HYPNOTIC ANALGESIA: *1. Somatosensory Event-Related Potential Changes to Noxious Stimuli and 2. Transfer Learning to Reduce Chronic Low Back Pain*[1]

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Abstract: Fifteen adults with chronic low back pain (M = 4 years), age 18 to 43 years (M = 29 years), participated. All but one were moderately to highly hypnotizable (M = 7.87; modified 11-point Stanford Hypnotic Susceptibility Scale, Form C [Weitzenhoffer & Hilgard, 1962]), and significantly reduced pain perception following hypnotic analgesia instructions during cold-pressor pain training. In Part 1, somatosensory event-related potential correlates of noxious electrical stimulation were evaluated during attend and hypnotic analgesia (HA) conditions at anterior frontal (Fpl, Fp2), midfrontal (F3, F4), central (C3, C4), and parietal (P3, P4) regions. During HA, hypothesized inhibitory processing was evidenced by enhanced N140 in the anterior frontal region and by a prestimulus positive-ongoing contingent cortical potential at Fpl only. During HA, decreased spatiotemporal perception was evidenced

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by reduced amplitudes of P200 (bilateral midfrontal and central, and left parietal) and P300 (right midfrontal and central). HA led to highly significant mean reductions in perceived sensory pain and distress. HA is an active process that requires inhibitory effort, dissociated from conscious awareness, where the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities. In Part 2, the authors document the development of self-efficacy through the successful transfer by participants of newly learned skills of experimental pain reduction to reduction of their own chronic pain. Over three experimental sessions, participants reported chronic pain reduction, increased psychological well-being, and increased sleep quality. The development of "neurosignatures of pain" can influence subsequent pain experiences (Coderre, Katz, Vaccarino, & Melzack, 1993; Melzack, 1993) and may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Therefore, hypnosis and other psychological interventions need to be introduced early as adjuncts in medical treatments for onset pain before the development of chronic pain.

Although the effectiveness of hypnosis in the relief of acute and chronic pain is well documented (e.g., Barber, 1996; Chaves, 1994; Evans, 1988; Hilgard & Hilgard, 1994; Hilgard & LeBaron, 1984), less is known about the cognitive and physiological processes involved in hypnotic analgesia. Electrophysiological and cerebral metabolic studies provide evidence for shifts in brain dynamics during hypnotically suggested analgesia in high but not low hypnotizable individuals in nonclinical populations (for reviews, see Crawford, 1994a, 1994b, in press; Crawford & Gruzelier, 1992). Yet to be addressed, however, is whether similar shifts in brain dynamics occur during hypnotic analgesia in chronic pain patients.

Highly hypnotizable persons (referred to as "highs") can partition their attentional resources more effectively than can low hypnotizable individuals (referred to as "lows"), as demonstrated at self-report, behavioral (e.g., Crawford, Brown, & Moon, 1993; Tellegen & Atkinson, 1974), and physiological (e.g., Crawford, 1994a, 1994b; Crawford, Corby, & Kopell, 1996; Crawford & Gruzelier, 1992) levels. Because of these abilities to control unwanted stimuli, such as pain, there is a moderate (around .50s) relationship between hypnotic susceptibility and pain reduction using hypnotic analgesia in normal populations (Hilgard & Hilgard, 1994). The degree to which hypnotic susceptibility plays a moderating role in hypnotic analgesia reduction in chronic pain populations continues to be debated (Chaves, 1994; Hilgard & Hilgard, 1994; Holroyd, 1996).

In adults with chronic low back pain, Part 1 of the present research reports somatosensory event-related potential correlates of noxious stimulation during attend and hypnotically suggested analgesia conditions. Part 2 evaluates the transfer of newly learned skills of experimental pain reduction to reduction of their own chronic pain.

PART 1. SOMATOSENSORY EVENT-RELATED POTENTIAL CHANGES TO NOXIOUS STIMULI

Hypnotic alterations in perceptual experiences such as suppressing auditory (e.g., Crawford et al., 1996; Kunzendorf & Boisvert, 1996; Lamas & Crawford, 1997), visual (e.g., Bányai, Mészáros, & Greguss, 1980; De Pascalis, 1994; Jasiukaitis, Nouriani, & Spiegel, 1996; Mészáros & Bányai, 1978; Spiegel, Cutcomb, Ren, & Pribram, 1985), or somatosensory input (see below) are accompanied by changes in scalp-recorded event-related potentials that also provide support for differences in inhibitory processing between lows and highs.

Scalp-recorded somatosensory event-related potentials (SERPs) have been found to be important indicators of pain processing. Several studies (Arendt-Nielsen, Zachariae, & Bjerring, 1990; Crawford, 1994b; De Pascalis, Crawford, & Marucci, 1992; Galbraith, Cooper, & London, 1972; Guerrero-Figueroa & Heath, 1964; Hernandez-Peon & Donoso, 1959; Mészáros, Bányai, & Greguss, 1980; Sharev & Tal, 1989; Spiegel, Bierre, & Rootenberg, 1989; Zachariae & Bjerring, 1994; Zachariae, Bjerring, Arendt-Nielsen, Nielsen, & Gotliebsen, 1991) show significant decreases in late SERP components in response to unpleasant cutaneous stimulation during hypnotic analgesia, whereas others (e.g., Meier, Klucken, Soyka, & Bromm, 1993) do not. Using median nerve stimulation, Mészáros et al. (1980) reported decreases in the P200 at vertex (Cz) accompanying hypnotic analgesia. De Pascalis et al. (1992) reported decreases in the N150-P200 component in the posterior region to a strongly noxious electrical stimulus. Spiegel et al. (1989) found highs showed significant P100 (F3, F4, Cz, P3, P4, O1, O2) and P300 (F4, P4, and O2) amplitude decreases when they hallucinated a local anesthetic at the wrist and hand to a mildly uncomfortable electrical stimulus.

However, thus far no hypnosis SERP study has considered the anterior frontal (prefrontal) region, although it is implicated in pain (Desmedt & Tomberg, 1989; Jones, Brown, Friston, Qi, & Frackowiak, 1991; Pribram, 1991) and differentiated from pain processes associated with the posterior regions (Head, 1920; Pribram, 1991; Price, 1988). On the other hand, anterior frontal shifts in brain dynamics during hypnotic analgesia, as measured by regional cerebral blood flow, are documented in highs but not found in lows (Crawford, Gur, Skolnick, Gur, & Benson, 1993). Posner and Petersen (1990) propose there are two major attentional systems: (a) one located in the posterior region of the brain and involved with selectively engaging and disengaging attention and (b) another located in the anterior region and involved in "attention for action" or effortful attention. For Pribram and McGuinness (1975, 1992; see also Pribram, 1991), as for Posner and Peterson, selective attention is a function of the posterior cerebral cortex, whereas effortful focused attention involves

inhibition and resistance to distraction, a function of the fronto-limbic systems (e.g., Bolster & Pribram, 1993; Pribram, 1991). As well, the supervisory attentional system (SAS; Shallice, 1988) of the anterior frontal cortex, involved in the monitoring of serial position of events and in sustaining focused attention, fits this scheme as does the hypothesized executive controller present in Hilgard's (1973, 1986) neodissociation theory of hypnotic analgesia. This executive controller, or SAS, is hypothesized to modulate "lower-level systems (other parts of the brain) by activating or inhibiting particular schemata" (Frith, 1991, p. 186).

On the basis of these earlier findings, we propose that hypnotic analgesia involves a supervisory, attentional control system of the anterior frontal cortex interacting with other cortical and subcortical regions, and that highly hypnotizable individuals can better control pain because of their more effective frontal attentional system (Crawford, 1990; Crawford, Brown, et al., 1993). Fronto-limbic operations apparently control input to the more posterior systems of the cortex (e.g., Skinner & Yingling, 1977). Specifically, the anterior frontal cortex gates the early stages of somatosensory processing as early as 28 m poststimulus (Yamaguchi & Knight, 1990; see also Desmedt, Nguyen, & Bourguet, 1983). Further evidence comes from 133Xe regional cerebral blood flow (CBF) imaging. Crawford, Gur, et al. (1993) found that highly hypnotizable persons showed a significant increase of bilateral CBF activation of the anterior frontal cortex, as well as the somatosensory cortex, during hypnotic suggested analgesia to ischemic pain. Positron emission tomography of fibromyalgia patients during hypnotic analgesia replicated these findings (B. Finer, personal communication, July 1996).

In addition to 30 painful electrical stimuli given to the left middle finger, Kropotov, Crawford, and Polyakov (1997) recorded intracranial SERPs from temporarily implanted electrodes in the anterior cingulate cortex, amygdala, temporal and parietal cortices of two obsessive-compulsive patients during attention and hypnotically suggested analgesia. In the hypnotically responsive patient, reduced pain perception was accompanied by a significant reduction of the positive SERP component within the range of 140-160 ms poststimulus recorded from the anterior cingulate cortex. This finding extends prior positron emission tomography (Casey et al., 1994; Derbyshire et al., 1994; Jones et al., 1991; Talbot et al., 1991) and functional magnetic resonance imaging (MRI) (Davis, Wood, Crawley, & Mikulis, 1995) that implicate the anterior cingulate in the processing of pain. A significant enhancement of a negative SERP component within the range of 200-260 ms was recorded from the anterior temporal cortex, and may be an indication of increased inhibitory processing. Note that this negativity occurred 50-100 ms later than changes recorded from the anterior cingulate cortex.

Present Study

In the present study, we anticipated that successful reductions in pain and distress to recurring noxious electrical stimulation during hypnotic analgesia would result in changes in SERP components between 50 and 500 ms known to reflect cognitive brain processing mechanisms: the well-known P70, N140 (also known as N150) and P260 occurring after electrical median nerve stimulation (for reviews, see Desmedt, 1979, 1988). Furthermore, we anticipated they would be differentially affected over time in anterior and posterior scalp regions. Past SERP pain research commonly reports a broad positive peak around 260 ms, but Miltner, Johnson, Braun, and Larbig (1989) clearly demonstrated that there are actually two distinct positive peaks (P200, P300), which vary in latency across participants. We likewise differentiate between these two peaks and the intervening N250 in the present study. Amplitude reductions of the P200 and P300 components reflect endogenous perceptual processes (for review, see Handwerker & Kobal, 1993), which are affected by changes in attention (e.g., Josiassen, Shagass, Roemer, Ercegovac, & Straumanis, 1982; Miltner et al., 1989) and vary with perceived pain level (Chen, Chapman, & Harkins, 1979; Miltner et al., 1989). In Miltner et al.'s study, N150 was not affected by attention under conditions where stimuli were randomized (weak vs. strong) over varying interstimulus intervals (ISIs) of 12-14 s, but has not been considered within our paradigm where active inhibitory processing can be developed to recurring strong stimuli.

We anticipated earlier time-locked effects in the anterior region due to the proposed early reallocation of attention to suppressing pain and distress from the anterior frontal region. Later SERP components would be affected due to the actualized perception of intensity (or the lack thereof during hypnotic analgesia) in midfrontal, central, and parietal regions. The present study assessed P70, N140, P200, N250, and P300 SERP components. We anticipated reductions of the positive components due to reduced pain perception during hypnotic analgesia, with increases in the negative components due to increased inhibitory processing.

To our knowledge, our laboratory is the first to investigate during hypnotic analgesia possible changes in contingent cortical potentials that occur *prior* to the repeating noxious stimuli and differentially reflect one's expectations to respond or inhibit motoric or cognitive responses. Such contingent cortical potentials may be "described as a measure reflecting the tuning of cortical excitability" and having an "influence on behavioral responses" (Birbaumer, Elbert, Canavan, & Rockstroh, 1990). They are specific to the cortical region, stimulus-response conditions, presence or absence of motoric response, and arousal or sustained cognitive activity (distraction) levels of the participant (for reviews, see Tecce, 1972; Tecce & Cattanach, 1982; Tecce & Hamilton, 1973). For instance, a maximal anterior frontal graded negativity is recorded during a period of focused, spotlighted attention (Asenbaum, Lang, Egkher,

Lindinger, & Deecke, 1992; Hansen & Hillyard, 1988). A contingent negative variation (CNV) or readiness potential (*Bereitschaftspotential*) occurs in the central region prior to the initiation of an intended movement (for review, see Birbaumer et al., 1990). A similar negative deflection is recorded from the parietal cortex prior to the anticipated cessation of an act (Donchin, Otto, Gerbrandt, & Pribram, 1973; Kornhuber & Deecke, 1965). Conversely, a slow positivity—a contingent positive variation (CPV)—occurs when participants refrain irrelevant movements, supposedly reflecting the participant's inhibitory effort (Karrer, Warren, & Ruth, 1978; Konttinen & Lyytinen, 1993).

Thus, inhibitory processing is associated with positive-going contingent variations, whereas excitatory processing is associated with negative-going contingent variations (Birbaumer et al., 1990; Tecce, 1972). If hypnotic analgesia involves active inhibitory processes at the cortical level, as proposed, we might anticipate greater positivity of prestimulus contingent cortical potentials—that is, an occurrence of a CPV—during hypnotic analgesia than attend conditions.

METHOD

Participants

Participants were 17 adults who were referred to the research project by a local physician or chiropractor because they were experiencing chronic low back pain for at least 6 months and had not obtained adequate pain relief from traditional medical approaches. They ranged in age from 19 to 43 years (M = 29 years).

They reported their chronic low back pain to have existed from 6 months to 11 years (M = 4 years). Motor vehicular accidents accounted for 41%, lifting/exercise (e.g., lifting heavy car motors or furniture) for 47%, and unknown etiology for 12%. Of the sample, 71% had damaged disks and 14% had undergone back surgery. None had been hypnotized previously. Participants were provided monetary remuneration ($30 per session) for their participation.

All 17 participants were interviewed, assessed for hypnotic susceptibility level, and trained to reduce cold-pressor pain during the first session. Two participants were dropped from further neurophysiological analyses (reasons: work made participation impossible, inadequate electroencephalograph [EEG] recording) and another was dropped from Phase 3 analyses due to the occurrence of a head concussion. The final sample for which we had SERP measures is composed of 15 right-handed adults (5 men and 10 women). Participants refrained from caffeine and pain medication use for at least 5 hours prior to the SERP experiment.

Overview of Procedure

There were three 3-hour experimental sessions, each separated by 1 week: (a) interview about pain history, administration of background

questionnaires, administration of the Stanford Hypnotic Susceptibility Scale, Form C (SHSS:C; Weitzenhoffer & Hilgard, 1962), and training to reduce cold-pressor pain; (b) assessment of somatosensory pain-related potentials; and (c) assessment of EEG during cold-pressor pain dips. Consent forms were signed at the beginning of each session. The present article reports Sessions 1 and 2, as well as psychological well-being measures for all sessions. EEG analyses during the cold-pressor pain dips recorded during Phase 3 are reported elsewhere (in preparation).

Prescreening and Baseline Questionnaires

A local physician or chiropractor provided an information sheet regarding the NIH-sponsored research project to individuals whom they thought met the criteria (significant ongoing low back pain for a minimum of 6 months with no other major neurological [including head concussions] or medical/psychiatric disorders, not presently in psychotherapy, between the ages of 18 and 45 years, and right-handed). If interested, potential participants contacted the principal investigator (HJC) by telephone. The project was thoroughly explained and discussed at that time and further screening questions were provided.

For 1 week prior to the first session, participants were requested to fill out a pain diary that logged the level of pain they experienced daily and a sleep diary that logged their night experiences. Amount and type of prescribed and over-the-counter medications taken were recorded. The pain and sleep diaries were maintained daily for the duration of the experiment and a minimum of 1 week afterward.

Session 1: Interview, Hypnotic Susceptibility Assessment, and Training to Reduce Cold-Pressor Pain

On arrival, participants had the project explained again and the consent form for Phase 1 was signed. It was emphasized this was a research project and there would be no clinical therapy addressing possible psychological issues (e.g., depression). Consent forms giving permission to obtain information concerning their chronic pain and associated disorders from their doctors were also signed.

During the first hour, each participant was interviewed about the onset of pain, possible causes, progression over time, and previous treatments. They returned pain and sleep diaries they had kept for the prior week. They filled out questionnaires including the following: the McGill Pain Questionnaire (Melzack, 1975), the Symptom Checklist 90-Revised (SCL-90-R; Derogatis, 1983), Beck Depression Inventory (Beck, 1967), Beck Hopelessness Inventory (Beck, Weissman, Lester, & Trexler, 1974), and the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). These questionnaires were completed at the beginning of subsequent sessions as well.

After a break, each participant participated in a short discussion about hypnosis and was introduced to cold-pressor dips. Each participant was administered individually an 11-item version of the SHSS:C. The dream suggestion item was deleted because the silent period could contribute to concentration on experienced pain and interrupt the hypnotic session. Following all SHSS:C suggestions and before the posthypnotic amnesia suggestion, the 20-min cold-pressor pain procedure (Hilgard & Hilgard, 1994) was administered.

Participants' left hands were placed into cold water (0-1°C) for 60-s periods in each of the following conditions: waking, hypnosis, and hypnosis with suggested analgesia (three times). They rated their sensory pain and distress on a standardized scale where 0 is *no pain*, 5 is *moderate pain*, and 10 is *unbearable pain*, but they could go higher than 10 on an open-ended scale to represent greater pain (Hilgard & Hilgard, 1994). Participants were instructed they could remove their hand at any time if the pain became too intense. One dip occurred prior to the administration of SHSS:C. Following the attend dip during hypnosis, participants were taught hypnotic analgesia techniques, including suggestions of the hand and arm being numb and insensitive, and imagining of a place such as the mountains or beach at which they would like to be. After the first two training dips, participants reported their successful strategies and these were incorporated into the suggestions for the next training dip.

The cold-pressor pain apparatus was a cooler chest filled with water and an ample supply of crushed ice so that its recorded temperature remained at approximately 0 to 1°C. A pump to circulate the water was not used, as is sometimes done, because we did not want to produce any recording artifacts during the session when EEG was recorded simultaneously. For each dip, immediately after the crushed ice was stirred by the experimenter, the participant's left hand was placed in the water for 60 s. During this time, the hand may have produced a layer of warmth around it to an unknown degree. For a discussion of these experimental issues see Hilgard (1967).

Session 2: Assessment of Somatosensory Pain-Related Potentials

On arrival, participants were informed that the study involved an evaluation of brain wave activity accompanying painful electrical stimuli that would be presented in waking and following a hypnotic induction when asked to attend and ignore the stimuli with previously learned hypnotic analgesia techniques. In addition, they were told that they would think of a pleasant trip with eyes open and eyes closed in waking and hypnosis conditions. Care was taken to develop rapport with the

participants and to put them at ease. The EEG and SERP recording procedures were described clearly to the participants, with recorded raw EEG data from a prior participant often shown to them. All questions were answered before the session. After signing the consent form, participants filled out questionnaires including the following: The McGill Pain Questionnaire, SCL-90-R, Beck Depression Inventory, Beck Hopelessness Inventory, and the State-Trait Anxiety Inventory. They returned pain and sleep diaries they had kept for the prior week.

In a quiet, moderately lighted room, participants were seated comfortably in a recliner chair with the experimenter nearby and a curtain blocking them from viewing the computer and computer operator on the other side of the room. After placement of the electrode cap on the head and the somatosensory stimulator on the finger, the participants had their sensory intensity levels titrated (see details below).

Participants served as their own controls in two A-B-A designs. First, there were waking, hypnosis, and waking conditions. In the two waking conditions before and after hypnosis, participants were asked to think of a pleasant trip once taken for counterbalanced 60-s eyes-open and eyes-closed periods, as well as asked to attend to the 30 painful stimuli; waking results are presented elsewhere (in preparation). Following the hypnotic induction, participants again thought of a pleasant trip for 60 s (not reported herein). Next, within hypnosis, participants served as their own controls in an A-B-A design in which they were presented with three sets of painful stimuli (described below) in the following conditions: attend, hypnotic analgesia, and attend. The hypnotic induction used a shortened version of SHSS:C instructions with all mention of sleep and drowsiness removed. The instructions for the attend conditions requested the participant to attend closely to their left hand and not use any techniques to reduce or eliminate the perception of pain. The hypnotic analgesia instructions were those used previously in Phase 1's cold-pressor pain training session with their previously successful strategies incorporated into the suggestions. After each set of painful stimuli, participants rated their sensory pain and distress on a standardized scale where 0 is *no pain*, 5 is *moderate pain*, and 10 is *unbearable pain*, but they could go higher than 10 on an open-ended scale to represent greater pain (Hilgard & Hilgard, 1994).

Immediately after removal of the electrodes, a short postexperimental interview was given to collect the participants' reactions to the conditions and to determine hypnotic analgesia techniques. Participants were shown their own EEG recordings. Great care was taken to ensure that they understood the experiment and that all questions were answered.

