

# Hypnotic Alteration of Somatosensory Perception

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*Effects of hypnotic alterations of perception on amplitude of somatosensory event-related potentials were studied in 10 highly hypnotizable subjects and 10 subjects with low hypnotizability. The highly hypnotizable individuals showed significant decreases in amplitude of the P<sub>100</sub> and P<sub>300</sub> waveform components during a hypnotic hallucination that blocked perception of the stimulus. When hypnosis was used to intensify attention to the stimulus, there was an increase in P<sub>100</sub> amplitude. These findings are consistent with observations that highly hypnotizable individuals can reduce or eliminate pain by using purely cognitive methods such as hypnosis. Together with data from the visual system, these results suggest a neurophysiological basis for hypnotic sensory alteration.*

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Highly hypnotizable individuals are capable of profound alterations in subjective experience, including the ability to reduce or eliminate pain, control anxiety, and produce hallucinations. Despite these intense and unusual subjective experiences, there has been little objective evidence of any accompanying neurophysiological change. If such phenomena involve more than mere subjective report, they should be reflected in altered processing of perception as measured by scalp electrodes. Cortical event-related potentials provide a useful test for studying perceptual and attentional processes in humans (1, 2). Event-related potentials are scalp EEG recordings time-locked to a series of approximately 50-100 perceptual stimuli, making it

possible to study brain electrical activity associated with perception of and neural response to the stimulus series. The amplitudes of the early components (100-200 msec after the stimulus) of event-related potentials reflect exogenous factors: the intensity of the stimulus and the process of selecting the perceptual channel that is used, such as visual versus auditory (3-5). That is, the stronger the input signal, the larger the amplitude of electrical activity approximately 100 msec after the stimulus has been presented, especially over the respective sensory-association cortex. The amplitudes of the later components (200-500 msec after the stimulus) are influenced by endogenous factors such as response to perception of the stimuli, by the degree to which the stimuli are unexpected (2, 5-9), and by the extent to which the stimuli are consciously perceived (10, 11). For example, stimuli that are rare, that require a response, or that demand conscious attention tend to produce larger positive amplitudes approximately 300 msec after the stimuli have been presented, especially at frontal (reflecting infrequency) and parietal (reflecting task relevance) recording sites (2, 12). In the present study, we examined the effects on event-related potential amplitudes of somatosensory perceptual distortion produced by hypnosis. This sensory alteration is analogous to that which is used successfully in clinical pain control techniques involving hypnosis.

Previous findings in this area have been inconsistent. Some studies (13-17) have shown reduction in the amplitude of visual or auditory event-related potentials when hypnotized subjects were instructed to attenuate perception of a stimulus or focus attention on a competing stimulus. Other studies (18-24) have failed to confirm such a relationship between hypnotic attention and amplitude of event-related potentials. There are several reasons for this disparity. The nature of the hypnotic instruction is critical to the outcome. A suggestion that a subject attenuate or diminish the apparent brightness of a stimulus requires that the subject pay attention to it. Thus, the process of following such a hypnotic instruction contradicts its content. Similarly, instructing subjects that they will not perceive anything at all may result in a startle response that increases rather than decreases the amplitude of event-related potentials if the obstruction is less than perfect (25-27). Other limitations of some studies include small sample sizes, the use of patients with severe neurological or psychiatric disorders, and semiquantitative analysis of event-related potentials. In one study

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(27), highly hypnotizable subjects were instructed to replace the stimulus with a competing image rather than reduce or eliminate it. This resulted in a general reduction of event-related potential amplitude that was statistically significant for the P<sub>300</sub> portion of the waveform throughout the scalp. In the present study we sought to demonstrate that altered event-related potentials are not specific to the visual system, but are also observed in the somatosensory system and may be a measure of altered perception induced by hypnosis.

We studied the effects of hypnotic perceptual alteration in the somatosensory system. This sensory modality was chosen because hypnosis has been shown to be an effective tool in pain reduction (28–30). Although we studied electrical stimulation that was beneath the pain threshold, the analogy to hypnotic analgesia holds: the event-related potential waveform does not differ in response to noxious versus subnoxious somatosensory stimulation (31). Our hypothesis was that when highly hypnotizable individuals experienced reduction or elimination of their perception of a somatic stimulus, they would produce event-related potentials with lower amplitudes. We predicted that this effect would be especially strong at P<sub>300</sub>, since this component of the waveform is influenced by the relevance of the stimulus and was the point of reduction most prominently observed in our study of the effects of obstructive hallucination in the visual system (27). Conversely, we sought to assess whether hypnotized subjects who were instructed to enhance attention to the somatosensory stimulus would demonstrate correspondingly increased amplitudes of event-related potentials. It was expected that these effects would be greatest in the parietal (somatosensory association) region contralateral to the stimulus.

