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# THE NATURE OF HYPNOTIC ANALGESIA: NEUROPHYSIOLOGICAL FOUNDATION AND EVIDENCE

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## Abstract

This paper reviews neurophysiological research (EEG, event-related potential, regional cerebral blood flow, neurochemical) that supports the proposal that hypnotic analgesia is an active inhibitory process involving several brain systems mediating attentional and nociceptive processes. Even though the processes of hypnotic analgesia may be dissociated from conscious awareness and appear to be out of volitional control, it is proposed that hypnotic analgesia depends on the activation of a supervisory, attention control system – involving the anterior frontal cortex – which then participates with other cortical and subcortical systems in the allocation of thalamocortical activities. Hypnotic analgesia affects the active allocation of attention and disattention associated with the anterior frontal region, as well as spatio-temporal aspects of pain perception associated with the posterior cortical systems.

**Key words**: hypnosis, hypnotic analgesia, pain, somatosensory evoked potentials, electroencephalography, cerebral blood flow

Hypnosis is one of the best documented behavioural interventions for controlling acute and chronic pain in adults and children (for reviews, see Barber, 1996; Barber and Adrian, 1982; Chaves, 1989, 1994; Crawford, 1994a,b; 1995, in press; Evans, 1988; Gibson, 1994; Hilgard and Hilgard, 1994; Hilgard and LeBaron, 1984; Holroyd, 1996; Olness and Gardner, 1988). Hypnotically suggested analgesia involves techniques of distraction, redefinition and dissociation to reduce or eliminate the perception of pain. The positive correlation between standardized hypnotic susceptibility test scores and the degree of reduced pain obtained following hypnotically suggested analgesia is significant, usually around 0.5. Low hypnotizable persons ('lows') can reduce the distress of accompanying pain due probably to the relaxation component of hypnosis. Whereas, moderately to highly hypnotizable persons ('highs') are more likely to reduce – and even eliminate – both the distress and the sensory dimensions of pain due to relaxation *and* attentional/disattentional (inhibitory) processes (Crawford, 1994a,b; Hilgard and Hilgard, 1994). Further acceptance of hypnotic analgesia as an early intervention, in conjunction with other medical interventions, for the treatment of acute as well as chronic pain may occur by an understanding of its neurophysiological underpinnings.

The purpose of the present paper is to review what we now know about the neurophysiological bases of hypnotic analgesia as moderated by hypnotic susceptibility level, in conjunction with other papers given at the CIBA symposium on 'The Nature of Hypnosis' (and found in this special issue of *Contemporary Hypnosis*). It is placed

within an evolving model that proposes (1) hypnotic analgesia depends upon the activation of a supervisory, attention control system that is involved in the suppression of incoming painful stimuli at cortical and subcortical levels, (2) highly hypnotizable persons can better control pain because of their more effective frontal attentional system that permits them to either attend to or disattend incoming stimuli (for reviews, see Crawford, 1994a,b, in press; Crawford and Gruzelier, 1992). This paper may be considered a midstream report, much like Crawford and Gruzelier's (1992) general review on the neuropsychophysiology of hypnosis.

## Neurophysiology of pain

Pain is a dynamic, multifaceted experience involving sensory-discriminative, motivational-affective, cognitive and motoric dimensions (e.g. Melzack, 1992; Melzack and Casey, 1968; Melzack and Wall, 1965; Price, 1988). Neuroimaging studies support the view that any sensory event engages an extensive network of brain systems working both sequentially and in parallel (Posner and Raichle, 1994). Pain is no exception. The physical significance, intensity, duration, somatic involvement, location, overall unpleasantness of pain is profoundly influenced by a complex array of environmental, social, emotional and situational variables synthesized through neural activation of cortical and subcortical systems (Chudler and Dong, 1995). The sensory aspects of pain are associated with processes in the central and posterior brain regions, while the distress aspects of pain are associated with anterior regions (Pribram, 1991).

During experimental pain, various regions of the brain are often activated, including primary (S1) and secondary (S2) somatosensory cortex (e.g. Howland et al., 1995), anterior cingulate cortex (e.g. Davis et al., 1995; Devinsky et al., 1995; Jones et al., 1991; Sweet, 1995; Talbot et al., 1991), basal ganglia (Chundler and Dong, 1995), anterior frontal cortex (e.g. Bromm and Chen, 1995; Derbyshire et al., 1994, 1996).

There are several descending inhibitory systems that modulate sensory input, including higher centres (e.g. anterior frontal cortex), the midbrain (e.g. hypothalamus), the periaqueductal gray area (Depaulis and Bandler, 1991; Franzén and Ahlquist, 1996), the spinal cord (for review, see Markenson, 1996). Phylogenetically speaking, regions of the anterior frontal cortex are relatively recent, and in humans these regions have been widely associated with programming and regulating behaviour including attention and inhibitory processes (e.g. Luria, 1966; Norman and Shallice, 1986; Pribram, 1991; Stuss et al., 1995). Since hypnotic analgesia is attention-based, we would anticipate the interactive involvement of both the attentional and the pain systems of the brain.

## Neurophysiology of hypnotic analgesia

The search for neurophysiological markers or changes during hypnotic analgesia has occurred for the past 30–40 years. The 1990's 'Decade of the Brain' has brought new, sophisticated techniques and theories that are only recently being applied to the study of pain processing and the manipulation of attentional and pain processes through hypnotic analgesia. New technologies available to us include electroencephalographic (EEG) frequency analysis, EEG topographic brain mapping, somatosensory event-related potential (SERP) topographic brain mapping, positron emission tomography (PET), regional cerebral blood flow (rCBF), single photon emission computed tomography (SPECT), functional Magnetic Resonance Imaging (fMRI).