As reported in detail in Part 2, all participants were encouraged to apply their newly learned pain control techniques at their discretion to their own chronic pain during the day, before going to sleep, and if wakened during the night. To further assist, usually while standing,

participants were taught a simple 1-min eye-roll attentional focusing technique, similar to that used by Spiegel (1974) but with *no* mention of entering hypnosis, and were asked to practice their newly learned pain control techniques. Finally, participants were given pain and sleep diaries to fill out in the subsequent week.

Somatosensory stimuli. For each condition, stimuli consisted of 30 single square wave electrical pulses of 0.2 ms duration (rise/fall time of 20 microsecond), with a 3-s ISI. They were delivered to the center of the palmar surface of the distal phalange of the left hand's middle finger by a Grass S10DSCM somatosensory stimulator with an SIU8T stimulus isolation unit triggered externally by the recording 486 computer. The finger was prepared by having the participant rub the skin with an emery board, followed by the experimenter's vigorously rubbing of it with skin prep and alcohol swab.

Determination of stimulus intensity levels used during the recording periods. Analgesia research should use painful stimuli that are clearly and definitely painful (e.g., Becker, Yingling, & Fein, 1993). Sensory threshold, pain threshold, and pain tolerance levels were assessed using an ascending method of limits (Gescheider, 1985). Participants knew that it was necessary to provide stimuli that were strongly painful but bearable to assess electrophysiological responses to painful stimuli. Because some habituation to the stimuli often occurs with multiple trials, three ascending trials were given to determine when the stimulus was perceived as being strongly painful but still bearable. Participants rated their sensory pain on the same scale as used during cold-pressor pain training. A practice block of five stimuli at the chosen level was used to familiarize the participant with the sensations of finger stimulation and verify SERP recording. In this determination of stimulus intensity levels, our participants chose maximum bearable levels that were rated as being moderately to strongly painful ($M = 6.88$; $SD = 1.07$; range 4.5-8). Participants were able to tolerate these levels and did not produce finger movements or excessive ocular or myogenic artifact. Thus, we concluded that the stimuli were clearly painful for our participants.

Recording. Using a Lycra electrode cap (Electro-Cap International, Eaton, Ohio), EEG was recorded from 19 scalp sites referred to linked earlobes (A1, A2) and grounded to a location directly above the nasion. Electro-oculogram was monitored from electrodes placed inferior and lateral to the right outer canthus. Electrode impedances were kept below 3K ohm and balanced as equally as possible (less than 500 ohm difference). EEG data were collected using 20 Grass P5 series amplifiers (gain setting 10K; band pass: 0.1-100 Hz). The EEG signals were digitized at a rate of 200 samples per second for a period from 500 ms before each stimulus to 1,500 ms poststimulus. All instrumentation (including stimulus generation, EEG sampling, hard disk storage, and averaging) was

controlled by the Brain Scope program (Xie & Zheng, 1994). A digital signal from the computer activated a Grass S10DSCM somatosensory stimulator.

Data analyses. EEG analog records were first submitted to and epochs with artifacts were marked by the Brain Scope (Xie & Zheng, 1994) automatic eye movement (50 μV) and artifact rejection sequence. Subsequently, each epoch was scanned visually for verification and noting of further eye movement, muscle, or other artifacts. Those SERP epochs containing artifacts were not included in the data analyses. Presented here are amplitudes and latencies of SERP components (P70, N140, P200, N250, P300) at anterior frontal (Fp1, Fp2), midfrontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) regions during attend and hypnotic analgesia conditions following the hypnotic induction. Because there were varying prestimulus slow cortical potentials across conditions, baseline was determined by readjusting 0-ms to 0-point baseline. Because habituation may diminish SERPs (e.g., Calloway, 1973), the *two attend hypnosis conditions* (pre- and posthypnotic analgesia) were averaged and compared to the *hypnotic analgesia condition*. To assess the prestimulus slow cortical potential, the slope of the 200-ms segment just prior to stimulus onset was determined. Repeated measures 2 × 2 × 4 (Condition × Hemisphere × Region [anterior frontal, frontal, central, and parietal regions]) analyses of variance (ANOVA) were performed with Huynh-Feldt with follow-up ANOVAs to tease apart hypothesized effects in each region. Additional ANOVAs performed for the midline sites reflected similar regional findings and are available from the first author.

Results: Hypnotic Assessment and Preliminary Pain Training with Cold-Pressor Test

Hypnotic Susceptibility Scores

In general, the chronic pain participants were moderately to highly hypnotizable on the modified 11-point SHSS:C. The SHSS:C mean was 7.87 (SD = 2.27). One participant was low hypnotizable (SHSS:C score of 2), six were moderately hypnotizable (6, 7, 7, 7, 7, 7), and eight were highly hypnotizable (8, 8, 8, 9, 10, 10, 11, 11). It should be noted that this skewed distribution surprised us because we had anticipated a normal distribution of hypnotic susceptibility. As a result, we could not make hypnotic level a factor as had been originally planned.

Cold-Pressor Dip Ratings

Table 1 presents the means and standard deviations for pain and distress ratings during wake attend, hypnosis attend, and three hypnotic analgesia 60-s dips. Sensory pain and distress reports for waking and hypnosis attend dips did not differ significantly. The first hypnotic analgesia training dip led to highly significant ($p < .001$) reductions in

Table 1
Cold-Pressor Pain 60-s Dips During Waking and Hypnosis: Means (standard deviations) of Pain and Distress Reports and Percentage of Wake

Dips	Pain Report	Pain: Percentage of Wake Report	Distress Report	Distress: Percentage of Wake Report
Wake attend	9.53 (4.82)		8.73 (5.11)	
Hypnosis attend	9.13 (3.82)	101 (0.33)	7.52 (4.78)	89 (0.40)
Hypnotic analgesia 1	3.73 (4.71)	34 (0.33)	2.73 (4.68)	25 (0.32)
Hypnotic analgesia 2	1.60 (2.23)	14 (0.15)	1.00 (2.07)	7 (0.13)
Hypnotic analgesia 3	1.13 (2.39)	10 (0.17)	0.73 (2.05)	5 (0.11)

sensory pain and distress, respectively, $Fs(1, 14) = 36.18, 25.45$. In comparison to the first training dip, the second hypnotic analgesia training dip led to further significant reductions in sensory pain and distress, respectively, $F = 5.57$, $p < .005$; $F = 6.20$, $p < .05$. By the third hypnotic analgesia dip, 60% had completely eliminated all pain perception, and 80% had completely eliminated all distress perception.

SHSS:C hypnosis scores, although skewed toward higher scores, correlated significantly ($p < .01$) with amount of reduction reported during the three dips, Pain $rs = -.63, -.71, -.76$; Distress $rs = -.66, -.61, -.77$.

Experimental Conditions During Hypnosis: Reported Pain and Distress Levels

In comparison to the averaged attend conditions during hypnosis, during hypnotic analgesia to experimental pain the participants reported a highly significant reduction ($p < .001$) in both maximum sensory pain, Attend $M = 6.43$, $SD = 1.74$; Hypnotic Analgesia $M = 2.27$, $SD = 1.91$; $F(1, 14) = 42.48$, and distress, Attend $M = 5.17$, $SD = 2.34$; Hypnotic Analgesia $M = 1.13$, $SD = 1.13$; $F(1, 14) = 42.05$. During hypnotic analgesia, maximum sensory pain experienced was 39% ($SD = .31$) of Attend, and maximum distress was 24% ($SD = .29$) of Attend.

Experimental Conditions During Hypnosis: Somatosensory Event-Related Potentials

Figure 1 presents the grand averages for the SERPs for (a) attend to pain condition, which is the average of the pre- and postattends during hypnosis, and (b) hypnotically suggested analgesia. Because habituation may diminish SERPs (e.g., Calloway, 1973), the two attend hypnosis conditions (pre- and posthypnotic analgesia) were averaged and compared to the hypnotic analgesia condition. The first identifiable waveform was at P70. Table 2 presents a summary of all significant regional ANOVAs and a breakdown of the complex interactions with follow-up analyses. Because these Fs are not redundantly presented in the text, the

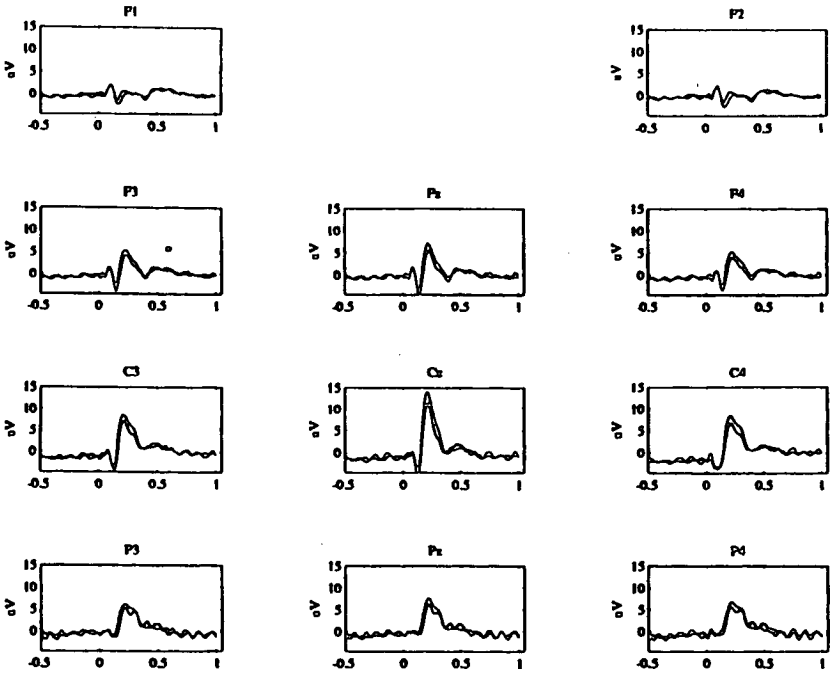

Figure 1. **Somatosensory ERP grand averages for Attend (light line; e.g., P200 higher amplitude at Cz) and Hypnotic Analgesia (dark line). Positivity is upward.**

reader is encouraged to refer to Table 2. Note that the *df*s are 1 and 14, unless noted otherwise.

Latencies did not differ significantly between conditions for P70 (*M*s: 66-77 ms), P200 (*M*s: 212-214 ms), P300 (*M*s: 303-310 ms), N140 (*M*s: 120-134 ms), and N250 (*M*s: 271-282 ms).

Mean amplitudes for P70, P200, P300, N140, and N250 components for the left and right anterior frontal, frontal, central, and parietal regions are presented in Figures 2 to 6, respectively.

Positive SERP Components: Mean Peak Amplitudes

As anticipated, during hypnotic analgesia, there were reductions in P200 and P300 amplitudes in the midfrontal, central, and parietal regions. Conditions had no significant effect on P70.

P70 amplitude. There was a significant Region × Hemisphere interaction, $F(3, 42) = 8.60$, $p < .05$. At the central region, P70 amplitude was significantly ($p < .001$) greater in the left (C3 = 1.79 μV) than right (C4 = 0.25 μV) hemisphere. Contrary to expectation, there were no significant

(text continued on p. 109)

Table 2

Summary of Significant Effects and Follow-Up Analyses of Condition × Hemisphere Analyses of Variance of Mean μV Amplitude for Each SERP Positive and Negative Components

Component	Region	Significant Effects	Significant Effects Analysis	Explanation
Positive SERP components				
P70	Central	Hemisphere $F(1, 14) = 16.81, p < .001$		C3 > C4
P200	Frontal	Condition $F(1, 14) = 4.92, p < .05$		Attend > Hypnotic Analgesia
	Central	Condition $F(1, 14) = 6.91, p < .02$		Attend > Hypnotic Analgesia
	Parietal	Condition × Hemisphere $F(1, 14) = 4.80, p < .05$	Hypnotic Analgesia: Hemisphere $F(1, 14) = 8.49, p < .02$	Hypnotic Analgesia: P3 < P4
			Attend vs. Hypnotic Analgesia Left Parietal (P3) $F(1, 14) = 3.72, p < .08$	P3: Attend > Hypnotic Analgesia
P300	Frontal	Condition $F(1, 14) = 10.87, p < .01$		Attend > Hypnotic Analgesia
		Condition × Hemisphere $F(1, 14) = 5.78, p < .05$	Attend vs. Hypnotic Analgesia Left Frontal (F3) $F(1, 14) = 4.00, p < .07$	F3: Attend > Hypnotic Analgesia
			Right Frontal (F4) $F(1, 14) = 22.94, p < .001$	F4: Attend > Hypnotic Analgesia
	Central	Condition × Hemisphere $F(1, 14) = 18.07, p < .001$	Attend vs. Hypnotic Analgesia Left Central (C3) $F(1, 14) = 1.77$, *ns*	C3: Attend = Hypnotic Analgesia
			Right Central (C4) $F(1, 14) = 23.91, p < .001$	C4: Attend > Hypnotic Analgesia

(Continued)

Table 2 Continued

Component	Region	Significant Effects	Significant Effects Analysis	Explanation
Negative SERP components				
N140	Anterior frontal	Condition $F(1, 14) = 9.04, p < .009$		Anterior frontal: Hypnotic Analgesia more negative
N250	Frontal	Condition $F(1, 14) = 11.09, p < .005$		Frontal: Hypnotic Analgesia more negative
	Central	Condition $F(1, 14) = 10.12, p < .007$		Central: Hypnotic Analgesia more negative
		Condition × Hemisphere $F(1,14) = 4.53, p = .05$	Attend vs. Hypnotic Analgesia Left Central (C3) $F(1, 14) = 4.88, p < .05$	Stronger effect in C4 than in C3 C3: Hypnotic Analgesia more negative
			Right Central (C4) $F(1, 14) = 14.75, p < .002$	C4: Hypnotic Analgesia more negative
Prestimulus contingent variation	Anterior frontal	Condition × Hemisphere $F(1, 14) = 8.34, p < .02$	Hypnotic Analgesia Hemisphere $F(1, 14) = 56.55, p < .001$	Fp1 > Fp2
			Attend Hemisphere $F(1, 14) = 1.17$, *ns*	Fp1 = Fp2

Note. SERP = somatosensory event-related potentials.

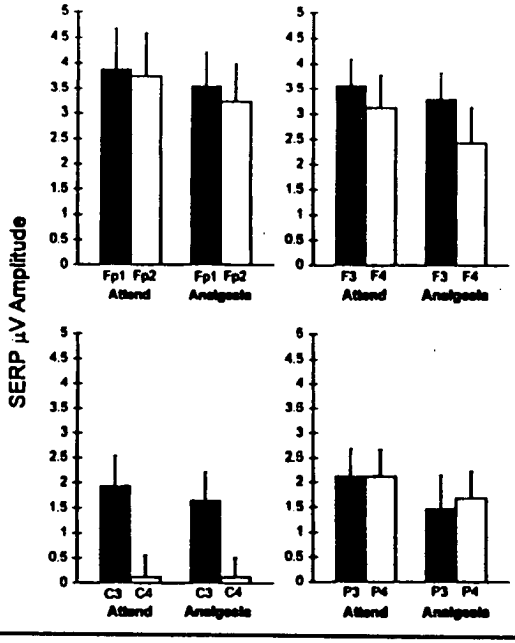

Figure 2. **P70 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

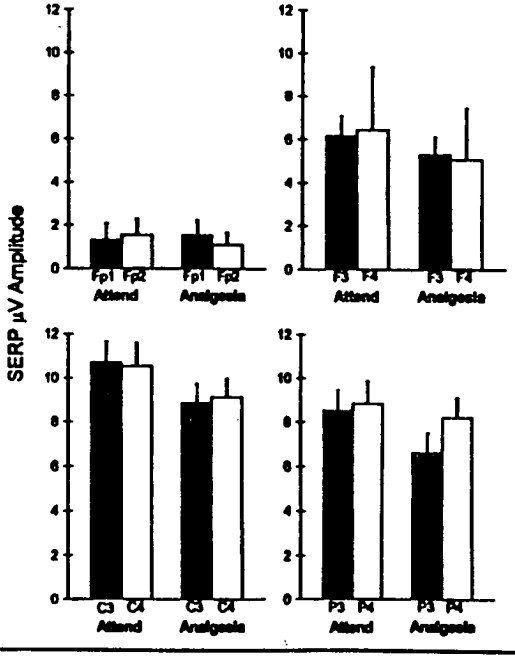

Figure 3. **P200 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

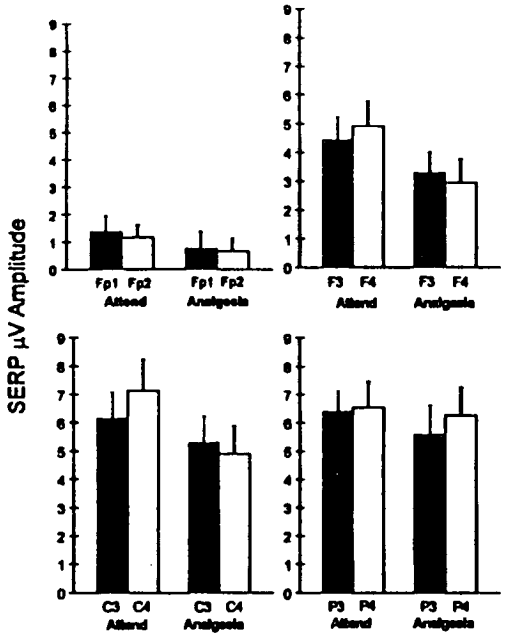

Figure 4. **P300 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

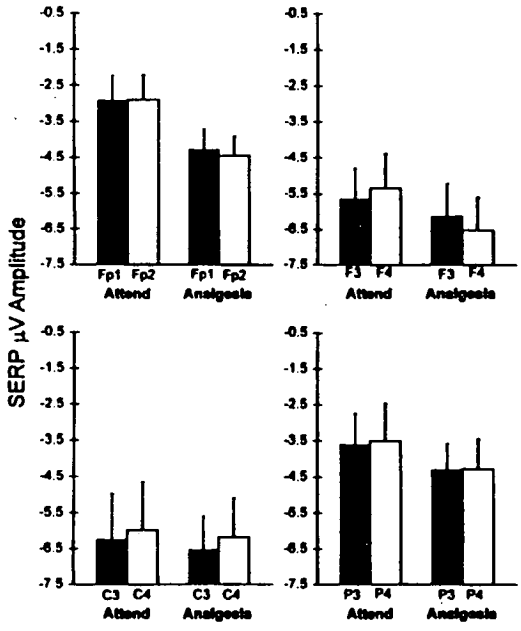

Figure 5. **N140 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

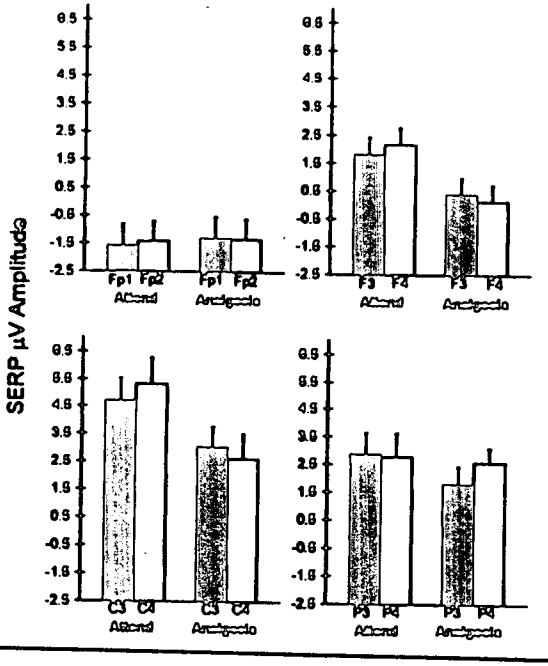

Figure 6. **N250 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

differences between Attend and Hypnotic Analgesia conditions at any measured region (see Figure 2).

P200 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 5.35$, $p < .05$. As anticipated, in comparison to Attend, during hypnotic analgesia there were significant P200 amplitude reductions at midfrontal (Attend = 6.30 µV; Hypnotic Analgesia = 5.21 µV) and central (Attend = 10.62 µV; Hypnotic Analgesia = 8.97) regions (see Figure 3). A significant Hemisphere × Condition interaction was observed at the parietal region. Although there were no significant differences between hemispheres in Attend, during hypnotic analgesia there was a substantially greater reduction in the left parietal (6.63 µV) than right parietal (8.22 µV) region.

P300 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 7.38$, $p < .05$. Significant Hemisphere × Condition interactions were observed at midfrontal and central regions. As shown in Figure 4, in both regions, significant P300 amplitude reductions during hypnotic analgesia were observed in the right but not in the left hemisphere.

Negative SERP Components: Mean Peak Amplitudes

As anticipated, during hypnotic analgesia, enhanced N140 occurred in the anterior frontal region and enhanced N250 occurred in the midfrontal and central regions.

