Hypnotic Analgesia Affects the Processing of Painful Stimuli

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This experiment explored the effects of hypnotic analgesia on painful stimuli in high and low susceptible participants (N = 33). Behavioural (target detection; RTs), subjective (pain ratings) and electrophysiological (SERP) responses of high and low susceptible participants were assessed during control, standard-hypnosis and hypnotic-analgesia conditions. The behavioural and subjective data showed that suggestion of hypnotic analgesia modulated the processing of painful stimuli, particularly in high susceptible participants. In contrast there were no significant changes in electrophysiological responses to these stimuli. Results in high susceptible participants demonstrate that hypnotic analgesia provides an important strategy for modulating experimentally induced pain. They also suggest that different brain mechanisms are involved in the processing of painful stimuli under hypnotic analgesia and attentional distraction instructions and support previous research findings that the differentiation of behavioural, subjective and electrophysiological responses may be a result of a dissociation between the processing of sensory information and the cognitive evaluation of that information.
Hypnosis as a clinical intervention has increased in the treatment of acute and chronic pain. Reports of the use of hypnotic analgesia in medicine include; the reduction of chronic pain in cancer patients (Hilgard & LeBaron, 1984; Spiegel & Bloom, 1983) and procedures involving limb amputation, mastectomy, Caesarean section and appendectomy (Waxman, 1989). It has been used in the syringomyelia related pain (Jack, 1999) and to reduce pain and distress experienced during wound debridement (Patterson, Everett, Burns, & Marvin, 1992). It has also been shown to be effective in reducing acute experimentally induced pain (Friederich et al., 2001; Halliday & Mason, 1964; Hilgard & Hilgard, 1983; Miltner, Braun, & Revenstorf, 1992).

There is little dispute that hypnotic analgesia has a beneficial effect in pain treatment, but the underlying mechanisms remain a source of debate. Proponents of the socio-cognitive viewpoint suggest hypnotic analgesia involves conscious strategies to divert attention away from the pain, and that behaviour associated with hypnotic analgesia can be attributed to normal processes such as compliance and suggestibility (Barber, 1969; Wagstaff, 1986, 1998; Spanos, 1986, 1991). In contrast, the state theory viewpoint (Hilgard, 1986; Spiegel, 1994) has its origins in Hilgard’s hierarchical model of cognitive control involving an executive controller that monitors and activates subsystems of control; the executive controller is bypassed and subsystems of control activated directly. Thus, pain perception is modulated without involvement of conscious executive function, a process known as dissociated control (Bowers, 1991; Hilgard & Hilgard, 1983; Miller & Bowers, 1993).

In line both with Hilgard’s (1986) model of cognitive control and Gruzelier’s (1998) model of frontolimbic inhibition, hypnotic analgesia has been viewed as an unconscious but active inhibitory process of sensory information (Crawford, Brown, & Moon, 1993; Crawford et al., 1996). See also Croft, Williams, Haenschel, and Gruzelier (2002). In support of this view, regional cerebral blood flow (rCBF) studies have reported neural activity in frontal and somatosensory cortices associated with hypnotic analgesia (Crawford, Gur, Skolnick, Gur, & Benson, 1993). Data from somatosensory event-related potential (ERP) studies also support this position: A significant decrease in P100 and P300 (Spiegel, Bierre, & Rootenberg 1989) and P200 and P300 amplitude (Crawford et al., 1996) was reported in high susceptibles during hypnotic analgesia. Similar evidence been reported by Arendt-Nielsen, Zacharie, and Bjerring (1990), Zacharie and Bjerring (1994) and Danziger et al. (1998). In contrast, neither Halliday and Mason (1964) nor Meier, Klucken, Soyka, and Bromm (1993) found evidence of any significant reductions in
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One interpretation of ERP component modulation is that it is a result of changes in attention. Miltner, Johnson, Braun, and Larbig (1989) have demonstrated a clear relationship between attention and LEP amplitude, and higher pain ratings have been reported when participants attend to pain (Hutt, 1996; Janssen & Arntz, 1996). However in many of the above studies the participants’ attention was diverted away from the pain. Arendt-Nielsen et al. (1990) instructed participants to “ignore everything except the pleasant and relaxed feelings,” and when instructed to attend to a source of pain during hypnotic analgesia experimental instructions were different. It is possible then that a change in attention and variable experimental instructions can explain the modulation of late ERPs. Indeed when compared to a control task, only an attention distraction task and not hypnotic analgesia produced an amplitude reduction in late evoked potentials, suggesting separate brain mechanisms for hypnotic analgesia and attentional distraction during pain processing (Friederich et al., 2001).

