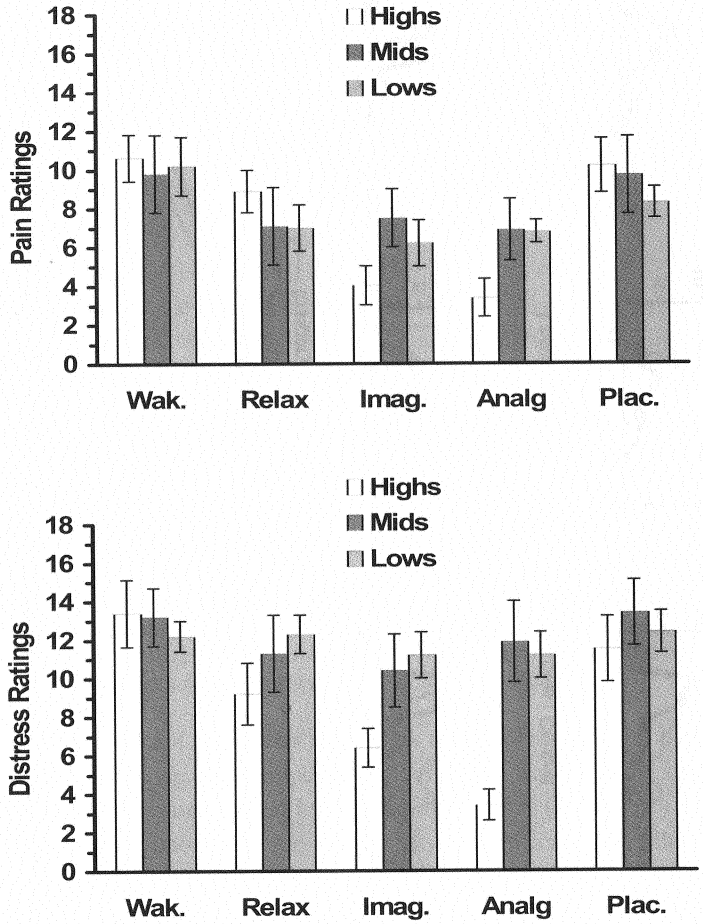

Figure 1. Mean Sensory Pain (top side) and Distress Ratings (bottom side) with standard errors during electric pulse stimulation in 10 highly, 9 moderately and 10 low hypnotizable subjects (Highs, Mids, and Lows). Measures obtained for target pulse onset during waking conditions of Waking (Wak.), Placebo (Plac.) and during hypnosis conditions of Relaxation (Relax), Dissociated Imagery (Imag.), Focused Analgesia (Analg).

painful target stimuli delivered using an odd-ball paradigm; (d) number of evoked SCRs and phasic electrodermal orienting responses (ORs); (e) phasic heart rate changes; (f) reaction time and number of omitted responses. High hypnotizable subjects exhibited significantly greater pain intensity reductions than did mid and low hypnotizables during Dissociated Imagery and Focused Analgesia while, in the other conditions, there were no pain differences between groups (see Fig. 1). High, mid, and low