#### METHOD

Two groups of subjects, 10 high and 10 low in hypnotizability, were selected on the basis of consistently high (8 to 12) or low (0 to 4) scores on the Harvard Group Scale of Hypnotic Susceptibility, Form A (32) and the Stanford Hypnotic Susceptibility Scale, Form C (33). These differences were confirmed with the Hypnotic Induction Profile (34). Informed consent was obtained after the nature and possible consequences of the study had been fully explained in accordance with the Stanford Human Subjects Committee guidelines. A total of 20 right-handed subjects, seven men and three women in each group, performed the experiment.

Four randomly ordered instruction conditions were used, during which subjects were given identical somatosensory stimulation while event-related potentials were recorded. In each instruction condition, subjects received 110 electrical stimuli. There were 99 single (standard) stimuli from which event-related potential recordings were drawn, mixed randomly with 11 triple (target) stimuli to which the subjects were expected to press a button (35). This was done to ensure maximal

attention to the stimuli, allowing us to monitor subjects' accuracy in identifying targets (36). The interstimulus interval was rectangularly distributed between 4.0 and 5.0 seconds. The EEG was digitally coded from seven monopolar leads (F<sub>3</sub>, F<sub>4</sub>, Cz, P<sub>3</sub>, O<sub>1</sub>, and O<sub>2</sub>) referenced to a lead linking the mastoid processes behind the left and right ears; the electrooculogram (EOG) was recorded as a bipolar channel to measure eye movement artifact.

The somatosensory stimuli consisted of biphasic pulses of 1.6-msec duration applied over the left radial nerve at the palmar surface of the wrist. Pulses were generated by means of a Grass SD-9 stimulator triggered externally by the recording PDP-11 computer. Stimulus electrodes were placed longitudinally along the radial nerve approximately 2 cm apart; the proximal lead had negative polarity. A subjective "level" of pulse intensity (voltage) was established using descending levels of stimulation until a level was reached that the subject perceived as just below threshold of discomfort. This resulted in stimulus intensities between 1.2 and 2.0 times the subject's threshold of sensation. To compensate for habituation to the stimulus, the voltage was adjusted slightly upward after each experimental condition to reestablish the subject's base level. Stimulus interelectrode skin resistance varied from subject to subject between 29 and 325 k $\Omega$  and did not change during the procedure with the use of biphasic stimuli. Mean  $\pm$  SD skin resistance for highly hypnotizable subjects was 106  $\pm$  108 k $\Omega$ ; for subjects with low hypnotizability it was 208  $\pm$  164 k $\Omega$ .

While this difference was not statistically significant, it did raise the possibility of between-group differences in stimulus intensity. This, however, was not the case. During the protocol the highly hypnotizable subjects had a slightly lower mean  $\pm$  SD threshold voltage (16.8  $\pm$  10.1 versus 16.8  $\pm$  7.7 V), but at a higher mean threshold current (168  $\pm$  82 versus 103  $\pm$  48  $\mu$ A) would be expected by virtue of lower skin resistance. Likewise, their average-run voltage was lower (19.7  $\pm$  14.7 versus 27.1  $\pm$  10.6 V), but their average-run current was higher (256  $\pm$  123 versus 172  $\pm$  70  $\mu$ A). The ratio of stimulus to threshold current averaged 1.7 for the highly hypnotizable subjects and 1.7 for the subjects with low hypnotizability. None of these differences was statistically significant.

An EEG recording helmet with Beckman silver-silver chloride recording electrodes mounted on 25-mm diameter tubes with saline-soaked tips was used for seven scalp and two mastoid sites. The EOG was recorded from two Grass gold-cup electrodes located on the lower orbital ridge and on the outer canthus of the right eye. The EEG was amplified 50,000 times and the EOG 5,000 times by means of Grass P511K amplifier with flat gain (to within  $-3$  dB) between 1 Hz and 100 Hz, except for a notch filter at 60 Hz. Incoming signals were amplified and digitally sampled at 4-msec intervals with 0.1- $\mu$ V amplitude resolution. Each recording epoch consisted of a 200-msec prestimulus baseline and an 800-msec poststimulus onset record. Ep

were sorted by stimulus type (standard stimuli versus target stimuli that required button pressing), and the target stimuli were eliminated, leaving 99 standard-stimulus epochs for further processing of event-related potentials. Epochs were then rejected for the following reasons: 1) false positives (button pressing on standard [nontarget] stimuli), 2) muscle artifact contamination, 3) outliers resulting from analog to digital conversion clipping, and 4) alpha-rhythm bursts. This process yielded a mean $\pm$ SD of  $67\pm 26$  nontarget epochs per condition for the highly hypnotizable subjects and  $70\pm 25$  per condition for the subjects with low hypnotizability. Epochs were arithmetically averaged (preserving amplitude for stimulus-locked waveform components), normalized to a 0- $\mu$ V baseline average level, smoothed using a two-pass, three-point Hanning function, and graphed.