The present study was an evaluation of hypnotic analgesia in conditions that had identical painful stimuli but where differences in attentional demands were kept to a minimum. The study was designed to have the same attentional focus during control, standard hypnosis and hypnotic-analgesia conditions. This allowed a comparison of subjective (pain ratings), behavioural (omission errors and RTs) and psychophysiological (ERP) data in high and low susceptible participants. It was hypothesized that hypnotic analgesia would result in lower pain report, higher omission errors (misses) and slower RTs in high participants. It was further hypothesized that if, as suggested by Friederich et al. (2001), hypnotic analgesia and attentional distraction involves separate cognitive processes then there would be no difference in the late ERP amplitudes of the two participant groups during the different ERP conditions.

**METHOD**

**Participants**

Thirty-three right-handed participants (17 male, 16 female) aged 17–37 (Mean = 22.03, SD = 3.4) were assessed using the Harvard Group Scale of Hypnotic Susceptibility, Form A (Shor & Orne, 1962). These participants were classified as high susceptibles (Harvard scores 8–12; N = 17) and low susceptibles (Harvard scores 0–4; N = 16). The study was approved by the Riverside Research Ethics Committee and informed consent was given by all participants.
Design
A 2*3 mixed design was used with hypnotizability (high and low) as the between subject variable, and condition (control, standard hypnosis and hypnotic analgesia) as the within subject variable.

Apparatus and Recording

Pain Stimulation Pain related stimuli were administered to the index finger of the right hand using a Digitimer Constant Current Stimulator, model DS7A. The index finger was prepared by the removal of dead skin with an emery board and cleaned with an alcohol swab. Cathode and anode bands were placed on the proximal and middle phalanx respectively. The stimuli comprised single 1.6 millisecond duration square wave electrical pulses (rise/fall time of 20 μsec), with a one second inter-stimulus interval. There were two types of pain stimuli: single pulse (Standard) and triple pulse (Target). Each condition comprised 550 randomly presented stimuli, 20% of which were target. To remove habituation effects the first 50 trials of each condition were precluded from analysis.

EEG Recording EEG was recorded from 9 tin surface electrodes (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4) referenced to the left ear, and amplified online using a Neuroscan data acquisition system (Scan version 4.00 and SynAmps amplifiers). Ground was positioned midway between Fz and Fpz. EOG electrodes were positioned above and below the right eye, and lateral to both eyes. Impedances were kept below 20 kohms. Bandpass filters were set to 0.01 Hz and 100 Hz with a sampling rate of 500 samples/second. EEG data were visually inspected for artefact and then processed offline: A common average reference was calculated and high and low bandpass filters (24 Hz/octave) were set at 30 Hz and 0.01 Hz respectively. Epochs (-100 ms pre-stimulus onset to 924 ms post stimulus) were extracted for analysis and baseline corrected. An artifact rejection algorithm was then used to remove any potentials outside the range -70μV to +70μV.

Procedure
All participants were tested individually. During the first 10 to 15 minutes the experimenter developed a rapport with the participant in order to alleviate any worries or misconceptions about hypnosis. Sensory threshold
and pain tolerance levels were then assessed using an ascending method of limits. Participants were asked to rate the degree of their sensory pain on a standardized scale where 0 = no pain, 5 = moderate pain and 10 = unbearable pain.