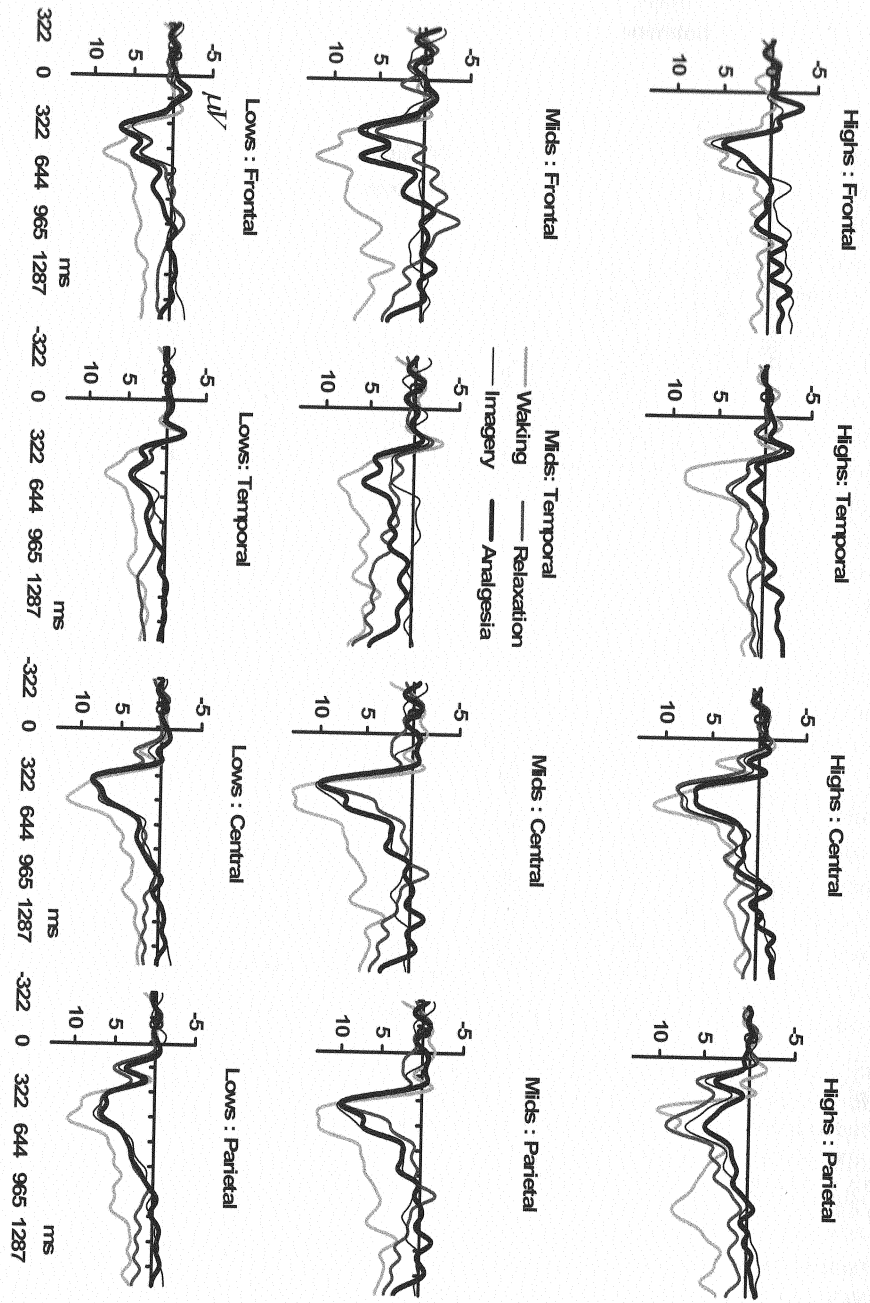

Figure 2. Somatosensory event-related potentials (ERPs) to electrical painful target stimuli averaged across left and right hemispheres for frontal, temporal, central, and parietal sites during Waking, and hypnotic analgesia suggestions of Relaxation, Dissociated Imagery, and Focused Analgesia. SERPs obtained in 10 highly, 9 moderately and 10 low hypnotizable subjects (Highs, Mids, and Lows).

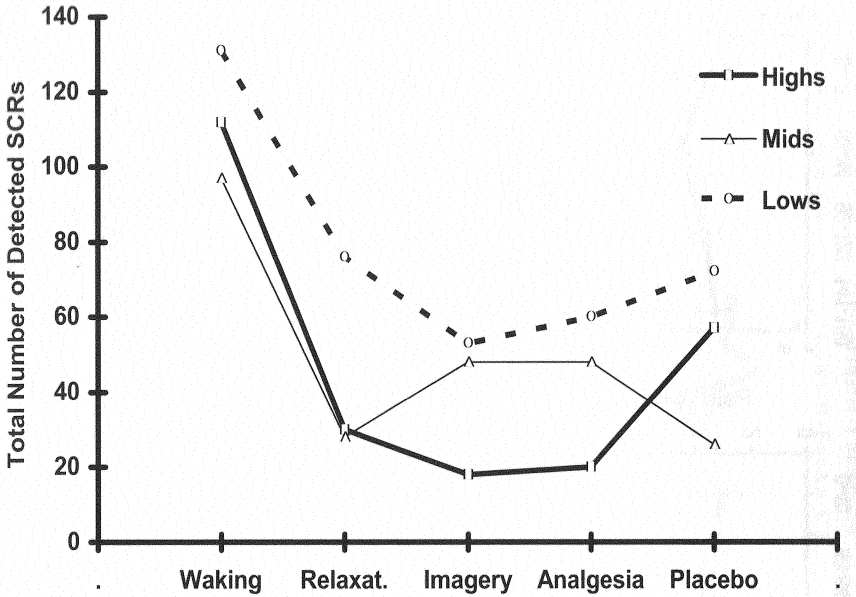

Figure 3: Total number of detected skin conductance responses (SCRs) to painful electric stimulation in high, mid, and low hypnotizable subjects (Highs, Mids, and Lows) during Waking, waking Placebo, and during hypnotic analgesia suggestions of Deep Relaxation, Dissociated Imagery, and Focused Analgesia.