N140 amplitude. As anticipated, at the anterior frontal (Fpl, Fp2) region, there was a highly significant greater negativity of the N140 component during hypnotic analgesia (–4.37 μV) than during attend (–2.97 μV) conditions (see Figure 5).

N250 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 5.11$, $p < .05$. There was significantly greater negativity of the N250 components at both midfrontal (Attend = 1.98; Hypnotic Analgesia = 0.25) and central (Attend = 5.02; Hypnotic Analgesia = 2.60) regions (see Figure 6). Although present in both hemispheres, these effects were significantly stronger in the right frontocentral region that was contralateral to the stimulated finger than in the left hemisphere.

Prestimulus Contingent Cortical Potentials

We examined the 200 ms prior to the stimulus to assess for negative-going (cortical activation) and positive-going (inhibitory processes) contingent cortical potentials. As can be seen in Figure 7, consistently in all regions there was little positivity or negativity during the Attend condition.

At the anterior frontal region there was a significant interaction between Condition and Hemisphere. Unlike the Attend condition in which there were no hemisphere differences and negligible positivity, during Hypnotic Analgesia there was significantly greater positivity in the left than in the right anterior frontal region.

DISCUSSION

Hypnotic analgesia had a significant effect on the somatosensory event-related potentials accompanying noxious electrical stimulation: first, in the greater negativity shown more anteriorally; second, in the reduced amplitude of the cortical response to pain shown more posteriorally; and third, in a dramatic asymmetrical prestimulus positivity recorded from the anterior frontal electrodes.

Increased Negativity Anteriorly

Increased inhibitory processing during hypnotic analgesia was indicated by a shift toward greater negativity for N140 that occurred in the anterior frontal region only, supporting the evidence obtained from regional cerebral blood flow research (Crawford, Gur, et al., 1993). The N140 component is thought to reflect the "complex reciprocal interactions between posterior and prefrontal [anterior frontal] cortex and

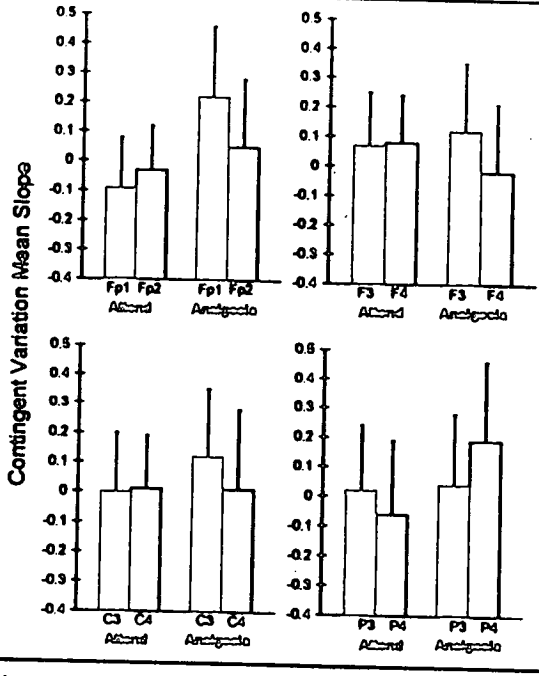

Figure 7. **Contingent variation: Mean slopes for 200 ms prestimulus for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

subcortical structures" that play "a key role in governing sequential attention processes" (Desmedt & Tomberg, 1989, p. 343). Additionally, during hypnotic analgesia, a greater N250 negativity was observed in the fronto-central region. We interpret these enhanced negative components to be indicative of active disattention during hypnotic analgesia, rather than the normally increasing spotlighted attention toward relevant incoming sensory signals.

Poststimulus Decreased Positivity

Reductions in perceived intensity of pain during hypnotic analgesia was observed (a) in the greatly reduced pain and distress reports and (b) in electrophysiological results: Reduced P200 amplitudes were observed in the midfrontal, central, and left parietal regions, and reduced P300 amplitudes were observed in the right midfrontal and central regions. These scalp-recorded positive components may reflect reduced involvement, during hypnotic analgesia, of the posterior parietal cortex whose function includes organizing sensory inputs received from the somatosensory and anterior frontal cortex to determine position of body parts (Desmedt & Tomberg, 1989).

Prestimulus Positivity

Dramatic additional evidence for the occurrence of inhibitory processing during hypnotic analgesia was obtained: A positive-going slope of the *pre*stimulus contingent cortical potential was found *only* in the left anterior frontal region. As reviewed by Birbaumer et al. (1990), maximal negativity of contingent cortical potentials occurs in the anterior frontal cortex "at the same time that prefrontal [anterior frontal] neurons critical for the solution of the task also show maximal activity" (p. 20). The contingent positive variation observed during hypnotic analgesia in the left anterior frontal region may therefore reflect a lowering of cortical activity (Birbaumer et al., 1990; Rockstroh, Müller, Wagner, Cohen, & Elbert, 1993) due to increased hyperpolarization that is inhibitory processing (Tecce, 1972; Tecce & Cattanach, 1982). The asymmetry is strong evidence against eye movement artifact as being the cause. The implications of the observed hemispheric difference cannot be evaluated fully because the present study only administered noxious stimuli to the left finger. Still, it should be noted that Basile, Rogers, Bourbon, and Papanicolaou (1994) reported magnetoencephalographically recorded CNVs to be asymmetrical in the frontal cortex, suggesting that tasks engaging the hemispheres differently "should result in asymmetric fields on the scalp" (p. 163). Furthermore, it should be noted that Gruzelier and his colleagues (for review, see Crawford & Gruzelier, 1992) suggest a decreased involvement of the right anterior region during hypnotic induction and certain hypnotic phenomena.

Tecce (1972; Tecce & Cattanach, 1982) argued that the prestimulus contingent cortical potentials are related to arousal and attention. Tecce (1972) also reviewed evidence that positivity occurs when responses have to be suppressed, thus reflecting "active inhibitory processes" (p. 76). This is born out in the work of Rockstroh et al. (1993), which has shown that depolarization in cortical dendritic trees generates a surface negativity while the positive-going waves result from a lowering of cortical excitability. Thus, "surface-negative shifts on the scalp, such as the CNV [contingent negative variation], are hypothesized to reveal enhanced cortical excitability enabling a preparatory state. . . . In contrast, slow positive shifts may result from a 'disfacilitation' in cortical neuronal networks" (Rockstroh et al., 1993, p. 236; for review, see Birbaumer et al., 1990).

Theoretical Implications

The development of hypnotic analgesia is seen as an active process, involving several brain systems, that requires inhibitory effort—although this effort may be dissociated from conscious awareness (e.g., Hilgard, 1986). We propose that during hypnotic analgesia a supervisory, attentional control system of the anterior frontal cortex participates in a

topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities (e.g., Birbaumer et al., 1990; Pribram & McGuinness, 1975, 1992; Skinner & Yingling, 1977; Yingling & Skinner, 1976). Taken together with recent research with highly hypnotizable students using the same experimental paradigm (Crawford, 1994b; Crawford et al., 1997), our SERP data suggest that the anterior frontal region deals with the active allocation of attention and disattention, whereas spatiotemporal aspects of the somatosensory perceptions involve the posterior cortical systems.

Most certainly, other inhibitory pain systems are actively interacting with the frontal attentional system, including the limbic and thalamic systems, as evidenced by recent work (B. Finer, personal communication, July 1996; Kropotov et al., 1997). Such inhibitory processing during hypnotic analgesia may extend as far as spinal cord antinociceptive mechanisms as evidenced by reductions in brief latency (Hagbarth & Finer, 1963) and R-III amplitude (Kiernan, Dane, Phillips, & Price, 1995) of spinal reflexes (for an exception, see Santarcangelo, Busse, & Carli, 1989).

Drawing from recent spatiotemporal brain electrical source analyses (Allison, McCarthy, & Wood, 1992; Bromm & Chen, 1995; Tarkka & Treede, 1993) that indicate certain SERP components (approximately between 140 to 220 ms in magnetoencephalographic studies) are generated primarily in the frontal cortex and cingulate gyrus, our study suggests that these structures are involved in hypnotic analgesia strategies. Thus, it is of particular importance that the N140 enhancement during hypnotic analgesia was observed only in the anterior frontal region (see Figure 5). How early the effect of shifts in attention allocation during hypnotic analgesia occurs and the location of such generators is yet unknown.

Other recent work using intracerebral SERP (Kropotov et al., 1997) and cerebral metabolism (Crawford, Gur, et al., 1993; B. Finer, personal communication, July 1996) support such an interpretation. Kropotov et al. (1997) suggest the involvement of the anterior cingulate cortex in both pain perception and strategies of pain control. The anterior cingulate is heavily connected with the anterior frontal cortex and is thought to be an area that organizes responses to noxious stimuli (for review, see Devinsky, Morrell, & Vogt, 1995) and possibly has an SERP dipole associated with it (Bromm & Chen, 1995).

Further research would clarify and expand our neurophysiological model of hypnotic analgesia. Stimulations to both left and right fingers, with spatiotemporal brain electrical source analyses, are needed. This can be accomplished through the use of more electrodes and re-referencing to a common average to produce accompanying isopotential maps. Research employing noninvasive functional MRI is additionally useful. We are presently carrying out such work.

PART 2. EXPERIMENTAL PAIN TRAINING TRANSFER LEARNING TO REDUCE CHRONIC LOW BACK PAIN AND DEVELOPMENT OF SELF-EFFICACY

> When the burden of cure is abrogated to the implicit magic of the technique—rather than the patient's taking an active role in his or her treatment—any initial attempt to use hypnosis would at best be unsuccessful, and at worst would precipitate an early termination of the therapeutic encounter. Treatment cannot be passive, nor can it be solely the responsibility of the therapist. The patient must learn the self control that is needed for the mastery experience of pain control. (Evans, 1988, p. 37)

Whereas 90% of low back pain will remit naturally within 12 weeks, the other 10% becomes chronic, debilitating, and costly (Nachemson, 1982). Additionally, it recurs frequently in 40-60% of those previously inflicted (Haanen, 1984, as cited in Spinhoven, 1987). The hypnotic reduction of low back pain, as well as other chronic pains, is often addressed with suggestions aimed directly at reducing sensory pain in the affected area and accompanying distress, as well as relaxing the affected area and the body as a whole (for review, see Spinhoven, 1987). However, a somewhat different approach may be taken by applying hypnotic control techniques *first* to an experimentally induced pain and subsequently transferring those techniques to chronic pain. Relying heavily on the concept of self-efficacy (Bandura, 1977; Pribram, 1963, 1971; White, 1960), Brown and Fromm (1987) advocate such a multimodal approach to chronic pain management and reduction aimed at enhancing a sense of self-efficacy. They suggest starting the training of hypnotic control techniques with an induced pain (pinching the hand) and then moving through a hierarchy of experienced pains from that which is least bothersome to that which is the target pain. Having the experience of *learning first to control experimental pain* (instead of the clinical pain that may carry "psychological baggage") enhances feelings of confidence and self-efficacy and changes belief systems that one can have personal control over debilitating chronic pain. Learning skills and understanding one's own abilities within the context of pain control is of utmost importance.

In the present study, we assessed the degree to which learning the hypnotic skills used to control experimental pain would lead to increased psychological well-being as indicated by reported reductions in chronic low back pain over the three experimental sessions. Such reports can be interpreted as the result of skill transfer from control over experimental pain to control over chronic pain. Assessment of psychological well-being included measures of depression (Beck Depression Inventory), hopelessness (Beck Hopelessness Inventory), psychological dis-

tress (SCL-90-R), and sleep quality reports. Persons who are moderately to highly responsive to hypnosis, as measured by SHSS:C, were expected to show greater transfer effects than those who were rated as low hypnotizable. Unexpectedly, our participants were, except for one low hypnotizable, all moderately to highly hypnotizable; thus, we were unable to assess the latter hypothesis.

METHOD

At the end of the first phase (described above in Part 1), the participant and researcher discussed the degree to which the experimental pain was reduced and techniques that seemed most effective. To develop self-efficacy among the participants, several actions were taken. Participants were congratulated on their newly found skills. To provide a context in which to understand why these techniques may have worked and to de-emphasize the uniqueness of the hypnotic state, an emphasis was placed on newly found uses of attentional abilities that they already used in other contexts (e.g., ignoring noisy environments, becoming deeply involved in positive experiences such as watching movies or lovemaking). All participants were encouraged to apply these techniques at their discretion during the day, before going to sleep, and if wakened during the night.

At the end of Sessions 2 (SERP measurement) and 3 (EEG correlates of cold-pressor pain), participants were taught a simple 1-min eye-roll attentional focusing technique, similar to that used by Spiegel (1974) but with *no* mention of entering hypnosis. Individuals responsive to hypnosis may not need a formal hypnotic induction to apply their learned disattentional skills to pain reduction outside the hypnosis context (Hilgard & Hilgard, 1994). Participants were told this eye-roll technique served as a cue and would help them alert and focus their attention so they could possibly reduce their own chronic pain. Participants were asked to roll up their eyes toward the ceiling, and then, while maintaining this upward stare, to slowly close their eyes. They were then asked to take a slow deep breath, relax their muscles, and use self-selected techniques (e.g., send pain reduction messages down to their back, imagine being elsewhere, relax) to assist in the suppression of their own pain. After each of three trials, participant and experimenter discussed successful techniques and other possible techniques to try, as well as how their bodies felt. Participants were encouraged to practice their newly learned pain control techniques, with or without the eye-roll attentional focusing technique, as they desired at home and work. (Several participants reported subsequently changing the cue to staring at a wall rather than rolling their eyes upward and closing them.)

RESULTS

Changes in Back Pain Subsequent to First Hypnosis Experience

The experience of hypnosis and hypnotic analgesia training for experimental cold-pressor pain contributed to major reductions in reported back pain at the end of the first session—even though no mention of back pain, or reduction thereof, had occurred during the SHSS:C hypnosis session. After the hypnosis debriefing, after sitting in the chair for 1 1/2 hours, participants were casually asked, "How does your back feel?" Subsequently, they rated their pain level on the open-ended 0-10-point scale.

In comparison to the beginning of the administration of SHSS:C, their low back pain decreased quite significantly, before SHSS:C M = 5.13, SD = 2.20; and after SHSS:C M = 1.07, SD = 1.39; $F(1, 14) = 58.93$, $p < .0001$. All participants reported low back pain prior to SHSS:C administration (range 2-8). During the post-SHSS:C interview, 53% reported complete cessation of low back pain. They expressed surprise and indicated that typically they could not sit comfortably in a chair for this length of time. As one woman said, "Normally it would be 7 to 10 if I sat this long, but now it is only 1 or 2."

Changes in Back Pain Within Experimental Setting: Attend Versus Hypnotic Analgesia

Session 3 involved EEG recordings (in preparation) during 60-s cold-pressor pain dips while participants (one did not participate in this last session) attended to or used hypnotic analgesia. Subsequent to this, while still in hypnosis, the participants were asked to sit quietly and attend to their lower back for 1 min, after which they were asked about what they had experienced, including pain and distress reports. They were then asked to apply the hypnotic analgesia techniques they had just previously used for cold-pressor pain reduction to reduce pain in their own back. For approximately 2 min, the experimenter verbally suggested use of previously reported images and counted from 1 to 10. After 3 min of silence, participants were asked to report pain and distress levels.

Use of hypnotic techniques to decrease lower back region discomfort led to highly significant ($p < .001$) reductions in both sensory pain, Attend M = 4.39, SD = 1.90; Hypnotic Analgesia M = 0.65, SD = 1.25; $F(1, 13)$ = 35.42, and distress, Attend M = 4.08, SD = 1.66; Hypnotic Analgesia M = 0.58, SD = 1.66; $F(1, 13) = 23.89$. All but one participant reported pain when concentrating on their backs. After applying their pain control techniques, 69% reduced low back sensory pain to 0 and 76% reduced distress to 0. This provides strong evidence for the successful transfer of experimental pain control techniques to chronic pain.

Low Back Pain Reductions Across the Three Experimental Sessions

Over the three experimental sessions, our participants reported significant reductions in overall low back pain ($p < .01$), as assessed by the McGill Pain Questionnaire (see Table 3). They reported their low back pain (PPI on a 0- to 10-point scale) reduced significantly ($p < .01$). There were significant reductions for sensory ($p < .02$) and evaluative ($p < .05$) components but not for the affective ($p < .09$) component.

Improvements in Psychological Well-Being Across the Three Experimental Sessions

Over the three experimental sessions, a significant improvement in the psychological well-being of the chronic pain participants occurred. Means, standard deviations, and ANOVA results for the various questionnaires and their subscales that were administered at the beginning of each session are presented in Table 3.

Depression was significantly reduced over the three sessions, as rated independently by the Beck Depression Inventory, the SCL-90-R Depression scale, and the SCL-90-R thoughts of death ideation item. Participants showed no significant changes in level of hopelessness or general anxiety level.

Our participants perceived themselves to be significantly more healthy psychologically after participation, as demonstrated by their significantly lower scores on the SCL-90-R dimensions of somatization, paranoid ideation, hostility, and psychoticism. Participants reported increased appetite and decreased overeating. They did not change on the obsessive-compulsive behavior or interpersonal sensitivity dimensions.

Changes in Sleep Quality and Medication Usage

When our first participant reported that he was falling asleep more rapidly at night due to the newly learned imagery exercises, we developed a sleep quality questionnaire to administer. Mean time to fall asleep at night was reduced significantly ($p < .001$) from over 1 hour during prebaseline week to less than ½ hour in the week after Phase 3 (see Figure 8). For example, prior to bedtime, a woman (SHSS:C = 7; car accident) reported "excruciating" low back pain (8 of 10) after milking cows and cleaning a barn for 3 hours. At nighttime, before entering the bedroom, she reported transferring her newly learned techniques by standing quietly and taking a deep breath, rolling her eyes up and then relaxing while thinking of riding her horses in the Appalachian mountains. She reported that the pain soon dissipated and she fell asleep "pretty much right away, rather than staying awake for several hours."

In terms of medications, 76% of the participants at initial interview took over-the-counter and prescription pain medications. At the end of

Table 3
Improvements in Psychological Well-Being Across the Three Experimental Sessions: Means (standard deviations) for Various Questionnaires

Scales	Session 1	Session 2	Session 3	*F*	*p* <
McGill Pain Questionnaire					
Sensory	23.50 (10.34)	17.50 (10.63)	19.25 (10.02)	4.65	.02
Affective	4.67 (3.65)	3.08 (3.03)	3.33 (2.77)	2.68	.09
Evaluative	3.42 (1.56)	2.25 (1.87)	2.25 (1.42)	3.62	.05
Total	38.83 (17.39)	27.93 (18.21)	30.42 (16.85)	5.70	.01
Pain (PPI) 0-10 pt.	5.46 (2.63)	4.08 (1.50)	3.69 (1.93)	5.46	.01
Beck Depression	10.29 (7.66)	7.07 (5.54)	6.50 (4.57)	3.28	.05
Beck Hopelessness	3.54 (2.76)	2.85 (2.51)	2.46 (2.40)	1.53	*ns*
STAI Trait Anxiety	37.67 (9.14)	35.33 (9.88)	35.50 (10.73)	0.86	*ns*
SCL-90-R Scales					
Somatization	11.50 (5.36)	10.64 (6.36)	8.93 (5.57)	4.50	.02
Depression	10.64 (8.63)	6.64 (3.48)	5.86 (4.33)	3.50	.05
Obsessive-Compulsive	1.21 (1.67)	0.64 (1.39)	0.71 (1.59)	1.83	*ns*
Paranoid Ideation					
Interpersonal	8.71 (7.15)	5.93 (4.12)	4.64 (3.71)	3.71	.04
Sensitivity	4.36 (4.05)	4.29 (3.29)	3.00 (2.29)	1.24	*ns*
Hostility	3.93 (2.81)	2.07 (2.13)	1.43 (1.60)	9.09	.001
Psychoticism	4.86 (2.66)	3.86 (2.71)	2.50 (2.25)	7.55	.003
SCL-90-R items					
Poor appetite	3.00 (2.88)	2.71 (2.59)	1.50 (1.23)	3.56	.05
Overeating	3.07 (2.67)	2.79 (2.46)	1.64 (2.10)	3.67	.04
Death thoughts	0.93 (1.00)	0.93 (1.14)	0.29 (0.47)	3.55	.05

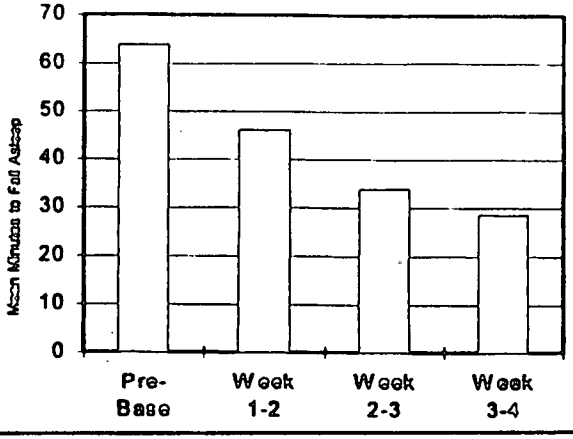

Figure 8. **Mean number of minutes to fall asleep reported by chronic pain participants across 4 weeks from preboseline week through 1 week postexperimental.**

the 3 experimental weeks, 35% reported a cessation of medications to assist in nighttime sleep. For example, one man (SHSS:C = 10; damaged disks from heavy lifting and two car accidents, with a history of back surgery) who reported commonly drinking 10 beers and taking several Valium to reduce pain and help in falling asleep at night, stopped such behavior and used the eye-focusing techniques to "send messages downward" to eliminate pain.