The three conditions were presented in a random order (with hypnosis and non–hypnosis conditions counterbalanced across participants). Well-established induction procedures—that is, eye fixation, systematic muscle relaxation, counting down from 20 to 1 and a further “deepening” technique using guided imagery—were used prior to the hypnosis conditions. Identical instructions were given to the participants during all three conditions, requiring them to attend to their right index finger in order to minimize attention-related effects. They were required to press a response key with the thumb of their left hand if they detected a painful stimulus. Prior to the hypnotic–analgesia condition participants engaged in guided imagery involving them being on a warm sandy beach and that they had buried their right hand deep under the sand. They then received a suggestion that their hand would become numb and that they would lose sensation in their finger so they would be unable to detect the painful stimulus.

RESULTS

Behavioural Data

Stimulus Misses To determine whether there were any significant differences between the miss rates of the painful stimuli a 2 (Group) x 3 (Condition) mixed ANOVA was performed on the data. The analysis showed a main condition effect, $F(1.97, 61.07) = 32.88, p = 0.0001$. Post hoc tests showed a higher percentage of misses in the hypnotic–analgesia condition compared to both the control, $t(32) = 5.93, p = 0.0001$, and the standard–hypnosis conditions, $t(32) = 3.97, p = 0.0001$. There was also a higher percentage of misses in the standard–hypnosis condition compared to the control condition, $t(32) = 3.50, p = 0.001$. A significant Group x Condition interaction was also observed, $F(1.97, 61.07) = 16.56, p = 0.0001$. This showed firstly that there was no significant difference during the control condition ($p > 0.6$). Secondly, that both high and low susceptibles had a significantly more misses during hypnotic analgesia compared to control (high: $t(16) = -8.37, p = 0.0001$; low: $t(15) = -2.12, p = 0.05$), but misses were more than twice as great in high than low susceptibles during hypnotic analgesia, $t(30.57) = 4.46, p = 0.0001$. Thirdly, the difference between standard hypnosis and control was significant in both groups (high: $t(16) = -3.11, p = 0.007$; low: $t(15) = -2.44, p = 0.028$)
conditions. Fourthly, the effects of hypnotic analgesia over standard hypnosis was to double the number of misses in highs ($t(16) = -5.39, p = 0.0001$) whereas it had no effect in lows who demonstrated a non-significant decrease ($p > 0.6$). Means and standard errors are presented in Figure 1.

In summary, highs produced a twofold increase in misses during standard hypnosis with a further twofold increase during hypnotic analgesia. In contrast, low susceptibles produced a significant increase in misses during standard hypnosis but no increase was observed during hypnotic analgesia.

**Figure 1:** Miss Rates During Control, Hypnotic Analgesia and Standard Hypnosis Conditions

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**Reaction Times**  
A 2 (Group) x 3 (Condition) ANOVA was performed on the RT data. A main Condition effect ($F(1.88, 50.86) = 29.75, p = 0.0001$) and Condition x Group interaction ($F(1.88, 50.86) = 10.23, p = 0.0001$) were significant. Post hoc tests showed the following: first, no significant differences during the control condition ($p > 0.36$). Second, highs ($t(15) = 2.83, p = 0.013$), but not lows ($p > 0.42$) had slower RTs during standard hypnosis compared to control. Third, highs had even slower RTs during analgesia compared both to control ($t(13) = 6.12, p = 0.0001$) and standard hypnosis ($t(15) = 4.66, p = 0.0001$). RTs of lows were also slower during analgesia relative to control ($t(15) = 2.86, p = 0.01$) and standard hypnosis ($t(14) = 2.38, p = 0.03$) but to more modest levels compared to highs. Means and standard errors are presented in Figure 2. In summary, highs not only had significantly higher miss rates than lows during hypnotic analgesia, but when they were able to detect painful stimuli they had slower RTs.
Figure 2: Reaction Times During Control, Hypnotic Analgesia and Standard Hypnosis Conditions