groups showed significant reductions in P3 peak amplitudes across all hypnosis conditions compared to Waking. The temporal cortical region was the most sensitive in differentiating SERP responses among hypnotizability groups (see Fig. 2). On this recording area the highly hypnotizable subjects displayed significantly smaller P3 and greater N2 peaks during Focused Analgesia than did the other hypnotizable groups. The condition of Focused Analgesia, in highly hypnotizable subjects, was the most effective in producing the greatest reductions in subjective ratings of pain and distress intensities and this effect was accompained by more significant task-related changes in P3 and N2 peaks on temporal sites. This condition also displayed higher pain thresholds and faster RTs that were paralleled by a smaller number of phasic ORs (see Fig. 3). Dissociated Imagery and to a less extent Deep Relaxation conditions also displayed reductions in distress levels and significant task-related changes in N2 and P3 peaks and SCRs, but these changes were not paralleled by shorter RTs and were less pronounced than that observed for Focused Analgesia. These findings confirmed those previously reported by Zachariae and Bjerring (1994) and suggested that different processes at cortical level may be operating among Focused Analgesia, Dissociated Imagery and Deep Relaxation conditions. Common effect among conditions appears to be an enhancement of inhibitory processing. But, in terms of behavioral and physiological measu-

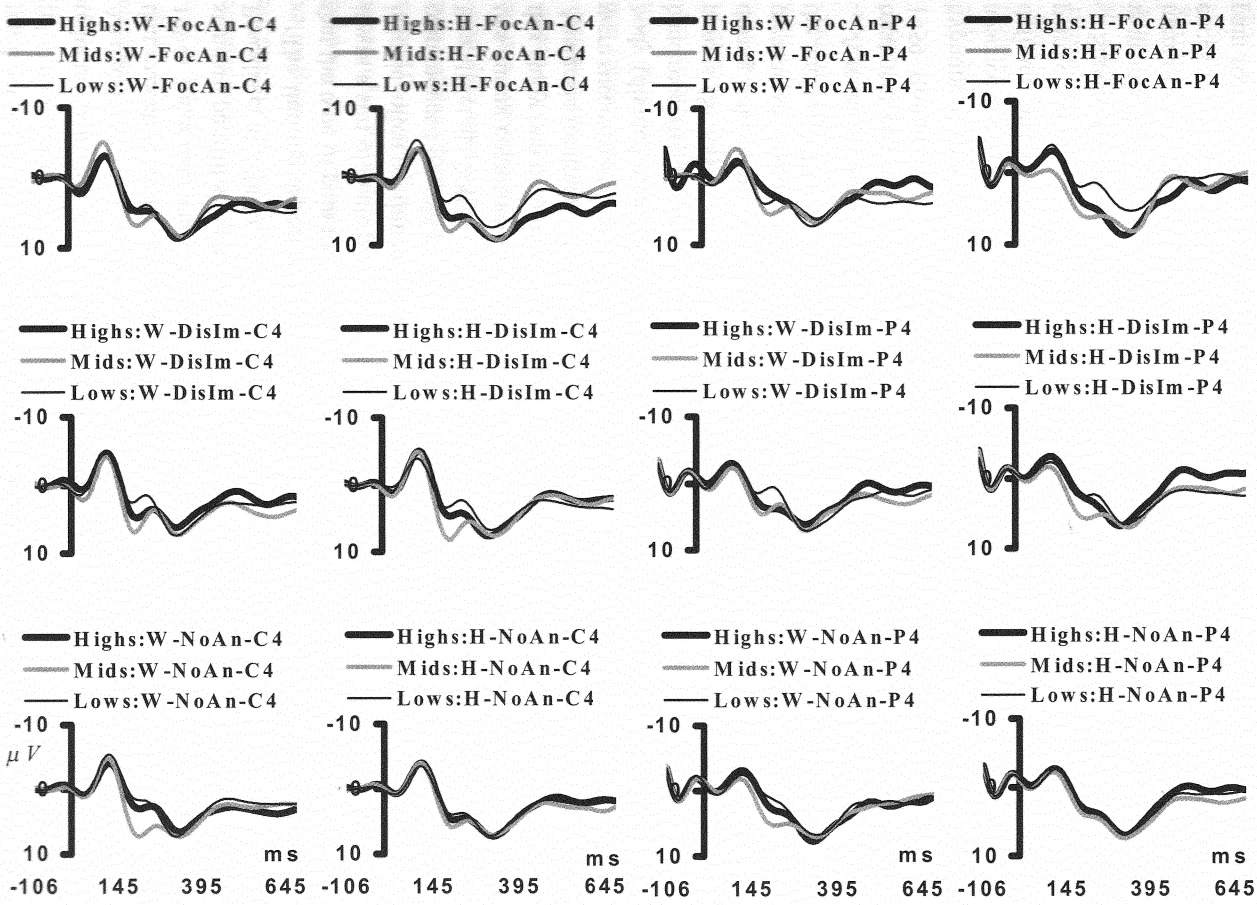

Figure 5. Event-related potentials (ERPs) to target tones in high, mid, and low hypnotizable subjects (Highs, Mids, and Lows) during Tonic Cold Bottle Test. ERPs are displayed for C4 and P4 scalp sites during waking (W) and hypnosis (H) for No-Analgesia (NoAn), Dissociated Imagery (DisIm), and Focused Analgesia (FocAn) treatments.