DISCUSSION

> You only believe what you already believe. If you are given something you don't believe, you have to change your schema in order to incorporate it. Since a year ago, my back hurts all the time. I have lived with extreme pain. It's weird now because I am without pain. I knew I believed beforehand that I would *not* be hypnotized and would *not* reduce pain in your experiment. It was an awkward feeling to know I had to change my beliefs. It's not too often in life that something slaps you in the face and you have to turn around and make a 360° turn. (Report given at third experimental session by highly hypnotizable [SHSS:C 11] chronic low back pain woman who had been in two major car accidents.)

Before discussing the results, the reader is reminded that the participants had a long history of chronic low back pain, existing for a mean of 4 years (range: 6 months to 11 years), with a prior history of treatment failure as reported by their physicians. Etiology of back pain was known for 88%—motor vehicular and lifting/exercise accidents. Neurologists had determined that 71% had one or more damaged disks. Physicians and chiropractors referred to us only those patients who had participated in long and unsuccessful medical interventions: pain medication regimes sometimes with additional physical therapy, biofeedback, and

chiropractic interventions. In fact, 14% had undergone back surgery without adequate relief, a not uncommon finding in the medical literature. They came to the experiment with little knowledge about hypnosis and little or no expectation of relief, but willing to try because their physicians had encouraged their participation.

It was indeed surprising to the majority of the participants—and to the researchers—that they were hypnotizable. Similarly, the participants were surprised that they learned to control experimental pain and were often able to successfully transfer such newly learned techniques to their own chronic pain. As reported in Part 1, during the training of hypnotic analgesia during cold-pressor pain, by the third hypnotic analgesia dip, 60% had completely eliminated all pain perception and 80% had completely eliminated all distress perception. It is both our opinion and that of one of our anonymous reviewers that this is of "extraordinary magnitude" in comparison to unselected undergraduate students (e.g., Hilgard, 1973; Hilgard & Hilgard, 1994). Possible theoretical and clinical ramifications of such findings are discussed below. There is a need for replication in larger and more diverse chronic pain populations.

Experimental Pain Training Transfer Results in Reduced Back Pain and Increased Psychological Well-Being

Participating in an experimental study involving the learning of control over experimental pain resulted in transfer of the control to experienced low back pain as well as improved psychological well-being in daily living. Pain experienced at the time of arrival at the experiment decreased significantly. Individuals with more pain tend to report poorer sleep quality and more awakenings during the night (e.g., Affleck, Urrows, Tennen, Higgins, & Abeles, 1996). Overall, our participants reported significant enhancements in sleep quality, as reflected by reduced time to fall asleep, over the experimental period. Similarly, self-reported depression reduced significantly and psychological health increased significantly. Finally, use of medications reduced significantly.

The experiment demonstrates the importance of developing self-efficacy through the learning of experimental pain control and the understanding of one's own attentional and disattentional abilities. Our data provide experimental support to Brown and Fromm's (1987) introduction of experimental pain control as a first step in enhancing self-efficacy in chronic pain management. Furthermore, it argues for the early introduction of behavioral techniques such as hypnosis and relaxation before the development of chronic pain (Crawford, 1995a, 1995b). Already our experimental pain training approach using the cold-pressor test is being applied in clinical settings in the United States (e.g., Holroyd, 1996) and Europe (P. Alden, personal communication, October 1996). Since our research only used an A-B design in Part 2, replication would be useful with the addition of a wait-list control group.

The experience of hypnosis and hypnotic analgesia training for experimental cold-pressor pain contributed to major reductions in reported back pain at the end of the first session—even though no mention of back pain, or reduction thereof, had occurred during the hypnosis session. After concentrating on the lower back during Phase 3, participants were able to dramatically reduce or eliminate felt pain and distress with their newly learned techniques. Reports of pain level reduced over the three sessions. Yet the sole low hypnotizable participant reported high pain throughout with little change during hypnotic conditions.

Follow-up interviews over the subsequent month with the participants indicated that all but three continued to experience chronic low back pain, but felt they had more control in moderating or eliminating pain when they reminded themselves. Two women reported the continuing total elimination of chronic pain for 1 month; further follow-up was not possible due to their moving without leaving forwarding addresses. One man who had continuing chronic pain due to lifting heavy engines and having two car accidents reported, even 2 years after participation, that "all I have to do is now look at the wall, take a breath, and send messages down to turn off the pain." Another woman reported additional applications of the focusing technique to control stress and anger. The sole low hypnotizable continued to have back pain without additional control. One difficulty noted was that some participants failed to practice their techniques until reminded to do so during the telephone conversations. This highlights the need to do telephone, computer-assisted, or in-person follow-ups for encouragement and further training in clinical chronic pain treatment.

Whereas none of the chronic low back pain patients had been hypnotized previously, all but one were moderately to highly hypnotizable. Unknown to us is whether they represent a biased selection because they were volunteers from local doctors' practices. They knew hypnosis would be involved and thus self-selection may have occurred. They came to the research project knowing little about hypnosis, other than what they had heard or seen in the mass media, and not expecting any dramatic changes in their chronic pain levels, but willing to participate because traditional approaches (medications, surgery, or chiropractic manipulations) had failed to relieve their pain adequately. We had anticipated a wide range of hypnotic susceptibility so that those who were responsive could be compared with those who were not, but this was impossible to carry out.

Unique to this project was that the primary emphasis was on the psychophysiological correlates of experimental pain during conditions of attend and hypnotically suggested analgesia in a chronic pain group. The secondary emphasis was on the question of whether successful laboratory training of experimental pain reduction would transfer to the participants' applying their newly learned skills to their own chronic pain.

At present we do not know if there is a consistent relationship between hypnotizability and enduring chronic pain in certain disorders, although several studies (Remler, 1990; Stam, McGrath, Brooke, & Cosire, 1986) suggest its occurrence. Stam et al. (1986) found hypnotizability was higher in patients with temporomandibular pain and dysfunction syndrome, hypothesized to be associated with stress-related muscular hyperactivity, than in a normal population. Hypnotizability correlates with phobic disorders (for a review, see Crawford & Barabasz, 1993), certain eating disorders (e.g., Pettinati et al., 1990), dissociative disorders (e.g., Spiegel, Hunt, & Dondershine, 1988), nightmare occurrence (Belicki & Belicki, 1986), and itching severity within chronic ureticaria (Shertzer & Lookingbill, 1987). It is not that hypnotizability per se contributes to the development and maintenance of certain clinical conditions, but rather the relationships may reflect certain coping strategies, information processing styles, or attentional abilities that underlie both (Crawford & Barabasz, 1993).

Studies of chronic musculoskeletal pain patients have led Flor and Birbaumer (1994) to make some generalizations about psychobiological mechanisms of chronic pain: Muscular response stereotypes play an important role in maintenance, there is specific increased muscular reactivity that is localized to the area of chronic pain, and there is prolonged return to baseline after stressor termination. Chronic pain can lead to conditioning to pain-relevant stimuli (Birbaumer & Flor, 1994) and the development of a strong "neurosignature" or "neuromatrix" of pain that may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Furthermore, certain persons may be more vulnerable to developing chronic pain states: those with a prior pain-related conditioning history (Coderre, Katz, Vaccarino, & Melzack, 1993; Flor & Birbaumer, 1994), those with a predisposition (genetic and/or cultural) to show overreactivity to pain or enhanced conditionability (Devor, Inbal, & Govrin-Lippman, 1982), and those who are less likely to habituate to aversive stimuli (Birbaumer, Flor, Lutzenberger, & Elbert, 1995).

In comparison to healthy controls, chronic pain patients showed significantly higher dimensional complexity of the EEG (Flor & Birbaumer, 1994), SERP (Flor et al., 1995; Marlowe, 1992, 1995), and visual ERP (Connolly, Gawel, & Rose, 1982) differences. These differences suggest a central sensitization for noxious stimulation. Similarly, fibromyalgia patients had significantly lower heat pain thresholds with higher amplitudes of middle- and long-latency laser-evoked potentials (Lorenz, Grasedyck, & Bromm, 1996). Using positron emission tomography, Derbyshire et al. (1994) found that patients with atypical facial pain showed increased anterior cingulate but decreased anterior frontal activation in comparison to controls.

Chronic pain patients, such as those with fibromyalgia, have increased attention and heightened sensitivity to internal and external noxious stimulation, be it pain or noise (McDermid, Rollman, & McCain, 1996; Rollman & Lautenbacher, 1993). Wickramasekera's (1993) high risk model suggests that highly hypnotizable persons may be more vulnerable to inadvertently learning to amplify pain perception. Crawford (1995a, 1995b) proposes that highly hypnotizable individuals might be more vulnerable to the development of chronic pain due to their absorptive attentional and imaginal abilities that may contribute to overreactivity to pain and possible enhanced conditionability. These assumptions are still speculative and go beyond the data that were presented. Our current research efforts are focused on the elucidation of these important questions about chronic pain states and their relationship to hypnotizability.

Overall General Conclusions

Hypnotic analgesia is an active process that requires inhibitory effort, dissociated from conscious awareness, where the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities. Hypnotic analgesia led to significant changes in somatosensory event-related potentials in chronic low back pain participants, most of whom were moderately to highly hypnotizable. The application of hypnotic analgesia techniques led to highly significant reductions in perceived sensory pain and distress to cold-pressor and noxious electrical stimulation. Enhanced positivity of potentials prestimulus and SERP changes poststimulus support the hypothesis that active inhibitory processes in the brain are involved in hypnotic analgesia.

Hypnotic analgesia is a powerful behavioral intervention that is effective in altering pain perception of both acute *and* chronic pain, particularly for the moderate to highly hypnotizable individual (e.g., Hilgard & Hilgard, 1994). Among persons with chronic low back pain, major reductions in reported low back pain occurred during the experimental sessions. In comparison to preexperimental baseline measures, there were significant improvements in the psychological well-being and sleep quality of the chronic pain participants across the three experimental sessions. The importance of developing self-efficacy through learning to control experimental pain and the understanding of one's own attentional and disattentional abilities was demonstrated as being a significant intervention in the modulation and control of chronic pain. The development of "neurosignatures of pain" can influence subsequent pain experiences (Coderre et al., 1993; Melzack, 1993) and may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Therefore, hypnosis and other psychological interventions

need to be *introduced early* as adjuncts in medical treatments for onset pain before the development of chronic pain.

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Hypnotische Analgesie: 1. Somatosensorische Ereignis korrelierte Potentialveränderungen auf noxische Reize und 2. Transfer Lernen zur Reduktion von chronischen Rückenschmerzen

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Zusammenfassung: Fünfzehn Erwachsene, im Alter von 18 bis 43 Jahren (*M* = 29 Jahre), mit chronischen Schmerzen im unteren Rückenbereich (*M* = 4 Jahre), nahmen an dieser Studie teil. Alle bis auf einen waren mittel bis gut hypnotisierbar. Im ersten Teil wurden somatosensorische Ereignis korrelierte Potentiale einer noxischen elektrischen Stimulation während den Bedingungen Aufmerksamkeit und hypnotische Analgesie (HA) ausgewertet. Die Ableitungsorte waren dabei folgende Regionen: anterior frontal (Fp1, Fp2), midfrontal (F3, F4), zentral (C3, C4), und parietal (P3, P4). Während der HA wurde die erwartete inhibitorische Verarbeitung nur durch eine erhöhte N140 in der anterioren frontalen Region und durch ein vor dem Stimulus beginnendes, positiv sich fortsetzendes kontingentes kortikales Potential bei Fp1 nachgewiesen. Während der HA zeigte sich eine verminderte räumlich temporale Wahrnehmung anhand von reduzierten Amplituden der P200 (bilateral, midfrontal und zentral, und links parietal) und der P300 (rechts midfrontal und zentral). HA ist ein aktiver Prozeß, der einer inhibitorischen Anstrengung bedarf, die dissoziiert von bewußter Aufmerksamkeit ist, wobei der anteriore frontale Kortex an einer topographisch spezifischen inhibitorischen Feedback Schleife beteiligt ist, der in der Zuteilung von thalamokortikalen Aktivitäten mitwirkt. Im zweiten Teil dokumentieren die Autoren die Entwicklung von Selbst-Wirksamkeit der Teilnehmer, die sie durch den erfolgreichen Transfer von neu erlernten Fähigkeiten experimenteller Schmerzreduktion, auf die Reduktion ihrer eigenen chronischen Schmerzen leisteten. Über den Verlauf von drei experimentellen Sitzungen berichteten die Teilnehmer eine Reduktion des chronischen Schmerzes, eine Zunahme des psychischen Wohlbefindens und eine verbesserte Schlafqualität. Die Entwicklung von "Neurosignaturen des Schmerzes" kann zukünftige Schmerzerlebnisse beeinflussen,

und möglicherweise in ihrer Größe erweitert und leichter reaktiviert werden. Deshalb sollte die Hypnose und andere psychologische Interventionen früh als Zusatz zu einer medizinischen Behandlung bei beginnendem Schmerz eingesetzt werden, bevor es zur Entwicklung von chronischen Schmerzen kommen kann.

Analgésie hypnotique: 1. Changements dans les potentiels évoqués somatosensoriels reliés à des stimuli nociceptifs et 2. Apprentissage par transfert pour réduire la douleur lombaire chronique

Helen J. Crawford, Timothy Knebel, Lyla Kaplan, Jennifer M. C. Vendemia, Min Xie, Scott Jamison, et Karl H. Pribram

Résumé: Quinze adultes souffrant de douleur lombaire chronique (*M* = 4 ans), âgés entre 18 et 43 ans (*M* = 29 ans), ont participé à cette étude. Tous, sauf un, étaient de moyennement à fortement hypnotisables. Dans la partie 1, des potentiels évoqués somatosensoriels reliés à des stimulations électriques nociceptives ont été évalués durant des conditions d'attente et d'analgésie hypnotique (AH), au niveau frontal antérieur (Fp1, Fp2), mi-frontal (F3, F4), central (C3, C4), et pariétal (P3, P4). Durant l'AH, le processus d'inhibition anticipé a été illustré par une augmentation de l'activité N140 dans la région frontal antérieure et par un potentiel cortical positif pré stimulus sur Fp1 seulement. Durant l'AH, une diminution de la perception spatio temporelle a été révélée par des amplitudes réduites en P200 (bilatéral mi-frontal et central ainsi que pariétal gauche) et en P300 (mi-frontal droit et central). L'AH est un processus actif qui requiert un effort d'inhibition, dissocié de l'attention consciente, et pour lequel le contex frontal antérieur participe à un circuit de rétroaction inhibiteur topographiquement spécifique qui contribue à l'allocation des activités thalamocorticales. Dans la partie 2, les auteurs illustrent le développement de l'auto efficacité à travers un transfert réussi, par les participants, des nouvelles habiletés de contrôle de la douleur expérimentale à la diminution de leur douleur chronique. En trois sessions expérimentales, les participants ont rapporté une diminution de la douleur chronique, une augmentation du bien-être psychologique et une augmentation de la qualité de leur sommeil. Le développement des "neurosignatures de la douleur" peut influencer les expériences ultérieures de douleur. On peut en augmenter l'étendue et les réactiver facilement. Conséquemment, l'hypnose et les autres interventions psychologiques se doivent d'être introduites précocement comme des ajouts aux traitements médicaux, dans les débuts de la douleur, avant que celle-ci ne se développe de façon chronique.

Analgesia hipnótica: 1. Cambios en los potenciales evocados sensoriosomáticos por estímulos novicos 2. Transferencia de aprendizaje para la reducción del dolor crónico de la zona lumbar

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Resumen: Participaron quince adultos con dolor crónico en la región lumbar (*M* = 4 años), entre los 18 y 43 años de edad (*M* = 29 años). Todos excepto uno

eran moderada a altamente hipnotizables. En la parte 1, los potenciales evocados sensoriosomáticos se correlacionaron con estimulación eléctrica nociva y fueron evaluados durante las condiciones de espera y de analgesia hipnótica (AH) en las zonas frontal anterior (Fp1, Fp2); frontal media (F3, F4); central (C3, C4); y parietal (P3, P4). Durante la AH, los procesos inhibitorios hipotetizados fueron evidenciados por el aumento a N140 de la zona anterior frontal y por un preestímulo positivo contingente al potencial cortical en Fp1 solamente. Durante la AH, se evidenció una disminución en la percepción espaciotemporal por la reducción de amplitudes de P200 (bilateral medio-frontal y central y parietal izquierdo) y P300 (medio frontal y central). La AH es un proceso activo que requiere un esfuerzo inhibitorio, disociado de la conciencia en donde participa la corteza frontal anterior, en un circuito topográficamente específico de inhibición retroalimentada que coopera en la asignación de actividades tálamocorticales. En la parte 2, los autores documentan el desarrollo de la eficacia propia a través de la transferencia exitosa de habilidades recientemente aprendidas de reducción experimental del dolor a la reducción del dolor crónico propio. Luego de tres sesiones experimentales, los participantes reportaron reducción del dolor crónico, aumento del sentimiento de bienestar y aumento de la calidad del sueño. El desarrollo de una "neuromatriz de dolor" puede influenciar experiencias posteriores de dolor, aumentar y reactivarse fácilmente. Por lo tanto, es necesario introducir tempranamente la hipnosis y otras intervenciones psicológicas como ayuda en los tratamientos médicos para el dolor, antes que se produzca la cronificación del dolor.

BOOK REVIEWS

SPIEGEL, HERBERT, & SPIEGEL, DAVID. *Trance and Treatment: Clinical Uses of Hypnosis* (paperback edition). Washington, DC: American Psychiatric Press, 1987. Pp. xiv + 382. $23.50 U.S.

The latest paperback reprint of *Trance and Treatment: Clinical Uses of Hypnosis* comes 16 years after its original publication (for review of the original, see Zinn, 1983). The book is based on the authors' extensive experience of clinical hypnosis, and the authors seek to provide clinicians with "a brief, disciplined technique for mobilizing and learning from an individual's ability to concentrate" (p. xi). Furthermore, they assess the scientific evidence for the clinical uses and limitations of hypnosis.

The book is divided into four sections, and its structure is intended to parallel the sequence of treatment in an encounter with a client: Section I defines and discusses hypnosis; Section II presents the Hypnotic Induction Profile (HIP), a 10-minute clinical assessment procedure; Section III offers hypotheses and data relating performance on the HIP to personality style and psychopathology; and Section IV explores the construction of a treatment strategy employing hypnosis and discusses a series of specific treatment strategies and clinical cases. In addition, the book advises on selecting the most appropriate treatment based on an assessment of hypnotizability, and it focuses on psychotherapy with highly hypnotizable individuals.

The HIP is a frequently used assessment tool and a controversial one in terms of what it is actually measuring. It includes a measurement of "eye roll" and a suggestion for arm levitation; response to this item is assessed by a number of posthypnotic subjective measures. It can be argued that the subjective nature of scoring the eye roll sign and the low difficulty level of the arm levitation item (about 90% of individuals pass) make the HIP a poor indicator of hypnotizability. Studies have found that the eye roll sign correlates poorly, and the score based on the arm levitation measures correlates only moderately, with the Stanford scales. Given recent discussions of the possibility of dual mechanisms driving hypnotic performance (specifically, compliance or nonhypnotic suggestibility for easy items, and cognitive abilities, such as absorption and imaginative involvement, for difficult items), and given the HIP's reliance on one very easy item in comparison with the Stanford scales' broad band of items, the HIP could be said to be measuring compliance rather than hypnotizability. It is worth discriminating, as Spiegel and Spiegel

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HYPNOTIC ANALGESIA: *1. Somatosensory Event-Related Potential Changes to Noxious Stimuli and 2. Transfer Learning to Reduce Chronic Low Back Pain*[1]

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Abstract: Fifteen adults with chronic low back pain (M = 4 years), age 18 to 43 years (M = 29 years), participated. All but one were moderately to highly hypnotizable (M = 7.87; modified 11-point Stanford Hypnotic Susceptibility Scale, Form C [Weitzenhoffer & Hilgard, 1962]), and significantly reduced pain perception following hypnotic analgesia instructions during cold-pressor pain training. In Part 1, somatosensory event-related potential correlates of noxious electrical stimulation were evaluated during attend and hypnotic analgesia (HA) conditions at anterior frontal (Fpl, Fp2), midfrontal (F3, F4), central (C3, C4), and parietal (P3, P4) regions. During HA, hypothesized inhibitory processing was evidenced by enhanced N140 in the anterior frontal region and by a prestimulus positive-ongoing contingent cortical potential at Fpl only. During HA, decreased spatiotemporal perception was evidenced by reduced amplitudes of P200 (bilateral midfrontal and central, and left parietal) and P300 (right midfrontal and central). HA led to highly significant mean reductions in perceived sensory pain and distress. HA is an active process that requires inhibitory effort, dissociated from conscious awareness, where the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities. In Part 2, the authors document the development of self-efficacy through the successful transfer by participants of newly learned skills of experimental pain reduction to reduction of their own chronic pain. Over three experimental sessions, participants reported chronic pain reduction, increased psychological well-being, and increased sleep quality. The development of "neurosignatures of pain" can influence subsequent pain experiences (Coderre, Katz, Vaccarino, & Melzack, 1993; Melzack, 1993) and may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Therefore, hypnosis and other psychological interventions need to be introduced early as adjuncts in medical treatments for onset pain before the development of chronic pain.