Pain Ratings  A 2 (Group) x 3 (Condition) mixed ANOVA of pain ratings showed a main condition effect, $F(1.90, 59.02) = 4.13, p = 0.02$. Post hoc tests showed pain ratings in all participants were significantly lower during the hypnotic-analgesia condition compared to the control condition, $t(32) = 2.84, p = 0.008$. Figure 3 demonstrates that most of the variance in this effect was accounted for by the highs. No significant main group ($F(1, 31) = 1.46, p >0.05$) or interaction effects ($F(1.9, 59.01) = 2.09, p >0.05$) were observed but exploratory analysis showed no baseline difference ($p > 0.36$). Moreover, highs but not lows ($p > 0.3$) reported significantly lower pain levels during hypnotic analgesia relative to both the control ($t(16) = 2.76, p = 0.01$) and standard-hypnosis conditions ($t(16) = 2.57, p = 0.02$).

In summary, hypnotic analgesia resulted in lower detection rates, slower RTs and lower pain ratings to painful stimuli in highs compared to lows.

EEG Data

Analysis of the average waveform in each condition demonstrated two major components post stimulus; 140–200 ms (P200) and 250ms to 450 ms (P300). A Condition (Baseline, Standard Hypnosis, Hypnotic Analgesia) x Stimulus (Target, Standard) x Electrode (Fz, Cz, Pz) x Group (High, Low) mixed factorial ANOVA was performed on the data for each of these two components.
P200 Analysis showed a significant stimulus effect $F(1, 31) = 44.87$, $p = .0001$, $\varepsilon = .59$, there being significantly higher amplitude in the target condition (Mean = 4.71, $SE = .69$) compared to the standard condition (Mean = 1.60, $SE = .27$). There was also a significant electrode effect, $F(1.48, 46) = 20.04, p = .0001, \varepsilon = .39$, with the amplitude at both at Fz (Mean = 4.4, $SE = .8$) and CZ (Mean = 4.3, $SE = .6$) significantly higher than Pz (Mean = 0.7, $SE = .3$). The electrode x stimulus interaction was also significant $F(1.35, 41.72) = 18.64, p = .0001, \varepsilon = .38$, there being significantly greater amplitude differences between the target and standard trials at Fz and Cz compared to Pz. Importantly, all other effects were found to be non-significant: main group effect $F(1, 31) = .001, p = .97, \varepsilon = .0001$; main condition effect $F(1.92, 59.4) = .175, p = .83, \varepsilon = .006$; condition x group interaction $F(1.92, 59.4) = 1.13, p = .33, \varepsilon = .03$; condition x electrode interaction $F(2.67, 82.83) = .49, p = .66, \varepsilon = .02$; condition x electrode x group interaction $F(2.67, 82.83) = 1.80, p = .13, \varepsilon = .05$; condition x electrode x stimulus interaction $F(3.31, 102.41) = 1.95, p = .11, \varepsilon = .06$; condition x electrode x stimulus x group interaction $F(3.31, 102.41) = 1.61, p = .18, \varepsilon = .05$; electrode x group interaction $F(1.48, 46.02) = 2.18, p = .14, \varepsilon = .07$; electrode x stimulus x group interaction $F(1.35, 41.72) = 0.63, p = .48, \varepsilon = .02$.