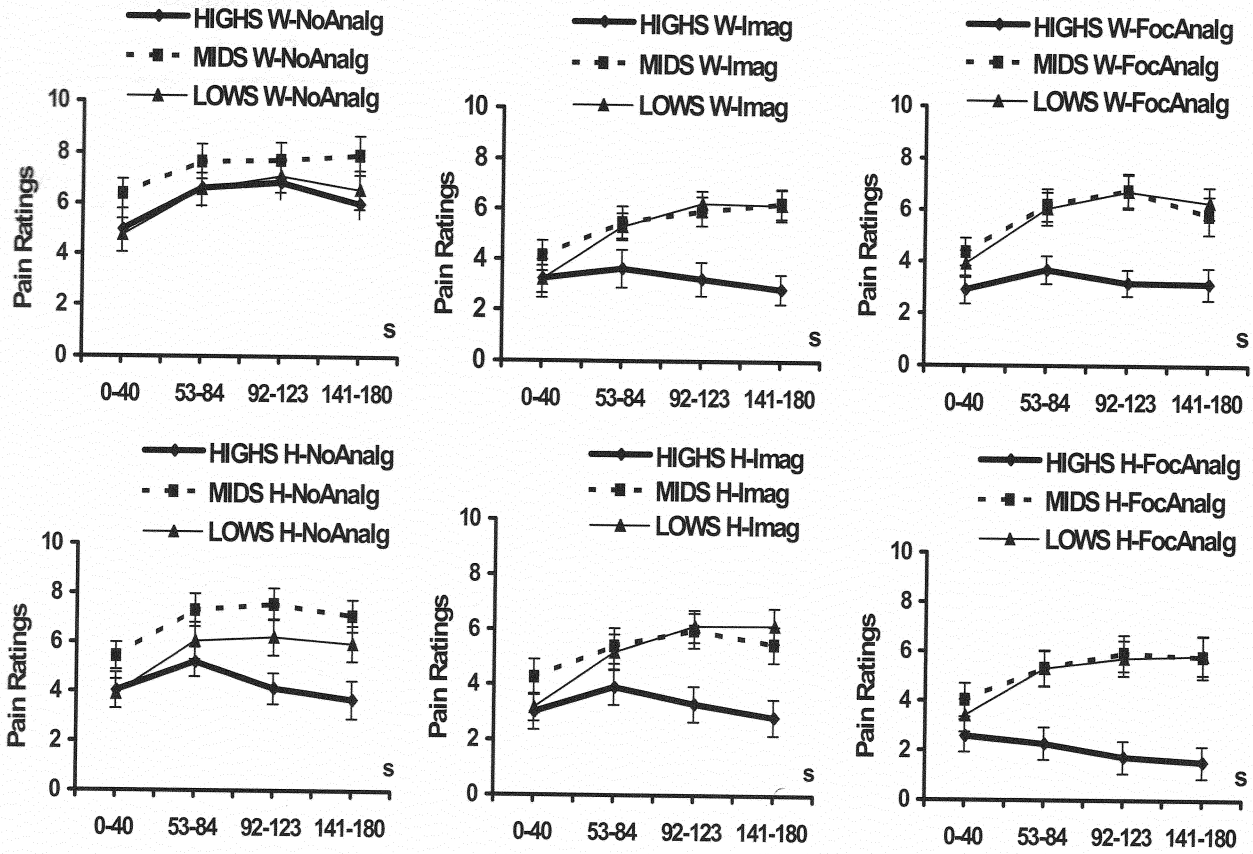

Figure 4. Mean Sensory Pain and Distress Ratings with standard errors during Tonic Cold Bottle Test in 10 highly, 10 moderately and 10 low hypnotizable subjects (Highs, Mids, and Lows). Averaged measures are reported for time intervals of 0-40, 53-84, 92-123, and 141-180 sec during conditions of waking (W) and hypnosis (H) for No-Analgesia (NoAnalg), Dissociated Imagery (Imag), and Focused Analgesia FocAnalg).

res, Focused Analgesia, which requires to focus attention on the hand receiving painful stimulations and to produce an obstructive hallucination, was the most effective in reducing pain. Since the suggestion to produce an obstructive hallucination (Focused Analgesia) seemed to require less processing capacity in its operation, it was advanced that executive initiative and effort were less involved during this treatment (Bowers, 1994). Later data analyses of SERPs components and autonomic responses elicited by standard painful stimuli, confirmed previous findings obtained with target stimuli (De Pascalis et al., 2001).

The limiting aspect of our above mentioned hypnosis-pain studies lies in the fact that the type of pain in these experiments was quite different from clinical pain, since it was a sort of acute phasic experimental pain caused by electric pulses. Clinical pain is tonic, often continuous and recurrent. Quite similar to clinical pain is a type of tonic pain induced by cold pressor test (Chen et al., 1989). Consequently, our last study (De Pascalis et al., 2002) was designed to evaluate changes of brain and autonomic activities during cognitive-attentional modulation of tonic pain in waking and hypnosis. In this study two pain reduction strategies were used: Dissociated Imagery and Focused Analgesia treatments. These were chosen since both strategies, in our previous study, were successful