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Although the effectiveness of hypnosis in the relief of acute and chronic pain is well documented (e.g., Barber, 1996; Chaves, 1994; Evans, 1988; Hilgard & Hilgard, 1994; Hilgard & LeBaron, 1984), less is known about the cognitive and physiological processes involved in hypnotic analgesia. Electrophysiological and cerebral metabolic studies provide evidence for shifts in brain dynamics during hypnotically suggested analgesia in high but not low hypnotizable individuals in nonclinical populations (for reviews, see Crawford, 1994a, 1994b, in press; Crawford & Gruzelier, 1992). Yet to be addressed, however, is whether similar shifts in brain dynamics occur during hypnotic analgesia in chronic pain patients.

Highly hypnotizable persons (referred to as "highs") can partition their attentional resources more effectively than can low hypnotizable individuals (referred to as "lows"), as demonstrated at self-report, behavioral (e.g., Crawford, Brown, & Moon, 1993; Tellegen & Atkinson, 1974), and physiological (e.g., Crawford, 1994a, 1994b; Crawford, Corby, & Kopell, 1996; Crawford & Gruzelier, 1992) levels. Because of these abilities to control unwanted stimuli, such as pain, there is a moderate (around .50s) relationship between hypnotic susceptibility and pain reduction using hypnotic analgesia in normal populations (Hilgard & Hilgard, 1994). The degree to which hypnotic susceptibility plays a moderating role in hypnotic analgesia reduction in chronic pain populations continues to be debated (Chaves, 1994; Hilgard & Hilgard, 1994; Holroyd, 1996).

In adults with chronic low back pain, Part 1 of the present research reports somatosensory event-related potential correlates of noxious stimulation during attend and hypnotically suggested analgesia conditions. Part 2 evaluates the transfer of newly learned skills of experimental pain reduction to reduction of their own chronic pain.

PART 1. SOMATOSENSORY EVENT-RELATED POTENTIAL CHANGES TO NOXIOUS STIMULI

Hypnotic alterations in perceptual experiences such as suppressing auditory (e.g., Crawford et al., 1996; Kunzendorf & Boisvert, 1996; Lamas & Crawford, 1997), visual (e.g., Bányai, Mészáros, & Greguss, 1980; De Pascalis, 1994; Jasiukaitis, Nouriani, & Spiegel, 1996; Mészáros & Bányai, 1978; Spiegel, Cutcomb, Ren, & Pribram, 1985), or somatosensory input (see below) are accompanied by changes in scalp-recorded event-related potentials that also provide support for differences in inhibitory processing between lows and highs.

Scalp-recorded somatosensory event-related potentials (SERPs) have been found to be important indicators of pain processing. Several studies (Arendt-Nielsen, Zachariae, & Bjerring, 1990; Crawford, 1994b; De Pascalis, Crawford, & Marucci, 1992; Galbraith, Cooper, & London, 1972; Guerrero-Figueroa & Heath, 1964; Hernandez-Peon & Donoso, 1959; Mészáros, Bányai, & Greguss, 1980; Sharev & Tal, 1989; Spiegel, Bierre, & Rootenberg, 1989; Zachariae & Bjerring, 1994; Zachariae, Bjerring, Arendt-Nielsen, Nielsen, & Gotliebsen, 1991) show significant decreases in late SERP components in response to unpleasant cutaneous stimulation during hypnotic analgesia, whereas others (e.g., Meier, Klucken, Soyka, & Bromm, 1993) do not. Using median nerve stimulation, Mészáros et al. (1980) reported decreases in the P200 at vertex (Cz) accompanying hypnotic analgesia. De Pascalis et al. (1992) reported decreases in the N150-P200 component in the posterior region to a strongly noxious electrical stimulus. Spiegel et al. (1989) found highs showed significant P100 (F3, F4, Cz, P3, P4, O1, O2) and P300 (F4, P4, and O2) amplitude decreases when they hallucinated a local anesthetic at the wrist and hand to a mildly uncomfortable electrical stimulus.

However, thus far no hypnosis SERP study has considered the anterior frontal (prefrontal) region, although it is implicated in pain (Desmedt & Tomberg, 1989; Jones, Brown, Friston, Qi, & Frackowiak, 1991; Pribram, 1991) and differentiated from pain processes associated with the posterior regions (Head, 1920; Pribram, 1991; Price, 1988). On the other hand, anterior frontal shifts in brain dynamics during hypnotic analgesia, as measured by regional cerebral blood flow, are documented in highs but not found in lows (Crawford, Gur, Skolnick, Gur, & Benson, 1993). Posner and Petersen (1990) propose there are two major attentional systems: (a) one located in the posterior region of the brain and involved with selectively engaging and disengaging attention and (b) another located in the anterior region and involved in "attention for action" or effortful attention. For Pribram and McGuinness (1975, 1992; see also Pribram, 1991), as for Posner and Peterson, selective attention is a function of the posterior cerebral cortex, whereas effortful focused attention involves

inhibition and resistance to distraction, a function of the fronto-limbic systems (e.g., Bolster & Pribram, 1993; Pribram, 1991). As well, the supervisory attentional system (SAS; Shallice, 1988) of the anterior frontal cortex, involved in the monitoring of serial position of events and in sustaining focused attention, fits this scheme as does the hypothesized executive controller present in Hilgard's (1973, 1986) neodissociation theory of hypnotic analgesia. This executive controller, or SAS, is hypothesized to modulate "lower-level systems (other parts of the brain) by activating or inhibiting particular schemata" (Frith, 1991, p. 186).

On the basis of these earlier findings, we propose that hypnotic analgesia involves a supervisory, attentional control system of the anterior frontal cortex interacting with other cortical and subcortical regions, and that highly hypnotizable individuals can better control pain because of their more effective frontal attentional system (Crawford, 1990; Crawford, Brown, et al., 1993). Fronto-limbic operations apparently control input to the more posterior systems of the cortex (e.g., Skinner & Yingling, 1977). Specifically, the anterior frontal cortex gates the early stages of somatosensory processing as early as 28 m poststimulus (Yamaguchi & Knight, 1990; see also Desmedt, Nguyen, & Bourguet, 1983). Further evidence comes from 133Xe regional cerebral blood flow (CBF) imaging. Crawford, Gur, et al. (1993) found that highly hypnotizable persons showed a significant increase of bilateral CBF activation of the anterior frontal cortex, as well as the somatosensory cortex, during hypnotic suggested analgesia to ischemic pain. Positron emission tomography of fibromyalgia patients during hypnotic analgesia replicated these findings (B. Finer, personal communication, July 1996).

In addition to 30 painful electrical stimuli given to the left middle finger, Kropotov, Crawford, and Polyakov (1997) recorded intracranial SERPs from temporarily implanted electrodes in the anterior cingulate cortex, amygdala, temporal and parietal cortices of two obsessive-compulsive patients during attention and hypnotically suggested analgesia. In the hypnotically responsive patient, reduced pain perception was accompanied by a significant reduction of the positive SERP component within the range of 140-160 ms poststimulus recorded from the anterior cingulate cortex. This finding extends prior positron emission tomography (Casey et al., 1994; Derbyshire et al., 1994; Jones et al., 1991; Talbot et al., 1991) and functional magnetic resonance imaging (MRI) (Davis, Wood, Crawley, & Mikulis, 1995) that implicate the anterior cingulate in the processing of pain. A significant enhancement of a negative SERP component within the range of 200-260 ms was recorded from the anterior temporal cortex, and may be an indication of increased inhibitory processing. Note that this negativity occurred 50-100 ms later than changes recorded from the anterior cingulate cortex.

Present Study

In the present study, we anticipated that successful reductions in pain and distress to recurring noxious electrical stimulation during hypnotic analgesia would result in changes in SERP components between 50 and 500 ms known to reflect cognitive brain processing mechanisms: the well-known P70, N140 (also known as N150) and P260 occurring after electrical median nerve stimulation (for reviews, see Desmedt, 1979, 1988). Furthermore, we anticipated they would be differentially affected over time in anterior and posterior scalp regions. Past SERP pain research commonly reports a broad positive peak around 260 ms, but Miltner, Johnson, Braun, and Larbig (1989) clearly demonstrated that there are actually two distinct positive peaks (P200, P300), which vary in latency across participants. We likewise differentiate between these two peaks and the intervening N250 in the present study. Amplitude reductions of the P200 and P300 components reflect endogenous perceptual processes (for review, see Handwerker & Kobal, 1993), which are affected by changes in attention (e.g., Josiassen, Shagass, Roemer, Ercegovac, & Straumanis, 1982; Miltner et al., 1989) and vary with perceived pain level (Chen, Chapman, & Harkins, 1979; Miltner et al., 1989). In Miltner et al.'s study, N150 was not affected by attention under conditions where stimuli were randomized (weak vs. strong) over varying interstimulus intervals (ISIs) of 12-14 s, but has not been considered within our paradigm where active inhibitory processing can be developed to recurring strong stimuli.

We anticipated earlier time-locked effects in the anterior region due to the proposed early reallocation of attention to suppressing pain and distress from the anterior frontal region. Later SERP components would be affected due to the actualized perception of intensity (or the lack thereof during hypnotic analgesia) in midfrontal, central, and parietal regions. The present study assessed P70, N140, P200, N250, and P300 SERP components. We anticipated reductions of the positive components due to reduced pain perception during hypnotic analgesia, with increases in the negative components due to increased inhibitory processing.

To our knowledge, our laboratory is the first to investigate during hypnotic analgesia possible changes in contingent cortical potentials that occur *prior* to the repeating noxious stimuli and differentially reflect one's expectations to respond or inhibit motoric or cognitive responses. Such contingent cortical potentials may be "described as a measure reflecting the tuning of cortical excitability" and having an "influence on behavioral responses" (Birbaumer, Elbert, Canavan, & Rockstroh, 1990). They are specific to the cortical region, stimulus-response conditions, presence or absence of motoric response, and arousal or sustained cognitive activity (distraction) levels of the participant (for reviews, see Tecce, 1972; Tecce & Cattanach, 1982; Tecce & Hamilton, 1973). For instance, a maximal anterior frontal graded negativity is recorded during a period of focused, spotlighted attention (Asenbaum, Lang, Egkher, Lindinger, & Deecke, 1992; Hansen & Hillyard, 1988). A contingent negative variation (CNV) or readiness potential (*Bereitschaftspotential*) occurs in the central region prior to the initiation of an intended movement (for review, see Birbaumer et al., 1990). A similar negative deflection is recorded from the parietal cortex prior to the anticipated cessation of an act (Donchin, Otto, Gerbrandt, & Pribram, 1973; Kornhuber & Deecke, 1965). Conversely, a slow positivity—a contingent positive variation (CPV)—occurs when participants refrain irrelevant movements, supposedly reflecting the participant's inhibitory effort (Karrer, Warren, & Ruth, 1978; Konttinen & Lyytinen, 1993).

Thus, inhibitory processing is associated with positive-going contingent variations, whereas excitatory processing is associated with negative-going contingent variations (Birbaumer et al., 1990; Tecce, 1972). If hypnotic analgesia involves active inhibitory processes at the cortical level, as proposed, we might anticipate greater positivity of prestimulus contingent cortical potentials—that is, an occurrence of a CPV—during hypnotic analgesia than attend conditions.

METHOD

Participants

Participants were 17 adults who were referred to the research project by a local physician or chiropractor because they were experiencing chronic low back pain for at least 6 months and had not obtained adequate pain relief from traditional medical approaches. They ranged in age from 19 to 43 years (M = 29 years).

They reported their chronic low back pain to have existed from 6 months to 11 years (M = 4 years). Motor vehicular accidents accounted for 41%, lifting/exercise (e.g., lifting heavy car motors or furniture) for 47%, and unknown etiology for 12%. Of the sample, 71% had damaged disks and 14% had undergone back surgery. None had been hypnotized previously. Participants were provided monetary remuneration ($30 per session) for their participation.

All 17 participants were interviewed, assessed for hypnotic susceptibility level, and trained to reduce cold-pressor pain during the first session. Two participants were dropped from further neurophysiological analyses (reasons: work made participation impossible, inadequate electroencephalograph [EEG] recording) and another was dropped from Phase 3 analyses due to the occurrence of a head concussion. The final sample for which we had SERP measures is composed of 15 right-handed adults (5 men and 10 women). Participants refrained from caffeine and pain medication use for at least 5 hours prior to the SERP experiment.

Overview of Procedure

There were three 3-hour experimental sessions, each separated by 1 week: (a) interview about pain history, administration of background

questionnaires, administration of the Stanford Hypnotic Susceptibility Scale, Form C (SHSS:C; Weitzenhoffer & Hilgard, 1962), and training to reduce cold-pressor pain; (b) assessment of somatosensory pain-related potentials; and (c) assessment of EEG during cold-pressor pain dips. Consent forms were signed at the beginning of each session. The present article reports Sessions 1 and 2, as well as psychological well-being measures for all sessions. EEG analyses during the cold-pressor pain dips recorded during Phase 3 are reported elsewhere (in preparation).

Prescreening and Baseline Questionnaires

A local physician or chiropractor provided an information sheet regarding the NIH-sponsored research project to individuals whom they thought met the criteria (significant ongoing low back pain for a minimum of 6 months with no other major neurological [including head concussions] or medical/psychiatric disorders, not presently in psychotherapy, between the ages of 18 and 45 years, and right-handed). If interested, potential participants contacted the principal investigator (HJC) by telephone. The project was thoroughly explained and discussed at that time and further screening questions were provided.

For 1 week prior to the first session, participants were requested to fill out a pain diary that logged the level of pain they experienced daily and a sleep diary that logged their night experiences. Amount and type of prescribed and over-the-counter medications taken were recorded. The pain and sleep diaries were maintained daily for the duration of the experiment and a minimum of 1 week afterward.

Session 1: Interview, Hypnotic Susceptibility Assessment, and Training to Reduce Cold-Pressor Pain

On arrival, participants had the project explained again and the consent form for Phase 1 was signed. It was emphasized this was a research project and there would be no clinical therapy addressing possible psychological issues (e.g., depression). Consent forms giving permission to obtain information concerning their chronic pain and associated disorders from their doctors were also signed.

During the first hour, each participant was interviewed about the onset of pain, possible causes, progression over time, and previous treatments. They returned pain and sleep diaries they had kept for the prior week. They filled out questionnaires including the following: the McGill Pain Questionnaire (Melzack, 1975), the Symptom Checklist 90-Revised (SCL-90-R; Derogatis, 1983), Beck Depression Inventory (Beck, 1967), Beck Hopelessness Inventory (Beck, Weissman, Lester, & Trexler, 1974), and the State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). These questionnaires were completed at the beginning of subsequent sessions as well.

After a break, each participant participated in a short discussion about hypnosis and was introduced to cold-pressor dips. Each participant was administered individually an 11-item version of the SHSS:C. The dream suggestion item was deleted because the silent period could contribute to concentration on experienced pain and interrupt the hypnotic session. Following all SHSS:C suggestions and before the posthypnotic amnesia suggestion, the 20-min cold-pressor pain procedure (Hilgard & Hilgard, 1994) was administered.

Participants' left hands were placed into cold water (0-1°C) for 60-s periods in each of the following conditions: waking, hypnosis, and hypnosis with suggested analgesia (three times). They rated their sensory pain and distress on a standardized scale where 0 is *no pain*, 5 is *moderate pain*, and 10 is *unbearable pain*, but they could go higher than 10 on an open-ended scale to represent greater pain (Hilgard & Hilgard, 1994). Participants were instructed they could remove their hand at any time if the pain became too intense. One dip occurred prior to the administration of SHSS:C. Following the attend dip during hypnosis, participants were taught hypnotic analgesia techniques, including suggestions of the hand and arm being numb and insensitive, and imagining of a place such as the mountains or beach at which they would like to be. After the first two training dips, participants reported their successful strategies and these were incorporated into the suggestions for the next training dip.

The cold-pressor pain apparatus was a cooler chest filled with water and an ample supply of crushed ice so that its recorded temperature remained at approximately 0 to 1°C. A pump to circulate the water was not used, as is sometimes done, because we did not want to produce any recording artifacts during the session when EEG was recorded simultaneously. For each dip, immediately after the crushed ice was stirred by the experimenter, the participant's left hand was placed in the water for 60 s. During this time, the hand may have produced a layer of warmth around it to an unknown degree. For a discussion of these experimental issues see Hilgard (1967).

Session 2: Assessment of Somatosensory Pain-Related Potentials

On arrival, participants were informed that the study involved an evaluation of brain wave activity accompanying painful electrical stimuli that would be presented in waking and following a hypnotic induction when asked to attend and ignore the stimuli with previously learned hypnotic analgesia techniques. In addition, they were told that they would think of a pleasant trip with eyes open and eyes closed in waking and hypnosis conditions. Care was taken to develop rapport with the

participants and to put them at ease. The EEG and SERP recording procedures were described clearly to the participants, with recorded raw EEG data from a prior participant often shown to them. All questions were answered before the session. After signing the consent form, participants filled out questionnaires including the following: The McGill Pain Questionnaire, SCL-90-R, Beck Depression Inventory, Beck Hopelessness Inventory, and the State-Trait Anxiety Inventory. They returned pain and sleep diaries they had kept for the prior week.

In a quiet, moderately lighted room, participants were seated comfortably in a recliner chair with the experimenter nearby and a curtain blocking them from viewing the computer and computer operator on the other side of the room. After placement of the electrode cap on the head and the somatosensory stimulator on the finger, the participants had their sensory intensity levels titrated (see details below).

Participants served as their own controls in two A-B-A designs. First, there were waking, hypnosis, and waking conditions. In the two waking conditions before and after hypnosis, participants were asked to think of a pleasant trip once taken for counterbalanced 60-s eyes-open and eyes-closed periods, as well as asked to attend to the 30 painful stimuli; waking results are presented elsewhere (in preparation). Following the hypnotic induction, participants again thought of a pleasant trip for 60 s (not reported herein). Next, within hypnosis, participants served as their own controls in an A-B-A design in which they were presented with three sets of painful stimuli (described below) in the following conditions: attend, hypnotic analgesia, and attend. The hypnotic induction used a shortened version of SHSS:C instructions with all mention of sleep and drowsiness removed. The instructions for the attend conditions requested the participant to attend closely to their left hand and not use any techniques to reduce or eliminate the perception of pain. The hypnotic analgesia instructions were those used previously in Phase 1's cold-pressor pain training session with their previously successful strategies incorporated into the suggestions. After each set of painful stimuli, participants rated their sensory pain and distress on a standardized scale where 0 is *no pain*, 5 is *moderate pain*, and 10 is *unbearable pain*, but they could go higher than 10 on an open-ended scale to represent greater pain (Hilgard & Hilgard, 1994).

Immediately after removal of the electrodes, a short postexperimental interview was given to collect the participants' reactions to the conditions and to determine hypnotic analgesia techniques. Participants were shown their own EEG recordings. Great care was taken to ensure that they understood the experiment and that all questions were answered.

As reported in detail in Part 2, all participants were encouraged to apply their newly learned pain control techniques at their discretion to their own chronic pain during the day, before going to sleep, and if wakened during the night. To further assist, usually while standing,

participants were taught a simple 1-min eye-roll attentional focusing technique, similar to that used by Spiegel (1974) but with *no* mention of entering hypnosis, and were asked to practice their newly learned pain control techniques. Finally, participants were given pain and sleep diaries to fill out in the subsequent week.