P300 Analysis showed a significant stimulus effect $F(1, 31) = 76.70, p = .0001, \varepsilon = .71$, there being significantly higher amplitude in the target
condition (Mean = 7.41, SE = .75) compared to the standard condition (Mean = 2.84, SE = .29). There was also a significant electrode effect, $F(1.72, 53.43) = 6.29, p = .005, \varepsilon = .17$, with the amplitude at both at Fz (Mean = 5.01, SE = .8) and CZ (Mean = 6.3, SE = .65) significantly higher than Pz (Mean = 4.04, SE = .5). All other effects were found to be non significant: main group effect $F(1, 31) = .008, p = .93, \varepsilon = .0001$; main condition effect $F(1.84, 58.4) = .12, p = .87, \varepsilon = .004$; condition x group interaction $F(1.88, 58.4) = 0.1, p = .89, \varepsilon = .003$; condition x electrode interaction $F(2.87, 8.89) = 1.17, p = .33, \varepsilon = .04$; condition x electrode x group interaction $F(2.87, 88.89) = .80, p = .49, \varepsilon = .02$; condition x electrode x stimulus interaction $F(2.65, 82.12) = .73, p = .52, \varepsilon = .02$; condition x electrode x stimulus x group interaction $F(2.65, 82.12) = .42, p = .71, \varepsilon = .01$; electrode x stimulus interaction $F(1.92, 59.57) = .19, p = .82, \varepsilon = .006$; electrode x group interaction $F(1.72, 53.43) = 2.84, p = .07, \varepsilon = .08$; electrode x stimulus x group interaction $F(1.92, 59.57) = 0.73, p = .47, \varepsilon = .02$.

**DISCUSSION**

This study shows that hypnotic analgesia had a significant effect on participants’ behavioural and subjective response to painful stimuli. Target misses in high participants during standard hypnosis produced a twofold increase compared to a control condition with a further two-fold increase with hypnotic analgesia. In contrast, while there was a significant increase in misses during standard-hypnosis in lows, no comparable increase was observed during hypnotic analgesia. High participants’ reaction times for correctly identified target stimuli were significantly slower during the standard hypnosis condition compared to the control condition, and a further significant slowing occurred during hypnotic analgesia. In contrast, RTs of lows during standard hypnosis were not significantly different from the control condition but were slower during hypnotic analgesia, though to a more modest level compared to highs. Furthermore, high participants reported lower levels of pain during hypnotic analgesia relative to both the no-hypnosis baseline and standard-hypnosis condition. These data demonstrate that hypnotic analgesia is able to modulate the processing of painful stimuli, particularly in high participants, and supports previous research demonstrating the efficacy of hypnotic analgesia in reducing the feeling of painful stimuli (Friederich et al., 2001; Halliday & Mason, 1964; Hilgard & Hilgard, 1983; Miltner et al., 1992).
An examination of participants’ SERP data showed that there were no significant changes in electrophysiological responses to painful stimuli. Analysis of the P200 and P300 components showed no amplitude difference between the control, standard-hypnosis and hypnotic-analgesia conditions. Moreover, there were no interactions with high and low susceptible participants. These results support previous research showing no amplitude differences between control and hypnotic-analgesia conditions (Friederich et al., 2001; Halliday & Mason, 1964; Meier et al., 1993; Miltner et al., 1992). These data do not however support other studies that showed a significant decrease in late ERP components during hypnotic analgesia (Arendt-Nielsen et al., 1990; Danziger et al., 1998; Zacharie & Bjerring, 1994). Friederich et al. (2001) note that a possible difference between their findings and those of Arendt-Nielsen et al., and Danziger et al., may have been due to the control condition always preceding the hypnotic analgesia order resulting in a habituation confound of the ERP amplitude. In the present study the control condition always preceded the two hypnosis conditions but the latter conditions were counterbalanced so the absence of any ERP amplitude reduction in the analgesia condition cannot be explained with reference to habituation. The present data suggest a dissociation between electrophysiological and subjective and behavioural measures of painful stimulation and support previous reports of this phenomenon (Meier et al., 1993). This is further demonstrated by an analysis of EEG oscillations that showed positive correlations between anterior gamma amplitude and the pain ratings prior to hypnosis were no longer maintained in high participants during hypnosis (Croft et al., 2002).

The present data then provide support to the findings by Friederich et al. (2001) and Miltner et al. (1989), who demonstrated a relationship between attention and SERP amplitude. It was noted above that changes in participants’ attention and variable experimental instructions may explain the modulation of late ERPs in Arendt-Nielsen et al. (1990), so in this study an attempt to minimize attention-related effects participants were instructed to attend to the same stimulus in all conditions. Assuming these effects were minimal then the data show that when participants attended to painful stimuli, both high and lows decreased in their ability to detect target stimuli during both standard hypnosis and hypnotic analgesia, but had significantly slower reaction times to these stimuli when they were able to detect them. Moreover, highs reported lower levels of pain during hypnotic analgesia relative to both the control and standard-hypnosis conditions.