The six standard event-related potential components ( $P_{100}$ ,  $N_{150}$ ,  $P_{200}$ ,  $N_{250}$ ,  $P_{300}$ , and  $N_{400}$ ) were maximal or minimal amplitudes occurring in intervals defined by the following process. 1) All event-related potential curves from all subjects, all experimental conditions, and all recording sites were arithmetically combined into one grand total curve. 2) The maximal and minimal amplitudes were identified. 3) The half-amplitude level between neighboring peaks (e.g., halfway in amplitude between  $N_{150}$  and  $P_{200}$ ) was established. 4) The point in time on the abscissa of this half-amplitude became the dividing boundary (e.g., between the  $N_{150}$  and the  $P_{200}$  windows). 5) Within each latency window, a maxima/minima finder was used to locate the amplitude and latency for each of the six event-related potential components for each subject in each condition. To test the experimental hypothesis, one three-way analysis of variance (ANOVA) (Group by Condition by Recording Site) was conducted for amplitude of event-related potentials at each of the six component peaks. The randomized presentation of attention conditions reduced the likelihood that unequal serial correlations would affect this analysis. Post hoc testing was conducted only when preceded by a significant overall ANOVA.

Four experimental attention conditions were presented in random order. In the normal attention condition, the subjects were instructed to press a button each time they felt the target stimulus. In the passive attention condition, the subjects were instructed to attend to the stimuli but not press the button. In the hypnotic attention condition, the subjects were first led through a hypnotic-induction exercise that involved closing the eyes and elevating the left hand in response to an instruction that it would feel "light and buoyant." These movements provided behavioral confirmation that the subjects were complying with instructions. They were then instructed to attend carefully to the stimuli, which they were told to experience as pleasant and interesting," and press the button in response to targets. In the hypnotic obstructive hallucination condition, the hypnotic-induction exercise was followed by the hypnotic suggestion that a local anes-

thetic, such as Novocain, was spreading from fingers to hand to forearm of the stimulated limb. Subjects were further instructed to make the limb cold, tingling, and numb. They were then told to press the button if they felt any of the target stimuli. The experimenter conducting the hypnosis session was blind to the subjects' hypnotizability scores to ensure that all subjects received identical instructions. This was important, since highly hypnotizable individuals are especially sensitive to interpersonal cues (37, 38).