in pain relief. Dissociated Imagery requires to divert attention from the body (divided attention) while Focused Analgesia requires to focus attention on the hand receiving painful stimulation (focused attention). ERPs and skin conductance responses (SCRs) to target stimuli were elicited using an auditory odd-ball paradigm during cold painful tonic stimulation. In this study, the hypothesis of an inhibitory mechanism responsible for hypnotic analgesia was also tested with P3 component of the auditory ERPs.

A main issue addressed in this investigation concerns the importance of defining a treatment context in waking state as hypnotic. Some studies (Weitzenhoffer et al., 1959; Rothmar & Bowers, 1982) reported that simply defining a waking treatment as hypnotic (hypnotic context) may activate a person's potential for hypnosis even in absence of a hypnotic induction (Spanos et al., 1984). If this assumption is correct, then defining that would otherwise be a nonhypnotic treatment as hypnotic should yield pain reductions similar to those found with hypnotic analgesia.

Shortly, the aims of the present investigation were to verify that: (a) pain reduction is mainly the effect of an inhibitory processing that is reflected on P3 peak amplitude increases to target stimuli of a secondary auditory odd-ball task; (b) individual differences in hypnotic susceptibility reliably accounts for individual differences in tonic pain reduction; (c) hypnotic context in absence of hypnotic induction is sufficient to induce analgesic effects of cognitive-attentional treatments and, consequently, to produce ERPs and autonomic changes similar to those obtained with the same treatments in hypnosis condition.

Pain perception, reaction time (RT), event-related potentials (ERPs), and skin conductance responses (SCRs) to auditory odd-ball stimuli were examined while right-