Somatosensory stimuli. For each condition, stimuli consisted of 30 single square wave electrical pulses of 0.2 ms duration (rise/fall time of 20 microsecond), with a 3-s ISI. They were delivered to the center of the palmar surface of the distal phalange of the left hand's middle finger by a Grass S10DSCM somatosensory stimulator with an SIU8T stimulus isolation unit triggered externally by the recording 486 computer. The finger was prepared by having the participant rub the skin with an emery board, followed by the experimenter's vigorously rubbing of it with skin prep and alcohol swab.

Determination of stimulus intensity levels used during the recording periods. Analgesia research should use painful stimuli that are clearly and definitely painful (e.g., Becker, Yingling, & Fein, 1993). Sensory threshold, pain threshold, and pain tolerance levels were assessed using an ascending method of limits (Gescheider, 1985). Participants knew that it was necessary to provide stimuli that were strongly painful but bearable to assess electrophysiological responses to painful stimuli. Because some habituation to the stimuli often occurs with multiple trials, three ascending trials were given to determine when the stimulus was perceived as being strongly painful but still bearable. Participants rated their sensory pain on the same scale as used during cold-pressor pain training. A practice block of five stimuli at the chosen level was used to familiarize the participant with the sensations of finger stimulation and verify SERP recording. In this determination of stimulus intensity levels, our participants chose maximum bearable levels that were rated as being moderately to strongly painful (M = 6.88; SD = 1.07; range 4.5-8). Participants were able to tolerate these levels and did not produce finger movements or excessive ocular or myogenic artifact. Thus, we concluded that the stimuli were clearly painful for our participants.

Recording. Using a Lycra electrode cap (Electro-Cap International, Eaton, Ohio), EEG was recorded from 19 scalp sites referred to linked earlobes (A1, A2) and grounded to a location directly above the nasion. Electro-oculogram was monitored from electrodes placed inferior and lateral to the right outer canthus. Electrode impedances were kept below 3K ohm and balanced as equally as possible (less than 500 ohm difference). EEG data were collected using 20 Grass P5 series amplifiers (gain setting 10K; band pass: 0.1-100 Hz). The EEG signals were digitized at a rate of 200 samples per second for a period from 500 ms before each stimulus to 1,500 ms poststimulus. All instrumentation (including stimulus generation, EEG sampling, hard disk storage, and averaging) was

controlled by the Brain Scope program (Xie & Zheng, 1994). A digital signal from the computer activated a Grass S10DSCM somatosensory stimulator.

Data analyses. EEG analog records were first submitted to and epochs with artifacts were marked by the Brain Scope (Xie & Zheng, 1994) automatic eye movement (50 μV) and artifact rejection sequence. Subsequently, each epoch was scanned visually for verification and noting of further eye movement, muscle, or other artifacts. Those SERP epochs containing artifacts were not included in the data analyses. Presented here are amplitudes and latencies of SERP components (P70, N140, P200, N250, P300) at anterior frontal (Fp1, Fp2), midfrontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) regions during attend and hypnotic analgesia conditions following the hypnotic induction. Because there were varying prestimulus slow cortical potentials across conditions, baseline was determined by readjusting 0-ms to 0-point baseline. Because habituation may diminish SERPs (e.g., Calloway, 1973), the *two attend hypnosis conditions* (pre- and posthypnotic analgesia) were averaged and compared to the *hypnotic analgesia condition*. To assess the prestimulus slow cortical potential, the slope of the 200-ms segment just prior to stimulus onset was determined. Repeated measures 2 × 2 × 4 (Condition × Hemisphere × Region [anterior frontal, frontal, central, and parietal regions]) analyses of variance (ANOVA) were performed with Huynh-Feldt with follow-up ANOVAs to tease apart hypothesized effects in each region. Additional ANOVAs performed for the midline sites reflected similar regional findings and are available from the first author.

Results: Hypnotic Assessment and Preliminary Pain Training with Cold-Pressor Test

Hypnotic Susceptibility Scores

In general, the chronic pain participants were moderately to highly hypnotizable on the modified 11-point SHSS:C. The SHSS:C mean was 7.87 (SD = 2.27). One participant was low hypnotizable (SHSS:C score of 2), six were moderately hypnotizable (6, 7, 7, 7, 7, 7), and eight were highly hypnotizable (8, 8, 8, 9, 10, 10, 11, 11). It should be noted that this skewed distribution surprised us because we had anticipated a normal distribution of hypnotic susceptibility. As a result, we could not make hypnotic level a factor as had been originally planned.

Cold-Pressor Dip Ratings

Table 1 presents the means and standard deviations for pain and distress ratings during wake attend, hypnosis attend, and three hypnotic analgesia 60-s dips. Sensory pain and distress reports for waking and hypnosis attend dips did not differ significantly. The first hypnotic analgesia training dip led to highly significant ($p < .001$) reductions in

Table 1
Cold-Pressor Pain 60-s Dips During Waking and Hypnosis: Means (standard deviations) of Pain and Distress Reports and Percentage of Wake

Dips	Pain: Pain Report	Pain: Percentage of Wake Report	Distress: Distress Report	Distress: Percentage of Wake Report
Wake attend	9.53 (4.82)		8.73 (5.11)	
Hypnosis attend	9.13 (3.82)	101 (0.33)	7.52 (4.78)	89 (0.40)
Hypnotic analgesia 1	3.73 (4.71)	34 (0.33)	2.73 (4.68)	25 (0.32)
Hypnotic analgesia 2	1.60 (2.23)	14 (0.15)	1.00 (2.07)	7 (0.13)
Hypnotic analgesia 3	1.13 (2.39)	10 (0.17)	0.73 (2.05)	5 (0.11)

sensory pain and distress, respectively, Fs(1, 14) = 36.18, 25.45. In comparison to the first training dip, the second hypnotic analgesia training dip led to further significant reductions in sensory pain and distress, respectively, $F = 5.57$, $p < .005$; $F = 6.20$, $p < .05$. By the third hypnotic analgesia dip, 60% had completely eliminated all pain perception, and 80% had completely eliminated all distress perception.

SHSS:C hypnosis scores, although skewed toward higher scores, correlated significantly ($p < .01$) with amount of reduction reported during the three dips, Pain rs = −.63, −.71, −.76; Distress rs = −.66, −.61, −.77.

Experimental Conditions During Hypnosis: Reported Pain and Distress Levels

In comparison to the averaged attend conditions during hypnosis, during hypnotic analgesia to experimental pain the participants reported a highly significant reduction ($p < .001$) in both maximum sensory pain, Attend $M = 6.43$, $SD = 1.74$; Hypnotic Analgesia $M = 2.27$, $SD = 1.91$; $F(1, 14) = 42.48$, and distress, Attend $M = 5.17$, $SD = 2.34$; Hypnotic Analgesia $M = 1.13$, $SD = 1.13$; $F(1, 14) = 42.05$. During hypnotic analgesia, maximum sensory pain experienced was 39% (SD = .31) of Attend, and maximum distress was 24% (SD = .29) of Attend.

Experimental Conditions During Hypnosis: Somatosensory Event-Related Potentials

Figure 1 presents the grand averages for the SERPs for (a) attend to pain condition, which is the average of the pre- and postattends during hypnosis, and (b) hypnotically suggested analgesia. Because habituation may diminish SERPs (e.g., Calloway, 1973), the two attend hypnosis conditions (pre- and posthypnotic analgesia) were averaged and compared to the hypnotic analgesia condition. The first identifiable waveform was at P70. Table 2 presents a summary of all significant regional ANOVAs and a breakdown of the complex interactions with follow-up analyses. Because these Fs are not redundantly presented in the text, the

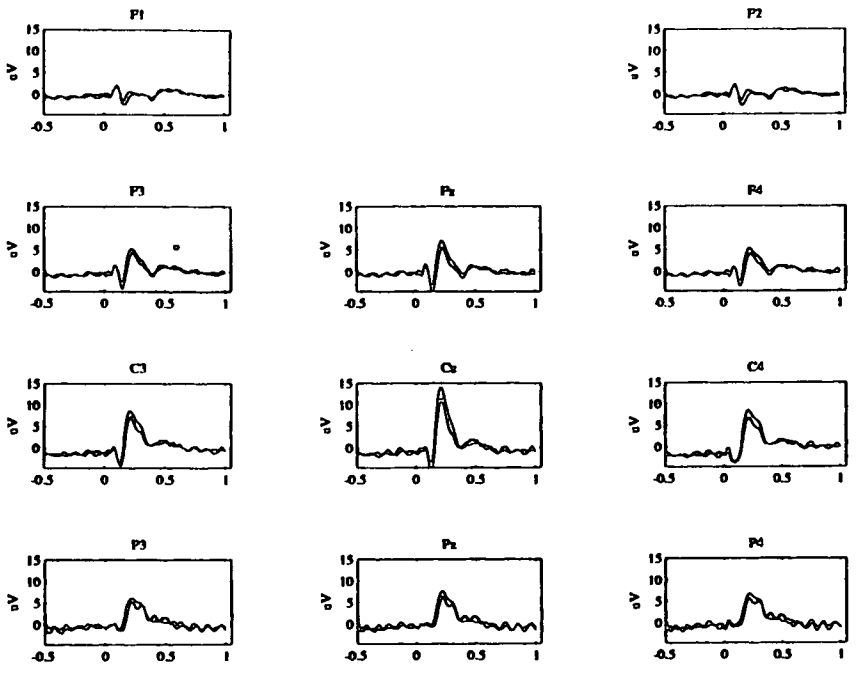

Figure 1. **Somatosensory ERP grand averages for Attend (light line; e.g., P200 higher amplitude at Cz) and Hypnotic Analgesia (dark line). Positivity is upward.**

reader is encouraged to refer to Table 2. Note that the *df*s are 1 and 14, unless noted otherwise.

Latencies did not differ significantly between conditions for P70 (*M*s: 66-77 ms), P200 (*M*s: 212-214 ms), P300 (*M*s: 303-310 ms), N140 (*M*s: 120-134 ms), and N250 (*M*s: 271-282 ms).

Mean amplitudes for P70, P200, P300, N140, and N250 components for the left and right anterior frontal, frontal, central, and parietal regions are presented in Figures 2 to 6, respectively.

Positive SERP Components: Mean Peak Amplitudes

As anticipated, during hypnotic analgesia, there were reductions in P200 and P300 amplitudes in the midfrontal, central, and parietal regions. Conditions had no significant effect on P70.

P70 amplitude. There was a significant Region × Hemisphere interaction, $F(3, 42) = 8.60$, $p < .05$. At the central region, P70 amplitude was significantly ($p < .001$) greater in the left (C3 = 1.79 μV) than right (C4 = 0.25 μV) hemisphere. Contrary to expectation, there were no significant

(text continued on p. 109)

Table 2
Summary of Significant Effects and Follow-Up Analyses of Condition × Hemisphere Analyses of Variance of Mean μV Amplitude for Each SERP Positive and Negative Components

Component	Region	Significant Effects	Significant Effects Analysis	Explanation
Positive SERP components				
P70	Central	Hemisphere $F(1, 14) = 16.81$, $p < .001$		C3 > C4
P200	Frontal	Condition $F(1, 14) = 4.92$, $p < .05$		Attend > Hypnotic Analgesia
	Central	Condition $F(1, 14) = 6.91$, $p < .02$		Attend > Hypnotic Analgesia
	Parietal	Condition × Hemisphere $F(1, 14) = 4.80$, $p < .05$	Hypnotic Analgesia: Hemisphere $F(1, 14) = 8.49$, $p < .02$	Hypnotic Analgesia: P3 < P4
			Attend vs. Hypnotic Analgesia Left Parietal (P3) $F(1, 14) = 3.72$, $p < .08$	P3: Attend > Hypnotic Analgesia
P300	Frontal	Condition $F(1, 14) = 10.87$, $p < .01$		Attend > Hypnotic Analgesia
		Condition × Hemisphere $F(1, 14) = 5.78$, $p < .05$	Attend vs. Hypnotic Analgesia Left Frontal (F3) $F(1, 14) = 4.00$, $p < .07$	F3: Attend > Hypnotic Analgesia
			Right Frontal (F4) $F(1, 14) = 22.94$, $p < .001$	F4: Attend > Hypnotic Analgesia
	Central	Condition × Hemisphere $F(1, 14) = 18.07$, $p < .001$	Attend vs. Hypnotic Analgesia Left Central (C3) $F(1, 14) = 1.77$, *ns*	C3: Attend = Hypnotic Analgesia
			Right Central (C4) $F(1, 14) = 23.91$, $p < .001$	C4: Attend > Hypnotic Analgesia

(Continued)

Table 2 Continued

Component	Region	Significant Effects	Significant Effects Analysis	Explanation
Negative SERP components				
N140	Anterior frontal	Condition $F(1, 14) = 9.04$, $p < .009$		Anterior frontal: Hypnotic Analgesia more negative
N250	Frontal	Condition $F(1, 14) = 11.09$, $p < .005$		Frontal: Hypnotic Analgesia more negative
	Central	Condition $F(1, 14) = 10.12$, $p < .007$		Central: Hypnotic Analgesia more negative
		Condition × Hemisphere $F(1,14) = 4.53$, $p = .05$	Attend vs. Hypnotic Analgesia	Stronger effect in C4 than in C3
			Left Central (C3) $F(1, 14) = 4.88$, $p < .05$	C3: Hypnotic Analgesia more negative
			Right Central (C4) $F(1, 14) = 14.75$, $p < .002$	C4: Hypnotic Analgesia more negative
Prestimulus contingent variation				
	Anterior frontal	Condition × Hemisphere $F(1, 14) = 8.34$, $p < .02$	Hypnotic Analgesia Hemisphere $F(1, 14) = 56.55$, $p < .001$	Fp1 > Fp2
			Attend Hemisphere $F(1, 14) = 1.17$, *ns*	Fp1 = Fp2

Note. SERP = somatosensory event-related potentials.

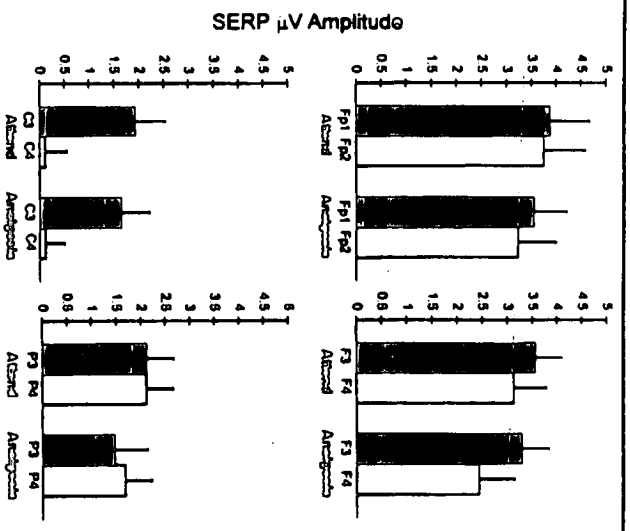

Figure 2. P70 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.

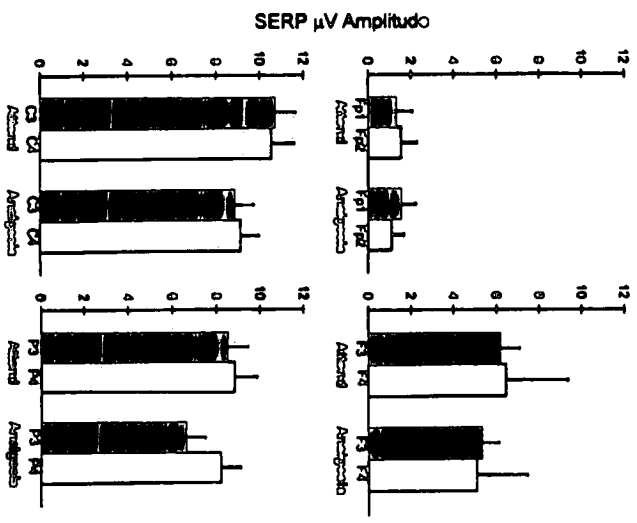

Figure 3. P200 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.

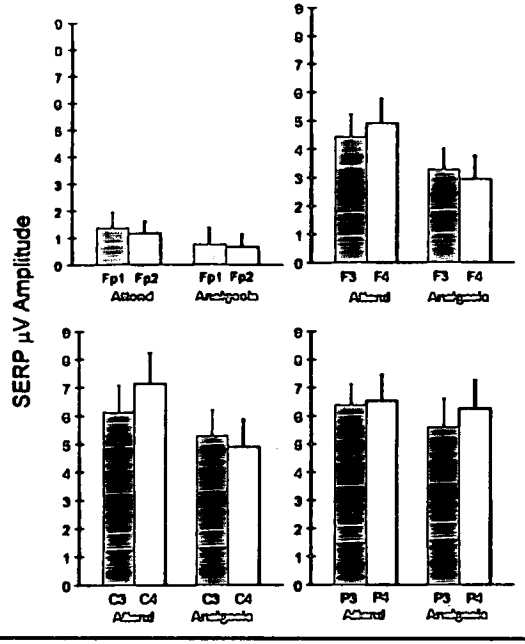

Figure 4. **P300 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

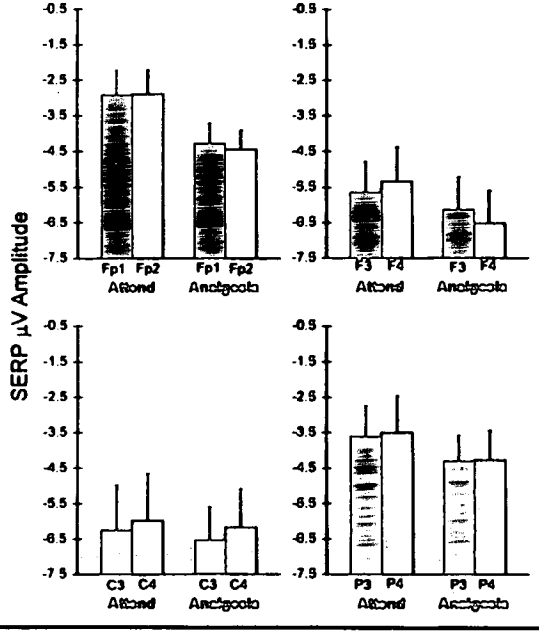

Figure 5. **N140 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

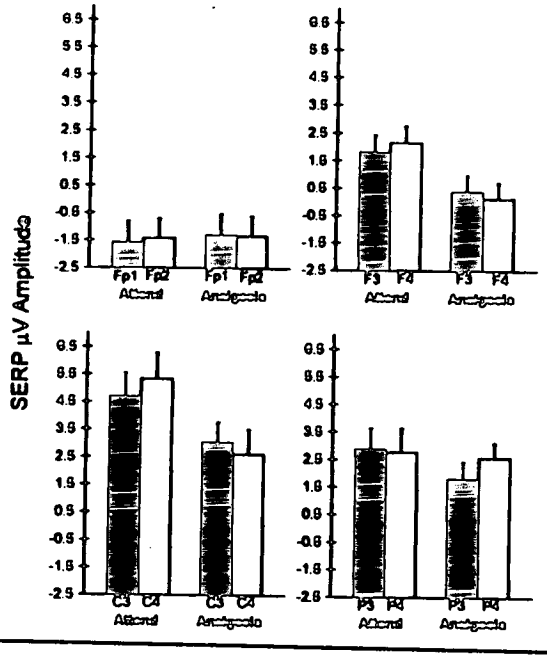

Figure 6. **N250 amplitude: Mean µV for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

differences between Attend and Hypnotic Analgesia conditions at any measured region (see Figure 2).

P200 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 5.35$, $p < .05$. As anticipated, in comparison to Attend, during hypnotic analgesia there were significant P200 amplitude reductions at midfrontal (Attend = 6.30 µV; Hypnotic Analgesia = 5.21 µV) and central (Attend = 10.62 µV; Hypnotic Analgesia = 8.97) regions (see Figure 3). A significant Hemisphere × Condition interaction was observed at the parietal region. Although there were no significant differences between hemispheres in Attend, during hypnotic analgesia there was a substantially greater reduction in the left parietal (6.63 µV) than right parietal (8.22 µV) region.

P300 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 7.38$, $p < .05$. Significant Hemisphere × Condition interactions were observed at midfrontal and central regions. As shown in Figure 4, in both regions, significant P300 amplitude reductions during hypnotic analgesia were observed in the right but not in the left hemisphere.

Negative SERP Components: Mean Peak Amplitudes

As anticipated, during hypnotic analgesia, enhanced N140 occurred in the anterior frontal region and enhanced N250 occurred in the midfrontal and central regions.

N140 amplitude. As anticipated, at the anterior frontal (Fpl, Fp2) region, there was a highly significant greater negativity of the N140 component during hypnotic analgesia (–4.37 μV) than during attend (–2.97 μV) conditions (see Figure 5).

N250 amplitude. There was a significant Region × Hemisphere × Condition interaction, $F(3, 42) = 5.11$, $p < .05$. There was significantly greater negativity of the N250 components at both midfrontal (Attend = 1.98; Hypnotic Analgesia = 0.25) and central (Attend = 5.02; Hypnotic Analgesia = 2.60) regions (see Figure 6). Although present in both hemispheres, these effects were significantly stronger in the right fronto-central region that was contralateral to the stimulated finger than in the left hemisphere.