The current data suggest that during hypnotic analgesia different brain
mechanisms are involved in the processing of painful stimuli and support suggestions of Friederich et al. (2001) and Hilgard and Hilgard (1983) that the differentiation of subjective and ERP responses may be a result of a dissociation between the processing of sensory information and the cognitive evaluation of that information. That is, somatosensory and association cortices receive and process painful stimuli but this information is not transmitted to other neural areas involved in appropriate (cognitive and motor) pain-related behaviour. These data also provide evidence that this dissociation between sensory and cognitive processing is more manifest in high compared to low susceptible participants. Highs reported less pain and had an increase in miss rates during hypnotic analgesia whereas both high and low susceptibles had a slowing of reaction times during the analgesia condition (though the effects were much greater in highs).

The data for lows are also important as they demonstrate that factors other than hypnosis are able to affect the processing of painful stimuli in these participants. It has been suggested that relaxation is a major component of hypnotic analgesia (Evans & Paul, 1970), a view supported by EEG spectral analysis demonstrating that low susceptibles become more relaxed during the hypnotic procedure (Williams & Gruzelier, 2001). Although Miller, Barabasz, and Barabasz (1991) have proposed that relaxation is not necessary to induce hypnotic analgesia, this does not mean that it cannot play a significant role in some individuals and we suggest that the pattern of results in lows may be due to relaxation effects. Important individual differences in the ability to relax during hypnosis-based procedures have been demonstrated by the fact that while some studies have reported hypnotic suggestions to be more effective than relaxation per se (Stacher, Schuster, Bauer, Lahoda, & Schultze, 1975) others have found the two techniques to be equally effective (Houle, McGrath, Moran, & Garrett, 1988). This interpretation has important implications for the clinical use of hypnotic analgesia as it highlights the possibility that if the beliefs, expectations and attitudes of non-susceptible individuals can be modified and their susceptibility increased (cf. Spanos et al., 1983) then potentially it could be of benefit to a larger percentage of the population than is thought currently. Recognition of individual differences both in hypnotic susceptibility and ability to relax has important implications for the clinical use of hypnotic analgesia and demonstrates the potential benefits of hypnotic procedures in pain management.

A valid criticism of the present study is that attention may not have been controlled as intended. The authors acknowledge that there is an absence
of independent confirmatory evidence that attention was kept constant in the different conditions intended and any future study should incorporate a further pain irrelevant task such as that used by Friederich et al. (2001). It could also be argued the hypnotic-analgesia condition was more difficult for high participants (greater attentional demand) and resulted in compliance as suggestions were given both of analgesia and a resultant difficulty in detecting the painful stimuli. However, this viewpoint does not explain why lows also had slower reaction times during hypnotic analgesia and increased their omission rates during the standard-hypnosis condition. These findings were not expected and suggest that factors other than compliance were responsible for the effects in high participants. On the contrary we suggest that the pattern of results in high susceptibles provides important evidence in support of the dissociation of sensory and cognitive processes (Hilgard & Hilgard, 1983).

A further valid criticism of the study is that the painful stimuli (electric shocks) are polymodal (they activated both large and small peripheral fibres) and the validity of the observed effects may be dependent on this type of stimuli. That is, they could have affected central sensory processes independent of pain processes. It is important therefore that future studies should adopt more well-defined painful stimuli (e.g., laser stimulation cf. Friederich et al., 2001).

The present data suggest that hypnotic analgesia provides an important strategy for modulating experimentally induced pain. They also suggest that there is a dissociation between sensory and cognitive processes, especially in high participants and that these processes rely on different neural mechanisms.

REFERENCES