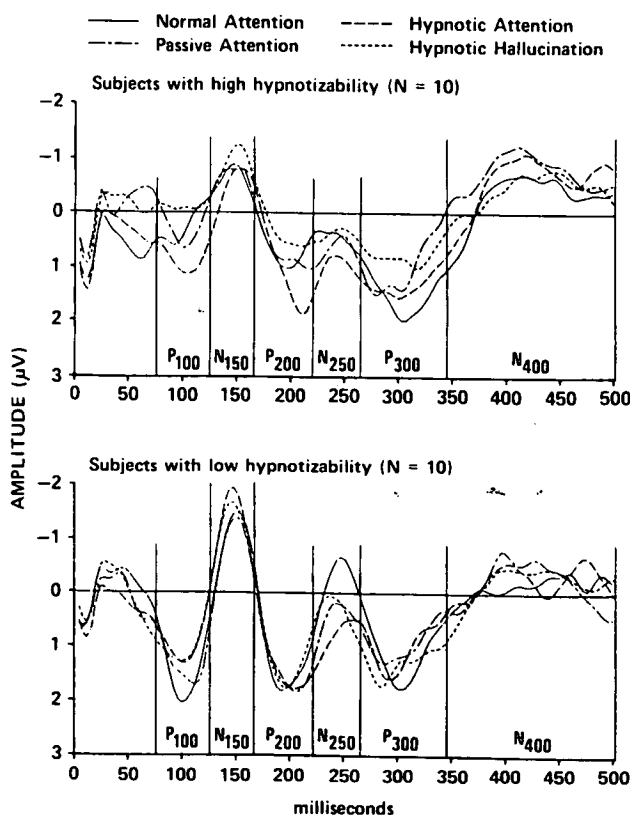
## RESULTS

The highly hypnotizable subjects were able to suppress perception of the stimulus in the hallucination condition as measured by behavioral criteria. There was a significant Group by Condition difference in button pressing ( $F=10.04$ ,  $df=2, 36$ ,  $p<0.001$ ) after elimination of the passive attention condition, which required no button pressing. The highly hypnotizable subjects pressed the button in response to 38% of the targets in the hypnotic obstructive hallucination condition, while the subjects with low hypnotizability pressed the button in response to 80% of the targets in that condition. In contrast, no significant differences were seen during the control conditions: both groups of subjects pressed the button in response to 86% or more of the targets in the normal attention and hypnotic attention conditions.

Among the highly hypnotizable subjects this perceptual suppression was accompanied by a reduction in amplitude of event-related potentials. There was a significant Group by Condition effect in the predicted direction on the amplitude of the event-related response. Figure 1 indicates, first, that the mean amplitudes of event-related potentials were lower for the subjects with high hypnotizability than for those with low hypnotizability regardless of condition. Indeed, the mean $\pm$ SD  $P_{100}$  amplitude was significantly lower among the highly hypnotizable subjects than among those with low hypnotizability ( $1.41\pm 0.93$  versus  $2.55\pm 1.82$   $\mu$ V;  $F=4.56$ ,  $df=1, 18$ ,  $p<0.04$ ). These differences were not expected, but they are consistent with a trait rather than a state conception of hypnotizability. If this interpretation is accepted, the data of interest in the present experiment are the changes from the baseline normal attention condition produced by the different experimental conditions. Therefore, a second ANOVA was performed on the difference between the normal attention condition and the other conditions at  $P_{100}$  to test the hypothesis that the highly hypnotizable subjects would show differences in amplitude among conditions which would not be seen in the subjects with low hypnotizability.

At the  $P_{100}$  portion of the waveform there was a significant Group by Condition interaction throughout the scalp ( $F=3.61$ ,  $df=2, 36$ ,  $p<0.02$ ). Of the eight mean  $P_{100}$  amplitudes (2 groups $\times$ 4 conditions), the highly hypnotizable subjects had the lowest mean $\pm$ SD

**FIGURE 1.** Mean Amplitudes of Somatosensory Event-Related Potentials in Four Conditions for Subjects With High and Low Hypnotizability



amplitudes in the hypnotic obstructive hallucination condition ( $0.80 \pm 0.71 \mu\text{V}$  versus  $1.45 \pm 0.90 \mu\text{V}$  in the normal attention condition and  $1.45 \pm 0.76 \mu\text{V}$  in the passive attention condition) (see figure 1). It appears, from subsequent matched-pairs *t* test comparisons (permitted by this ANOVA) among the highly hypnotizable subjects, that  $P_{100}$  amplitude during the hypnotic obstructive hallucination condition was lower than it was during the passive attention condition and the hypnotic attention condition. Indeed, these subjects exhibited the lowest mean  $P_{100}$  amplitudes in the hypnotic obstructive hallucination condition at all seven recording sites. During this condition,  $P_{100}$  amplitude was reduced by 45% relative to normal attention. By contrast, the subjects with low hypnotizability did not show a significant difference in amplitude among conditions.

In addition to lower  $P_{100}$  amplitudes, the highly hypnotizable subjects demonstrated lower  $P_{300}$  amplitudes during the hypnotic obstructive hallucination condition. There was a significant Group by Condition by Recording Site interaction for  $P_{300}$  amplitude ( $F=1.9$ ,  $df=18, 324$ ,  $p<0.05$ ) (see figure 1). Highly hypnotizable subjects'  $P_{300}$  amplitudes were lower during hypnotic hallucination than during both normal attention and passive attention at the right frontal, parietal, and occipital leads ( $F_4$ ,  $P_4$ , and  $O_2$ ) and were lower than those during normal attention only at  $O_1$  as well. The

only similar difference observed among the subjects with low hypnotizability was at  $O_2$ , but, contrary to task instruction, hypnotic attention amplitudes were reduced as well at  $O_2$ .

In contrast, in the highly hypnotizable subjects, a 35% increase in mean  $P_{100}$  amplitude was observed during hypnotic attention ( $1.95 \pm 0.91 \mu\text{V}$ ) as compared with that during normal attention ( $1.45 \pm 0.76 \mu\text{V}$ ). Indeed, the increase in  $P_{100}$  amplitude during hypnotic attention was greater than the change observed during both the passive attention and hypnotic hallucination conditions.