Prestimulus Contingent Cortical Potentials

We examined the 200 ms prior to the stimulus to assess for negative-going (cortical activation) and positive-going (inhibitory processes) contingent cortical potentials. As can be seen in Figure 7, consistently in all regions there was little positivity or negativity during the Attend condition.

At the anterior frontal region there was a significant interaction between Condition and Hemisphere. Unlike the Attend condition in which there were no hemisphere differences and negligible positivity, during Hypnotic Analgesia there was significantly greater positivity in the left than in the right anterior frontal region.

Discussion

Hypnotic analgesia had a significant effect on the somatosensory event-related potentials accompanying noxious electrical stimulation: first, in the greater negativity shown more anteriorally; second, in the reduced amplitude of the cortical response to pain shown more posteriorally; and third, in a dramatic asymmetrical prestimulus positivity recorded from the anterior frontal electrodes.

Increased Negativity Anteriorly

Increased inhibitory processing during hypnotic analgesia was indicated by a shift toward greater negativity for N140 that occurred in the anterior frontal region only, supporting the evidence obtained from regional cerebral blood flow research (Crawford, Gur, et al., 1993). The N140 component is thought to reflect the "complex reciprocal interactions between posterior and prefrontal [anterior frontal] cortex and

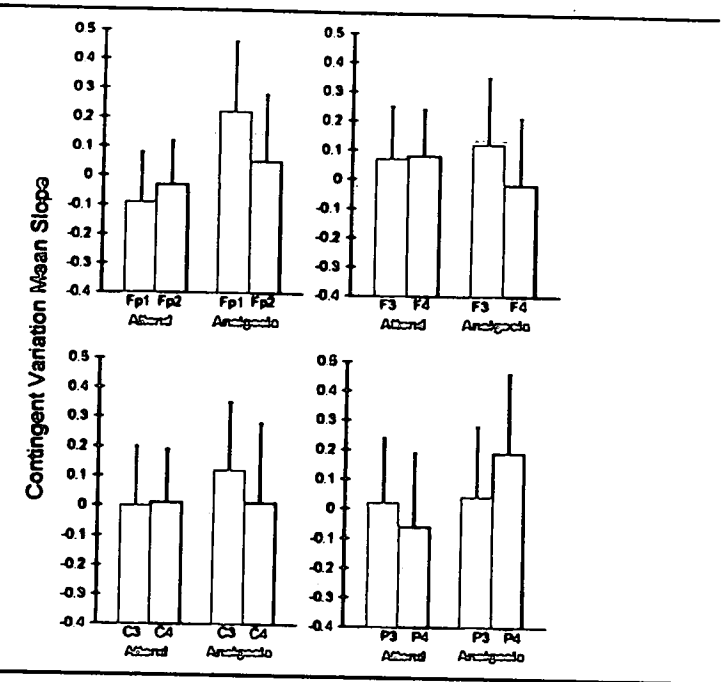

Figure 7. **Contingent variation: Mean slopes for 200 ms prestimulus for Attend and Hypnotic Analgesia conditions for left and right anterior frontal, frontal, central, and parietal regions.**

subcortical structures" that play "a key role in governing sequential attention processes" (Desmedt & Tomberg, 1989, p. 343). Additionally, during hypnotic analgesia, a greater N250 negativity was observed in the fronto-central region. We interpret these enhanced negative components to be indicative of active disattention during hypnotic analgesia, rather than the normally increasing spotlighted attention toward relevant incoming sensory signals.

Poststimulus Decreased Positivity

Reductions in perceived intensity of pain during hypnotic analgesia was observed (a) in the greatly reduced pain and distress reports and (b) in electrophysiological results: Reduced P200 amplitudes were observed in the midfrontal, central, and left parietal regions, and reduced P300 amplitudes were observed in the right midfrontal and central regions. These scalp-recorded positive components may reflect reduced involvement, during hypnotic analgesia, of the posterior parietal cortex whose function includes organizing sensory inputs received from the somatosensory and anterior frontal cortex to determine position of body parts (Desmedt & Tomberg, 1989).

Prestimulus Positivity

Dramatic additional evidence for the occurrence of inhibitory processing during hypnotic analgesia was obtained: A positive-going slope of the *pre*stimulus contingent cortical potential was found *only* in the left anterior frontal region. As reviewed by Birbaumer et al. (1990), maximal negativity of contingent cortical potentials occurs in the anterior frontal cortex "at the same time that prefrontal [anterior frontal] neurons critical for the solution of the task also show maximal activity" (p. 20). The contingent positive variation observed during hypnotic analgesia in the left anterior frontal region may therefore reflect a lowering of cortical activity (Birbaumer et al., 1990; Rockstroh, Müller, Wagner, Cohen, & Elbert, 1993) due to increased hyperpolarization that is inhibitory processing (Tecce, 1972; Tecce & Cattanach, 1982). The asymmetry is strong evidence against eye movement artifact as being the cause. The implications of the observed hemispheric difference cannot be evaluated fully because the present study only administered noxious stimuli to the left finger. Still, it should be noted that Basile, Rogers, Bourbon, and Papanicolaou (1994) reported magnetoencephalographically recorded CNVs to be asymmetrical in the frontal cortex, suggesting that tasks engaging the hemispheres differently "should result in asymmetric fields on the scalp" (p. 163). Furthermore, it should be noted that Gruzelier and his colleagues (for review, see Crawford & Gruzelier, 1992) suggest a decreased involvement of the right anterior region during hypnotic induction and certain hypnotic phenomena.

Tecce (1972; Tecce & Cattanach, 1982) argued that the prestimulus contingent cortical potentials are related to arousal and attention. Tecce (1972) also reviewed evidence that positivity occurs when responses have to be suppressed, thus reflecting "active inhibitory processes" (p. 76). This is born out in the work of Rockstroh et al. (1993), which has shown that depolarization in cortical dendritic trees generates a surface negativity while the positive-going waves result from a lowering of cortical excitability. Thus, "surface-negative shifts on the scalp, such as the CNV [contingent negative variation], are hypothesized to reveal enhanced cortical excitability enabling a preparatory state. . . . In contrast, slow positive shifts may result from a 'disfacilitation' in cortical neuronal networks" (Rockstroh et al., 1993, p. 236; for review, see Birbaumer et al., 1990).

Theoretical Implications

The development of hypnotic analgesia is seen as an active process, involving several brain systems, that requires inhibitory effort—although this effort may be dissociated from conscious awareness (e.g., Hilgard, 1986). We propose that during hypnotic analgesia a supervisory, attentional control system of the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities (e.g., Birbaumer et al., 1990; Pribram & McGuinness, 1975, 1992; Skinner & Yingling, 1977; Yingling & Skinner, 1976). Taken together with recent research with highly hypnotizable students using the same experimental paradigm (Crawford, 1994b; Crawford et al., 1997), our SERP data suggest that the anterior frontal region deals with the active allocation of attention and disattention, whereas spatiotemporal aspects of the somatosensory perceptions involve the posterior cortical systems.

Most certainly, other inhibitory pain systems are actively interacting with the frontal attentional system, including the limbic and thalamic systems, as evidenced by recent work (B. Finer, personal communication, July 1996; Kropotov et al., 1997). Such inhibitory processing during hypnotic analgesia may extend as far as spinal cord antinociceptive mechanisms as evidenced by reductions in brief latency (Hagbarth & Finer, 1963) and R-III amplitude (Kiernan, Dane, Phillips, & Price, 1995) of spinal reflexes (for an exception, see Santarcangelo, Busse, & Carli, 1989).

Drawing from recent spatiotemporal brain electrical source analyses (Allison, McCarthy, & Wood, 1992; Bromm & Chen, 1995; Tarkka & Treede, 1993) that indicate certain SERP components (approximately between 140 to 220 ms in magnetoencephalographic studies) are generated primarily in the frontal cortex and cingulate gyrus, our study suggests that these structures are involved in hypnotic analgesia strategies. Thus, it is of particular importance that the N140 enhancement during hypnotic analgesia was observed only in the anterior frontal region (see Figure 5). How early the effect of shifts in attention allocation during hypnotic analgesia occurs and the location of such generators is yet unknown.

Other recent work using intracerebral SERP (Kropotov et al., 1997) and cerebral metabolism (Crawford, Gur, et al., 1993; B. Finer, personal communication, July 1996) support such an interpretation. Kropotov et al. (1997) suggest the involvement of the anterior cingulate cortex in both pain perception and strategies of pain control. The anterior cingulate is heavily connected with the anterior frontal cortex and is thought to be an area that organizes responses to noxious stimuli (for review, see Devinsky, Morrell, & Vogt, 1995) and possibly has an SERP dipole associated with it (Bromm & Chen, 1995).

Further research would clarify and expand our neurophysiological model of hypnotic analgesia. Stimulations to both left and right fingers, with spatiotemporal brain electrical source analyses, are needed. This can be accomplished through the use of more electrodes and re-referencing to a common average to produce accompanying isopotential maps. Research employing noninvasive functional MRI is additionally useful. We are presently carrying out such work.

PART 2. EXPERIMENTAL PAIN TRAINING TRANSFER LEARNING TO REDUCE CHRONIC LOW BACK PAIN AND DEVELOPMENT OF SELF-EFFICACY

> When the burden of cure is abrogated to the implicit magic of the technique—rather than the patient's taking an active role in his or her treatment—any initial attempt to use hypnosis would at best be unsuccessful, and at worst would precipitate an early termination of the therapeutic encounter. Treatment cannot be passive, nor can it be solely the responsibility of the therapist. The patient must learn the self control that is needed for the mastery experience of pain control. (Evans, 1988, p. 37)

Whereas 90% of low back pain will remit naturally within 12 weeks, the other 10% becomes chronic, debilitating, and costly (Nachemson, 1982). Additionally, it recurs frequently in 40-60% of those previously inflicted (Haanen, 1984, as cited in Spinhoven, 1987). The hypnotic reduction of low back pain, as well as other chronic pains, is often addressed with suggestions aimed directly at reducing sensory pain in the affected area and accompanying distress, as well as relaxing the affected area and the body as a whole (for review, see Spinhoven, 1987). However, a somewhat different approach may be taken by applying hypnotic control techniques *first* to an experimentally induced pain and subsequently transferring those techniques to chronic pain. Relying heavily on the concept of self-efficacy (Bandura, 1977; Pribram, 1963, 1971; White, 1960), Brown and Fromm (1987) advocate such a multimodal approach to chronic pain management and reduction aimed at enhancing a sense of self-efficacy. They suggest starting the training of hypnotic control techniques with an induced pain (pinching the hand) and then moving through a hierarchy of experienced pains from that which is least bothersome to that which is the target pain. Having the experience of *learning first to control experimental pain* (instead of the clinical pain that may carry "psychological baggage") enhances feelings of confidence and self-efficacy and changes belief systems that one can have personal control over debilitating chronic pain. Learning skills and understanding one's own abilities within the context of pain control is of utmost importance.

In the present study, we assessed the degree to which learning the hypnotic skills used to control experimental pain would lead to increased psychological well-being as indicated by reported reductions in chronic low back pain over the three experimental sessions. Such reports can be interpreted as the result of skill transfer from control over experimental pain to control over chronic pain. Assessment of psychological well-being included measures of depression (Beck Depression Inventory), hopelessness (Beck Hopelessness Inventory), psychological distress (SCL-90-R), and sleep quality reports. Persons who are moderately to highly responsive to hypnosis, as measured by SHSS:C, were expected to show greater transfer effects than those who were rated as low hypnotizable. Unexpectedly, our participants were, except for one low hypnotizable, all moderately to highly hypnotizable; thus, we were unable to assess the latter hypothesis.

METHOD

At the end of the first phase (described above in Part 1), the participant and researcher discussed the degree to which the experimental pain was reduced and techniques that seemed most effective. To develop self-efficacy among the participants, several actions were taken. Participants were congratulated on their newly found skills. To provide a context in which to understand why these techniques may have worked and to de-emphasize the uniqueness of the hypnotic state, an emphasis was placed on newly found uses of attentional abilities that they already used in other contexts (e.g., ignoring noisy environments, becoming deeply involved in positive experiences such as watching movies or lovemaking). All participants were encouraged to apply these techniques at their discretion during the day, before going to sleep, and if wakened during the night.

At the end of Sessions 2 (SERP measurement) and 3 (EEG correlates of cold-pressor pain), participants were taught a simple 1-min eye-roll attentional focusing technique, similar to that used by Spiegel (1974) but with *no* mention of entering hypnosis. Individuals responsive to hypnosis may not need a formal hypnotic induction to apply their learned disattentional skills to pain reduction outside the hypnosis context (Hilgard & Hilgard, 1994). Participants were told this eye-roll technique served as a cue and would help them alert and focus their attention so they could possibly reduce their own chronic pain. Participants were asked to roll up their eyes toward the ceiling, and then, while maintaining this upward stare, to slowly close their eyes. They were then asked to take a slow deep breath, relax their muscles, and use self-selected techniques (e.g., send pain reduction messages down to their back, imagine being elsewhere, relax) to assist in the suppression of their own pain. After each of three trials, participant and experimenter discussed successful techniques and other possible techniques to try, as well as how their bodies felt. Participants were encouraged to practice their newly learned pain control techniques, with or without the eye-roll attentional focusing technique, as they desired at home and work. (Several participants reported subsequently changing the cue to staring at a wall rather than rolling their eyes upward and closing them.)

RESULTS

Changes in Back Pain Subsequent to First Hypnosis Experience

The experience of hypnosis and hypnotic analgesia training for experimental cold-pressor pain contributed to major reductions in reported back pain at the end of the first session—even though no mention of back pain, or reduction thereof, had occurred during the SHSS:C hypnosis session. After the hypnosis debriefing, after sitting in the chair for 1 1/2 hours, participants were casually asked, "How does your back feel?" Subsequently, they rated their pain level on the open-ended 0-10-point scale.

In comparison to the beginning of the administration of SHSS:C, their low back pain decreased quite significantly, before SHSS:C M = 5.13, SD = 2.20; and after SHSS:C M = 1.07, SD = 1.39; $F(1, 14) = 58.93$, $p < .0001$. All participants reported low back pain prior to SHSS:C administration (range 2-8). During the post-SHSS:C interview, 53% reported complete cessation of low back pain. They expressed surprise and indicated that typically they could not sit comfortably in a chair for this length of time. As one woman said, "Normally it would be 7 to 10 if I sat this long, but now it is only 1 or 2."

Changes in Back Pain Within Experimental Setting: Attend Versus Hypnotic Analgesia

Session 3 involved EEG recordings (in preparation) during 60-s cold-pressor pain dips while participants (one did not participate in this last session) attended to or used hypnotic analgesia. Subsequent to this, while still in hypnosis, the participants were asked to sit quietly and attend to their lower back for 1 min, after which they were asked about what they had experienced, including pain and distress reports. They were then asked to apply the hypnotic analgesia techniques they had just previously used for cold-pressor pain reduction to reduce pain in their own back. For approximately 2 min, the experimenter verbally suggested use of previously reported images and counted from 1 to 10. After 3 min of silence, participants were asked to report pain and distress levels.

Use of hypnotic techniques to decrease lower back region discomfort led to highly significant ($p < .001$) reductions in both sensory pain, Attend M = 4.39, SD = 1.90; Hypnotic Analgesia M = 0.65, SD = 1.25; $F(1, 13)$ = 35.42, and distress, Attend M = 4.08, SD = 1.66; Hypnotic Analgesia M = 0.58, SD = 1.66; $F(1, 13) = 23.89$. All but one participant reported pain when concentrating on their backs. After applying their pain control techniques, 69% reduced low back sensory pain to 0 and 76% reduced distress to 0. This provides strong evidence for the successful transfer of experimental pain control techniques to chronic pain.

Low Back Pain Reductions Across the Three Experimental Sessions

Over the three experimental sessions, our participants reported significant reductions in overall low back pain ($p < .01$), as assessed by the McGill Pain Questionnaire (see Table 3). They reported their low back pain (PPI on a 0- to 10-point scale) reduced significantly ($p < .01$). There were significant reductions for sensory ($p < .02$) and evaluative ($p < .05$) components but not for the affective ($p < .09$) component.

Improvements in Psychological Well-Being Across the Three Experimental Sessions

Over the three experimental sessions, a significant improvement in the psychological well-being of the chronic pain participants occurred. Means, standard deviations, and ANOVA results for the various questionnaires and their subscales that were administered at the beginning of each session are presented in Table 3.

Depression was significantly reduced over the three sessions, as rated independently by the Beck Depression Inventory, the SCL-90-R Depression scale, and the SCL-90-R thoughts of death ideation item. Participants showed no significant changes in level of hopelessness or general anxiety level.

Our participants perceived themselves to be significantly more healthy psychologically after participation, as demonstrated by their significantly lower scores on the SCL-90-R dimensions of somatization, paranoid ideation, hostility, and psychoticism. Participants reported increased appetite and decreased overeating. They did not change on the obsessive-compulsive behavior or interpersonal sensitivity dimensions.

Changes in Sleep Quality and Medication Usage

When our first participant reported that he was falling asleep more rapidly at night due to the newly learned imagery exercises, we developed a sleep quality questionnaire to administer. Mean time to fall asleep at night was reduced significantly ($p < .001$) from over 1 hour during prebaseline week to less than ½ hour in the week after Phase 3 (see Figure 8). For example, prior to bedtime, a woman (SHSS:C = 7; car accident) reported "excruciating" low back pain (8 of 10) after milking cows and cleaning a barn for 3 hours. At nighttime, before entering the bedroom, she reported transferring her newly learned techniques by standing quietly and taking a deep breath, rolling her eyes up and then relaxing while thinking of riding her horses in the Appalachian mountains. She reported that the pain soon dissipated and she fell asleep "pretty much right away, rather than staying awake for several hours."

In terms of medications, 76% of the participants at initial interview took over-the-counter and prescription pain medications. At the end of

Table 3
Improvements in Psychological Well-Being Across the Three Experimental Sessions: Means (standard deviations) for Various Questionnaires

Scales	Session 1	Session 2	Session 3	*F*	*p* <
McGill Pain Questionnaire					
Sensory	23.50 (10.34)	17.50 (10.63)	19.25 (10.02)	4.65	.02
Affective	4.67 (3.65)	3.08 (3.03)	3.33 (2.77)	2.68	.09
Evaluative	3.42 (1.56)	2.25 (1.87)	2.25 (1.42)	3.62	.05
Total	38.83 (17.39)	27.93 (18.21)	30.42 (16.85)	5.70	.01
Pain (PPI) 0-10 pt.	5.46 (2.63)	4.08 (1.50)	3.69 (1.93)	5.46	.01
Beck Depression	10.29 (7.66)	7.07 (5.54)	6.50 (4.57)	3.28	.05
Beck Hopelessness	3.54 (2.76)	2.85 (2.51)	2.46 (2.40)	1.53	*ns*
STAI Trait Anxiety	37.67 (9.14)	35.33 (9.88)	35.50 (10.73)	0.86	*ns*
SCL-90-R Scales					
Somatization	11.50 (5.36)	10.64 (6.36)	8.93 (5.57)	4.50	.02
Depression	10.64 (8.63)	6.64 (3.48)	5.86 (4.33)	3.50	.05
Obsessive-Compulsive	1.21 (1.67)	0.64 (1.39)	0.71 (1.59)	1.83	*ns*
Paranoid Ideation					
Interpersonal	8.71 (7.15)	5.93 (4.12)	4.64 (3.71)	3.71	.04
Sensitivity	4.36 (4.05)	4.29 (3.29)	3.00 (2.29)	1.24	*ns*
Hostility	3.93 (2.81)	2.07 (2.13)	1.43 (1.60)	9.09	.001
Psychoticism	4.86 (2.66)	3.86 (2.71)	2.50 (2.25)	7.55	.003
SCL-90-R items					
Poor appetite	3.00 (2.88)	2.71 (2.59)	1.50 (1.23)	3.56	.05
Overeating	3.07 (2.67)	2.79 (2.46)	1.64 (2.10)	3.67	.04
Death thoughts	0.93 (1.00)	0.93 (1.14)	0.29 (0.47)	3.55	.05

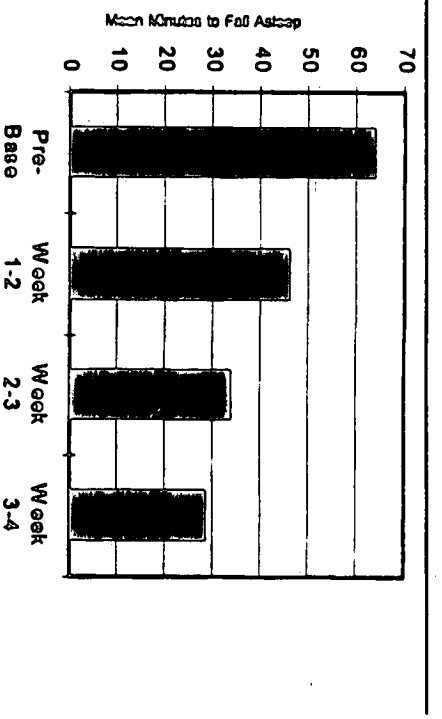

Figure 8. Mean number of minutes to fall asleep reported by chronic pain participants across 4 weeks from prebaseline week through 1 week postexperimental.

the 3 experimental weeks, 35% reported a cessation of medications to assist in nighttime sleep. For example, one man (SHSS:C = 10; damaged disks from heavy lifting and two car accidents, with a history of back surgery) who reported commonly drinking 10 beers and taking several Valium to reduce pain and help in falling asleep at night, stopped such behavior and used the eye-focusing techniques to "send messages downward" to eliminate pain.