handed women were holding a pre-chilled (-10-12°C) bottle on the left hand (Tonic Cold Bottle Test). Ten high, 10 mid, and 10 low hypnotizable subjects participated in the experiment. Subjects received suggestions of Dissociated Imagery and Focused Analgesia during waking hypnotic context and hypnosis conditions. A No-Analgesia condition served as a control. The following measures were obtained for target stimuli: (1) pain intensity (rated for each target stimulus); (2) reaction time; (3) P3 (322 ± 23.1 ms) ERP peak amplitudes to target stimuli; (4) prestimulus skin conductance levels (SCLs) as indices of tonic arousal existing at each stimulus presentation, and the subsequent skin conductance responses (SCRs) as indices of the phasic orienting reflex elicited by each stimulus. High hypnotizables, for Dissociated Imagery and Focused Analgesia, in both hypnotic context and hypnosis condition, displayed significant reductions in pain ratings. However, the treatment of Focused Analgesia in hypnosis condition produced the most pronounced reduction of pain. These findings suggest that Focused Analgesia in hypnosis was more effective for pain reduction than only defining a waking condition as a hypnotic one (see Fig.4). Thus, hypnotizability can be regarded as necessary and sufficient, while hypnosis per se can be considered as necessary but not sufficient, for pain reduction. Moreover, there were significant correlation coefficients between hypnotic ability scores (as measured by SHSS:C scale) and pain reduction scores (obtained by subtracting pain ratings of each treatment from those obtained in baseline no-treatment condition). These correlation coefficients, calculated for Dissociated Imagery and Focused Analgesia in waking hypnotic-context conditions, were both positive and significant ($r=0.37$, $p=0.040$ and $r=0.45$, $p=0.013$, respectively for Dissociated Imagery and Focused Analgesia). Similar relationships were found for treatments in hypnosis ($r=0.38$, $p=0.039$ and $r=0.42$, $p=0.022$ respectively for Dissociated Imagery and Focused Analgesia). These findings clearly indicated that there were similar effects of treatments in waking hypnotic context and in hypnosis.

Reaction time to auditory targets detection was affected by treatments across hypnotizability groups in waking and hypnosis conditions. In waking hypnotic-context high hypnotizable persons displayed longer RTs during both pain treatments as compared to a control baseline. This difference was interpreted as reflecting the greater cognitive effort required, in waking condition, for generating a mental strategy of coping with pain. In hypnosis condition, on the other hand, high and mid hypnotizable subjects displayed longer RTs during Dissociated Imagery and shorter RTs during Focused Analgesia as compared with No-Analgesia treatment. This effect was particularly pronounced in high hypnotizable subjects, and was explained assuming that Focused Analgesia in hypnosis requires a lower cognitive effort than Dissociated Imagery for pain reduction. The reduction of the effort release more processing capacity available for auditory odd-ball task. This interpretation is in agreement with predictions derived from dissociated control model (Bowers, 1990, 1994). This model predicts that the more pain is effectively reduced by dissociated control, the more high-level cognitive

resources remain available for further information processings.

In terms of P3 peak to auditory targets, high hypnotizable persons with respect to mid and low hypnotizable ones, also reported significant P3 peak increases across right-central and parietal cortical regions during Focused Analgesia in hypnosis (see Fig.5). These P300 findings have evidenced that in high hypnotizable subjects the effect of a treatment in waking hypnotic-context condition is different from that obtained with the same treatment in hypnosis. In particular, the grater P3 peak detected during Focused Analgesia in high hypnotizable subjects during hypnosis can be seen as indicating that during this treatment there was more available processing capacity (or less effort reactivity) for the secondary task than during treatments in waking conditions.

Results obtained with electrodermal responses to tone targets appear in line with predictions derived from the above mentioned information processing theories. Findings obtained with SCRs evidenced that tone target detections, in high hypnotizable subjects, during Focused Analgesia in hypnosis, produced more pronounced phasic orienting responses than stimuli did in mid and low hypnotizable subjects. In sum, differences on RTs, P300 peak amplitudes and SCRs found between treatments in hypnosis and waking hypnotic-context conditions bring us to maintain that in hypnosis there is evidently more than simply defining the situation as hypnotic.

Conclusion

Findings from our reviewed studies suggest that hypnotizability and obstructive hallucination are modulating factors of analgesic effect in hypnosis. Obstructive hallucination in hypnosis has resulted the most effective treatment in pain reduction. In terms of P3 peak of the ERP and autonomic activity, high hypnotizable persons, using obstructive hallucination of painful stimuli, disclosed a pronounced inhibitory effect over frontal, central and parietal cortical regions. Findings of our reviewed studies are suggesting that obstructive hallucination is the product of the operation of fronto-central and posterior cortical systems which modulate mental imagery and lead the creation of a new dominant schema for consciousness (Chapman and Nakamura, 1998).

More recent findings from our laboratory also suggest that obstructive hallucination of tonic painful stimulation may prime dissociated control in hypnosis, by requiring a lower cognitive effort for pain reduction. This interpretation is in line with predictions derived from dissociated control model (Bowers, 1990, 1994) suggesting that the more pain is effectively reduced by dissociated control, the more high-level cognitive resources remain available for further information processings.

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