Thus, the highly hypnotizable subjects showed directional task-related changes: increased  $P_{100}$  amplitude during hypnotic attention and decreased  $P_{100}$  and  $P_{300}$  amplitudes during hypnotic hallucination, while the subjects with low hypnotizability did not. Examination of standardized interaction terms for amplitude changes among conditions indicated that the primary effect at  $P_{100}$  was the relative increase in amplitude during the hypnotic attention condition among the highly hypnotizable subjects ( $+0.48 \mu\text{V}$ ), while the predominant effect at  $P_{300}$  was the relative decrease in amplitude during the hypnotic hallucination condition ( $-0.33 \mu\text{V}$ ).

## DISCUSSION

The results of this study confirmed the hypothesis that highly hypnotizable subjects would show task-related changes in the amplitudes of their somatosensory event-related responses. The amplitude of highly hypnotizable subjects'  $P_{100}$  event-related potentials was increased during hypnotic attention and substantially reduced during hypnotic obstruction, as was the amplitude of their  $P_{300}$  potentials. Such task-related differences were not observed among the subjects with low hypnotizability. Thus, hypnosis-induced subjective changes in perception were accompanied by congruent alterations in amplitude of event-related potentials. These results confirm our previous findings in the visual system (27). However, the reduction of event-related response to the somatosensory stimulus occurred not only at  $P_{300}$ , as previously observed in the visual system, but also earlier, at  $P_{100}$ .

How can these changes in amplitude of event-related potentials be explained? This new finding may relate to the prominence of  $P_{100}$  components in somatosensory event-related potentials (36, 39, 40). Nonhypnotic suppression of somatosensory event-related potentials has been observed when there is cognitive dissonance regarding a painful stimulus that motivates the subject to suppress it (41) and during effects of analgesic drugs (42). An analogy may be drawn between signal detection theory and the early and late components of event-related potential: the early components represent more the effect of signal detection; the later components, response bias or interpretation of the signal. While  $P_{300}$  amplitude increases with better signal

ection (2, 43), it is strongly increased by stricter response bias (44)—for example, confidence in the evaluation of a detected signal as painful.

Which is involved in hypnotic analgesia? Schizophrenic patients were shown to have lower  $P_{100}$ ,  $N_{120}$ , and  $P_{200}$  amplitudes than normal subjects in response to marginally painful stimuli (45). The between-group differences in signal detection disappeared with administration of naloxone, an opiate antagonist, while response bias differences did not. However, since hypnotic analgesia is not reversed by the administration of aloxone (46, 47), the present findings seem to suggest that hypnotic alteration of pain perception operates at the level of the response bias by reducing the painfulness of the stimulus rather than the detection of the signal (48). The fact that we found effects at  $P_{100}$  as well as at  $P_{300}$  suggests that alteration in signal detection may also be involved.

$P_{300}$  amplitude is influenced by several factors: stimulus infrequency, task relevance, attention (2, 5–9), novelty (12), and conscious processing (10, 11). In the present experiment, the expectancy (frequency) of stimuli was held constant across conditions, but the other variables could have been influenced by hypnotic allucination. Hypnosis has long attracted interest because of its role in altering the boundary between conscious and unconscious experience (28, 34). The hypnotic hallucination may have made perception of the stimulus less conscious, relevant, or novel.

The strong involvement of the frontal region may be of particular importance, since it suggests that the signal generator for the hallucinated image which reduced attention to the sensory stimulus may be located frontally or in subcortical structures which project to the frontal cortex. It may also mean that the strength of the normally perceived signal is due in part to processing by the right frontal cortex, which has been shown to be involved in recognition of novel stimuli (9, 50). Reduction in right frontal processing may make the stimulus seem routine and thereby reduce its signal strength.

The difference in overall  $P_{100}$  amplitude between highly hypnotizable subjects and subjects with low hypnotizability was unexpected. We found no such differences in hypnotizability in our study of visual event-related response, but it is conceivable that these are trait differences that reflect different styles of somatosensory information processing, with highly hypnotizable subjects being more capable of turning inward imagined experience (51) and thereby suppressing somatosensory perception.

Highly hypnotizable subjects, but not those with low hypnotizability, showed changes in amplitude of event-related potentials consistent with hypnotic task demands. This study suggests that such sensory alterations are accomplished by an alteration in neuronal response to stimuli. These findings provide evidence that hypnotically induced subjective changes such as anesthesia or visual hallucinations involve alterations in perceptual processing. Further, they demonstrate a

neurophysiological difference between individuals with high and low hypnotizability. Such findings provide a basis for further exploring the neurophysiological mechanism underlying hypnotic analgesia.

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