Discussion

You only believe what you already believe. If you are given something you don't believe, you have to change your schema in order to incorporate it. Since a year ago, my back hurts all the time. I have lived with extreme pain. It's weird now because I am without pain. I knew I believed beforehand that I would *not* be hypnotized and would *not* reduce pain in your experiment. It was an awkward feeling to know I had to change my beliefs. It's not too often in life that something slaps you in the face and you have to turn around and make a 360° turn. (Report given at third experimental session by highly hypnotizable [SHSS:C 11] chronic low back pain woman who had been in two major car accidents.)

Before discussing the results, the reader is reminded that the participants had a long history of chronic low back pain, existing for a mean of 4 years (range: 6 months to 11 years), with a prior history of treatment failure as reported by their physicians. Etiology of back pain was known for 88%—motor vehicular and lifting/exercise accidents. Neurologists had determined that 71% had one or more damaged disks. Physicians and chiropractors referred to us only those patients who had participated in long and unsuccessful medical interventions: pain medication regimes sometimes with additional physical therapy, biofeedback, and

chiropractic interventions. In fact, 14% had undergone back surgery without adequate relief, a not uncommon finding in the medical literature. They came to the experiment with little knowledge about hypnosis and little or no expectation of relief, but willing to try because their physicians had encouraged their participation.

It was indeed surprising to the majority of the participants—and to the researchers—that they were hypnotizable. Similarly, the participants were surprised that they learned to control experimental pain and were often able to successfully transfer such newly learned techniques to their own chronic pain. As reported in Part 1, during the training of hypnotic analgesia during cold-pressor pain, by the third hypnotic analgesia dip, 60% had completely eliminated all pain perception and 80% had completely eliminated all distress perception. It is both our opinion and that of one of our anonymous reviewers that this is of "extraordinary magnitude" in comparison to unselected undergraduate students (e.g., Hilgard, 1973; Hilgard & Hilgard, 1994). Possible theoretical and clinical ramifications of such findings are discussed below. There is a need for replication in larger and more diverse chronic pain populations.

Experimental Pain Training Transfer Results in Reduced Back Pain and Increased Psychological Well-Being

Participating in an experimental study involving the learning of control over experimental pain resulted in transfer of the control to experienced low back pain as well as improved psychological well-being in daily living. Pain experienced at the time of arrival at the experiment decreased significantly. Individuals with more pain tend to report poorer sleep quality and more awakenings during the night (e.g., Affleck, Urrows, Tennen, Higgins, & Abeles, 1996). Overall, our participants reported significant enhancements in sleep quality, as reflected by reduced time to fall asleep, over the experimental period. Similarly, self-reported depression reduced significantly and psychological health increased significantly. Finally, use of medications reduced significantly.

The experiment demonstrates the importance of developing self-efficacy through the learning of experimental pain control and the understanding of one's own attentional and disattentional abilities. Our data provide experimental support to Brown and Fromm's (1987) introduction of experimental pain control as a first step in enhancing self-efficacy in chronic pain management. Furthermore, it argues for the early introduction of behavioral techniques such as hypnosis and relaxation before the development of chronic pain (Crawford, 1995a, 1995b). Already our experimental pain training approach using the cold-pressor test is being applied in clinical settings in the United States (e.g., Holroyd, 1996) and Europe (P. Alden, personal communication, October 1996). Since our research only used an A-B design in Part 2, replication would be useful with the addition of a wait-list control group.

The experience of hypnosis and hypnotic analgesia training for experimental cold-pressor pain contributed to major reductions in reported back pain at the end of the first session—even though no mention of back pain, or reduction thereof, had occurred during the hypnosis session. After concentrating on the lower back during Phase 3, participants were able to dramatically reduce or eliminate felt pain and distress with their newly learned techniques. Reports of pain level reduced over the three sessions. Yet the sole low hypnotizable participant reported high pain throughout with little change during hypnotic conditions.

Follow-up interviews over the subsequent month with the participants indicated that all but three continued to experience chronic low back pain, but felt they had more control in moderating or eliminating pain when they reminded themselves. Two women reported the continuing total elimination of chronic pain for 1 month; further follow-up was not possible due to their moving without leaving forwarding addresses. One man who had continuing chronic pain due to lifting heavy engines and having two car accidents reported, even 2 years after participation, that "all I have to do is now look at the wall, take a breath, and send messages down to turn off the pain." Another woman reported additional applications of the focusing technique to control stress and anger. The sole low hypnotizable continued to have back pain without additional control. One difficulty noted was that some participants failed to practice their techniques until reminded to do so during the telephone conversations. This highlights the need to do telephone, computer-assisted, or in-person follow-ups for encouragement and further training in clinical chronic pain treatment.

Whereas none of the chronic low back pain patients had been hypnotized previously, all but one were moderately to highly hypnotizable. Unknown to us is whether they represent a biased selection because they were volunteers from local doctors' practices. They knew hypnosis would be involved and thus self-selection may have occurred. They came to the research project knowing little about hypnosis, other than what they had heard or seen in the mass media, and not expecting any dramatic changes in their chronic pain levels, but willing to participate because traditional approaches (medications, surgery, or chiropractic manipulations) had failed to relieve their pain adequately. We had anticipated a wide range of hypnotic susceptibility so that those who were responsive could be compared with those who were not, but this was impossible to carry out.

Unique to this project was that the primary emphasis was on the psychophysiological correlates of experimental pain during conditions of attend and hypnotically suggested analgesia in a chronic pain group. The secondary emphasis was on the question of whether successful laboratory training of experimental pain reduction would transfer to the participants' applying their newly learned skills to their own chronic pain.

At present we do not know if there is a consistent relationship between hypnotizability and enduring chronic pain in certain disorders, although several studies (Remler, 1990; Stam, McGrath, Brooke, & Cosire, 1986) suggest its occurrence. Stam et al. (1986) found hypnotizability was higher in patients with temporomandibular pain and dysfunction syndrome, hypothesized to be associated with stress-related muscular hyperactivity, than in a normal population. Hypnotizability correlates with phobic disorders (for a review, see Crawford & Barabasz, 1993), certain eating disorders (e.g., Pettinati et al., 1990), dissociative disorders (e.g., Spiegel, Hunt, & Dondershine, 1988), nightmare occurrence (Belicki & Belicki, 1986), and itching severity within chronic ureticaria (Shertzer & Lookingbill, 1987). It is not that hypnotizability per se contributes to the development and maintenance of certain clinical conditions, but rather the relationships may reflect certain coping strategies, information processing styles, or attentional abilities that underlie both (Crawford & Barabasz, 1993).

Studies of chronic musculoskeletal pain patients have led Flor and Birbaumer (1994) to make some generalizations about psychobiological mechanisms of chronic pain: Muscular response stereotypes play an important role in maintenance, there is specific increased muscular reactivity that is localized to the area of chronic pain, and there is prolonged return to baseline after stressor termination. Chronic pain can lead to conditioning to pain-relevant stimuli (Birbaumer & Flor, 1994) and the development of a strong "neurosignature" or "neuromatrix" of pain that may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Furthermore, certain persons may be more vulnerable to developing chronic pain states: those with a prior pain-related conditioning history (Coderre, Katz, Vaccarino, & Melzack, 1993; Flor & Birbaumer, 1994), those with a predisposition (genetic and/or cultural) to show overreactivity to pain or enhanced conditionability (Devor, Inbal, & Govrin-Lippman, 1982), and those who are less likely to habituate to aversive stimuli (Birbaumer, Flor, Lutzenberger, & Elbert, 1995).

In comparison to healthy controls, chronic pain patients showed significantly higher dimensional complexity of the EEG (Flor & Birbaumer, 1994), SERP (Flor et al., 1995; Marlowe, 1992, 1995), and visual ERP (Connolly, Gawel, & Rose, 1982) differences. These differences suggest a central sensitization for noxious stimulation. Similarly, fibromyalgia patients had significantly lower heat pain thresholds with higher amplitudes of middle- and long-latency laser-evoked potentials (Lorenz, Grasedyck, & Bromm, 1996). Using positron emission tomography, Derbyshire et al. (1994) found that patients with atypical facial pain showed increased anterior cingulate but decreased anterior frontal activation in comparison to controls.

Chronic pain patients, such as those with fibromyalgia, have increased attention and heightened sensitivity to internal and external noxious stimulation, be it pain or noise (McDermid, Rollman, & McCain, 1996; Rollman & Lautenbacher, 1993). Wickramasekera's (1993) high risk model suggests that highly hypnotizable persons may be more vulnerable to inadvertently learning to amplify pain perception. Crawford (1995a, 1995b) proposes that highly hypnotizable individuals might be more vulnerable to the development of chronic pain due to their absorptive attentional and imaginal abilities that may contribute to overreactivity to pain and possible enhanced conditionability. These assumptions are still speculative and go beyond the data that were presented. Our current research efforts are focused on the elucidation of these important questions about chronic pain states and their relationship to hypnotizability.

Overall General Conclusions

Hypnotic analgesia is an active process that requires inhibitory effort, dissociated from conscious awareness, where the anterior frontal cortex participates in a topographically specific inhibitory feedback circuit that cooperates in the allocation of thalamocortical activities. Hypnotic analgesia led to significant changes in somatosensory event-related potentials in chronic low back pain participants, most of whom were moderately to highly hypnotizable. The application of hypnotic analgesia techniques led to highly significant reductions in perceived sensory pain and distress to cold-pressor and noxious electrical stimulation. Enhanced positivity of potentials prestimulus and SERP changes poststimulus support the hypothesis that active inhibitory processes in the brain are involved in hypnotic analgesia.

Hypnotic analgesia is a powerful behavioral intervention that is effective in altering pain perception of both acute *and* chronic pain, particularly for the moderate to highly hypnotizable individual (e.g., Hilgard & Hilgard, 1994). Among persons with chronic low back pain, major reductions in reported low back pain occurred during the experimental sessions. In comparison to preexperimental baseline measures, there were significant improvements in the psychological well-being and sleep quality of the chronic pain participants across the three experimental sessions. The importance of developing self-efficacy through learning to control experimental pain and the understanding of one's own attentional and disattentional abilities was demonstrated as being a significant intervention in the modulation and control of chronic pain. The development of "neurosignatures of pain" can influence subsequent pain experiences (Coderre et al., 1993; Melzack, 1993) and may be expanded in size and easily reactivated (Flor & Birbaumer, 1994; Melzack, 1991, 1993). Therefore, hypnosis and other psychological interventions

need to be *introduced early* as adjuncts in medical treatments for onset pain before the development of chronic pain.

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Hypnotische Analgesie: 1. Somatosensorische Ereignis korrelierte Potentialveränderungen auf noxische Reize und 2. Transfer Lernen zur Reduktion von chronischen Rückenschmerzen

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Zusammenfassung: Fünfzehn Erwachsene, im Alter von 18 bis 43 Jahren (*M* = 29 Jahre), mit chronischen Schmerzen im unteren Rückenbereich (*M* = 4 Jahre), nahmen an dieser Studie teil. Alle bis auf einen waren mittel bis gut hypnotisierbar. Im ersten Teil wurden somatosensorische Ereignis korrelierte Potentiale einer noxischen elektrischen Stimulation während den Bedingungen Aufmerksamkeit und hypnotische Analgesie (HA) ausgewertet. Die Ableitungsorte waren dabei folgende Regionen: anterior frontal (Fp1, Fp2), midfrontal (F3, F4), zentral (C3, C4), und parietal (P3, P4). Während der HA wurde die erwartete inhibitorische Verarbeitung nur durch eine erhöhte N140 in der anterioren frontalen Region und durch ein vor dem Stimulus beginnendes, positiv sich fortsetzendes kontingentes kortikales Potential bei Fp1 nachgewiesen. Während der HA zeigte sich eine verminderte räumlich temporale Wahrnehmung anhand von reduzierten Amplituden der P200 (bilateral, midfrontal und zentral, und links parietal) und der P300 (rechts midfrontal und zentral). HA ist ein aktiver Prozeß, der einer inhibitorischen Anstrengung bedarf, die dissoziiert von bewußter Aufmerksamkeit ist, wobei der anteriore frontale Kortex an einer topographisch spezifischen inhibitorischen Feedback Schleife beteiligt ist, der in der Zuteilung von thalamokortikalen Aktivitäten mitwirkt. Im zweiten Teil dokumentieren die Autoren die Entwicklung von Selbst-Wirksamkeit der Teilnehmer, die sie durch den erfolgreichen Transfer von neu erlernten Fähigkeiten experimenteller Schmerzreduktion, auf die Reduktion ihrer eigenen chronischen Schmerzen leisteten. Über den Verlauf von drei experimentellen Sitzungen berichteten die Teilnehmer eine Reduktion des chronischen Schmerzes, eine Zunahme des psychischen Wohlbefindens und eine verbesserte Schlafqualität. Die Entwicklung von "Neurosignaturen des Schmerzes" kann zukünftige Schmerzerlebnisse beeinflussen,

und möglicherweise in ihrer Größe erweitert und leichter reaktiviert werden. Deshalb sollte die Hypnose und andere psychologische Interventionen früh als Zusatz zu einer medizinischen Behandlung bei beginnendem Schmerz eingesetzt werden, bevor es zur Entwicklung von chronischen Schmerzen kommen kann.

Analgésie hypnotique: 1. Changements dans les potentiels évoqués somatosensoriels reliés à des stimuli nociceptifs et 2. Apprentissage par transfert pour réduire la douleur lombaire chronique

Helen J. Crawford, Timothy Knebel, Lyla Kaplan, Jennifer M. C. Vendemia, Min Xie, Scott Jamison, et Karl H. Pribram

Résumé: Quinze adultes souffrant de douleur lombaire chronique (*M* = 4 ans), âgés entre 18 et 43 ans (*M* = 29 ans), ont participé à cette étude. Tous, sauf un, étaient de moyennement à fortement hypnotisables. Dans la partie 1, des potentiels évoqués somatosensoriels reliés à des stimulations électriques nociceptives ont été évalués durant des conditions d'attente et d'analgésie hypnotique (AH), au niveau frontal antérieur (Fp1, Fp2), mi-frontal (F3, F4), central (C3, C4), et pariétal (P3, P4). Durant l'AH, le processus d'inhibition anticipé a été illustré par une augmentation de l'activité N140 dans la région frontal antérieure et par un potentiel cortical positif pré stimulus sur Fp1 seulement. Durant l'AH, une diminution de la perception spatio temporelle a été révélée par des amplitudes réduites en P200 (bilatéral mi-frontal et central ainsi que pariétal gauche) et en P300 (mi-frontal droit et central). L'AH est un processus actif qui requiert un effort d'inhibition, dissocié de l'attention consciente, et pour lequel le contex frontal antérieur participe à un circuit de rétroaction inhibiteur topographiquement spécifique qui contribue à l'allocation des activités thalamocorticales. Dans la partie 2, les auteurs illustrent le développement de l'auto efficacité à travers un transfert réussi, par les participants, des nouvelles habiletés de contrôle de la douleur expérimentale à la diminution de leur douleur chronique. En trois sessions expérimentales, les participants ont rapporté une diminution de la douleur chronique, une augmentation du bien-être psychologique et une augmentation de la qualité de leur sommeil. Le développement des "neurosignatures de la douleur" peut influencer les expériences ultérieures de douleur. On peut en augmenter l'étendue et les réactiver facilement. Conséquemment, l'hypnose et les autres interventions psychologiques se doivent d'être introduites précocement comme des ajouts aux traitements médicaux, dans les débuts de la douleur, avant que celle-ci ne se développe de façon chronique.

Analgesia hipnótica: 1. Cambios en los potenciales evocados sensoriosomáticos por estímulos novicos 2. Transferencia de aprendizaje para la reducción del dolor crónico de la zona lumbar

Helen J. Crawford, Timothy Knebel, Lyla Kaplan, Jennifer M. C. Vendemia, Min Xie, Scott Jamison y Karl H. Pribram

Resumen: Participaron quince adultos con dolor crónico en la región lumbar (*M* = 4 años), entre los 18 y 43 años de edad (*M* = 29 años). Todos excepto uno

eran moderada a altamente hipnotizables. En la parte 1, los potenciales evocados sensoriosomáticos se correlacionaron con estimulación eléctrica nociva y fueron evaluados durante las condiciones de espera y de analgesia hipnótica (AH) en las zonas frontal anterior (Fp1, Fp2); frontal media (F3, F4); central (C3, C4); y parietal (P3, P4). Durante la AH, los procesos inhibitorios hipotetizados fueron evidenciados por el aumento a N140 de la zona anterior frontal y por un preestímulo positivo contingente al potencial cortical en Fp1 solamente. Durante la AH, se evidenció una disminución en la percepción espaciotemporal por la reducción de amplitudes de P200 (bilateral medio-frontal y central y parietal izquierdo) y P300 (medio frontal y central). La AH es un proceso activo que requiere un esfuerzo inhibitorio, disociado de la conciencia en donde participa la corteza frontal anterior, en un circuito topográficamente específico de inhibición retroalimentada que coopera en la asignación de actividades tálamocorticales. En la parte 2, los autores documentan el desarrollo de la eficacia propia a través de la transferencia exitosa de habilidades recientemente aprendidas de reducción experimental del dolor a la reducción del dolor crónico propio. Luego de tres sesiones experimentales, los participantes reportaron reducción del dolor crónico, aumento del sentimiento de bienestar y aumento de la calidad del sueño. El desarrollo de una "neuromatriz de dolor" puede influenciar experiencias posteriores de dolor, aumentar y reactivarse fácilmente. Por lo tanto, es necesario introducir tempranamente la hipnosis y otras intervenciones psicológicas como ayuda en los tratamientos médicos para el dolor, antes que se produzca la cronificación del dolor.

BOOK REVIEWS

SPIEGEL, HERBERT, & SPIEGEL, DAVID. *Trance and Treatment: Clinical Uses of Hypnosis* (paperback edition). Washington, DC: American Psychiatric Press, 1987. Pp. xiv + 382. $23.50 U.S.

The latest paperback reprint of *Trance and Treatment: Clinical Uses of Hypnosis* comes 16 years after its original publication (for review of the original, see Zinn, 1983). The book is based on the authors' extensive experience of clinical hypnosis, and the authors seek to provide clinicians with "a brief, disciplined technique for mobilizing and learning from an individual's ability to concentrate" (p. xi). Furthermore, they assess the scientific evidence for the clinical uses and limitations of hypnosis.

The book is divided into four sections, and its structure is intended to parallel the sequence of treatment in an encounter with a client: Section I defines and discusses hypnosis; Section II presents the Hypnotic Induction Profile (HIP), a 10-minute clinical assessment procedure; Section III offers hypotheses and data relating performance on the HIP to personality style and psychopathology; and Section IV explores the construction of a treatment strategy employing hypnosis and discusses a series of specific treatment strategies and clinical cases. In addition, the book advises on selecting the most appropriate treatment based on an assessment of hypnotizability, and it focuses on psychotherapy with highly hypnotizable individuals.

The HIP is a frequently used assessment tool and a controversial one in terms of what it is actually measuring. It includes a measurement of "eye roll" and a suggestion for arm levitation; response to this item is assessed by a number of posthypnotic subjective measures. It can be argued that the subjective nature of scoring the eye roll sign and the low difficulty level of the arm levitation item (about 90% of individuals pass) make the HIP a poor indicator of hypnotizability. Studies have found that the eye roll sign correlates poorly, and the score based on the arm levitation measures correlates only moderately, with the Stanford scales. Given recent discussions of the possibility of dual mechanisms driving hypnotic performance (specifically, compliance or nonhypnotic suggestibility for easy items, and cognitive abilities, such as absorption and imaginative involvement, for difficult items), and given the HIP's reliance on one very easy item in comparison with the Stanford scales' broad band of items, the HIP could be said to be measuring compliance rather than hypnotizability. It is worth discriminating, as Spiegel and Spiegel

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