As this research becomes more interdisciplinary in nature, researchers must continue to be vigilant to ensure that they incorporate adequate research designs developed within the field of hypnosis (Barabasz and Barabasz, 1992; Crawford and Gruzelier, 1992; Hilgard, 1965). It is imperative to use persons who have been well-screened and categorized using standardized hypnotic susceptibility scales, adequate comparisons of attention to the pain ('attend') and hypnotic analgesia conditions that are counterbalanced or placed within an A-B-A design. The studying of Aptitude (trait) X Treatment interactions, through a combination of experimental and correlational methods (Crawford and Allen, 1983; Cronbach; 1975; Tellegen, 1981) is crucial in hypnosis research. Hypnotic susceptibility level is important to evaluate since it appears that highly hypnotizable persons can better control pain because of their more effective frontal attentional system that permits them to either attend to or disattend incoming stimuli (e.g. Crawford et al., 1993; for reviews, see Crawford, 1994a,b, in press; Crawford and Gruzelier, 1992). Comparisons between low and highly hypnotizable persons is crucial to sort out the effects of hypnotic analgesia per se from more general relaxation and other effects. The highly hypnotizable person should have been screened for inclusion based upon previously demonstrated abilities to greatly reduce or eliminate the perception of distress and sensory pain.

Anterior frontal (prefrontal) cortex activation occurs in response to pain. We propose it is also involved in gating or inhibiting somatosensory input when hypnotic analgesia is employed. The anterior frontal cortex is activated during directed attention *and* inhibition (e.g. Posner and Petersen, 1990), and operates at early stages of sensory processing on both cortical and subcortical structures from 'the periphery through dorsal column nuclei and thalamus to the sensory cortex' (Yamaguchi and Knight, 1990, p. 281). Surprisingly, on-going chronic pain may actually disrupt anterior frontal activation (Derbyshire et al., 1994, 1996). If that is the case, then hypnotic analgesia techniques may activate inhibitory processing of the anterior frontal cortex and thereby result in downward inhibition of incoming pain and pain memories. Recent studies of regional cerebral blood flow and brain wave electrical activity (EEG and somatosensory event-related potentials at surface and subcortical region recording sites) support the proposal that the anterior frontal attention system is, through interaction with other cortical and subcortical regions, actively involved in suppressing incoming information about noxious stimuli during hypnotic analgesia. These studies will now be reviewed.

### *Regional cerebral blood flow*

Changes in rCBF metabolism, as measured by the 133-xenon method, accompanying rest, ischemic pain without suggested analgesia, ischemic pain with suggested analgesia conditions in sessions with and without hypnosis were investigated by Crawford, Gur, Skolnick, Gur and Benson (1993). Participants were healthy young men: six were very low in hypnotizability (as measured by three standardized scales of hypnotic susceptibility) and five were very high in hypnotizability. Training in applying techniques of hypnotic analgesia to both cold pressor pain and ischemic pain preceded the rCBF study. Those highs who participated had consistently eliminated all pain perception in these training sessions. No rCBF differences were observed during waking, but following a hypnotic induction only the highs showed a dramatic increase in rCBF, ranging from 13% to 28% during rest, that may reflect increased cognitive effort or arousal during hypnosis.

During hypnotic analgesia, only highly hypnotizable persons showed increased bilateral regional cerebral blood flow (rCBF) activation in anterior frontal cortex as

well as the somatosensory cortex (Crawford et al., 1993). We have interpreted such increased anterior frontal rCBF to reflect greater inhibitory processing occurring in these regions during hypnotic analgesia. This supports the view that hypnotic analgesia involves the supervisory, attentional control system (Hilgard, 1986) of the anterior frontal cortex 'in a topographically specific inhibitory feedback circuit that cooperates in the regulation of thalamocortical activities (e.g. Birbaumer et al., 1990)' (Crawford, 1998, pp. 112–113). The increased blood flow in the somatosensory region is difficult to interpret in light of recent conflicting findings. While the somatosensory cortex is assumed to be involved in pain processing, cerebral blood flow studies have reported increased blood flow (e.g. Casey et al., 1994; Coghill et al., 1994), no changes (Derbyshire et al., 1994; Jones et al., 1991), or decreased activity (Apkarian et al., 1992).

*Somatosensory event-related potentials*

Scalp-recorded somatosensory event-related potentials (SERPs) show significant changes in mid to late components (after 100 msec) in response to unpleasant cutaneous stimulation during hypnotic analgesia (e.g. Arendt-Nielsen et al., 1990; Crawford, 1994a; De Pascalis et al., 1992; Mészáros and Bányai, 1978; Mészáros et al., 1978; Sharev and Tal, 1989; Spiegel et al., 1989; Zachariae and Bjerring, 1994; for reviews, see Crawford, 1994a,b, in press; Crawford and Gruzelier, 1992, Spiegel, 1996; Crawford et al., 1998) in mid-frontal, central, parietal, and/or occipital regions. None of these studies, with the exception of Crawford et al. (1998), evaluated SERP changes in the anterior frontal region during hypnotic analgesia, yet recent neurophysiological models of attention and pain point to the importance of evaluating anterior frontal activity during hypnotic analgesia processes.

Our research has been expanded to include anterior frontal cerebral dynamics as well as more posterior regions by evaluating SERP activity in (1) scalp-recorded temporal anterior frontal, mid frontal, central and parietal regions in participants drawn from two populations: healthy young adults (Crawford et al., 1997; in preparation) and adults with chronic low back pain (Crawford et al., 1998); and (2) temporarily implanted intracranial electrodes in the anterior cingulate and anterior temporal cortex, as well as other areas (Kropotov et al., 1997).

In this review, an emphasis is placed upon findings from our recent study (Crawford et al., 1998) of 17 men and women, aged 19–43 years, with chronic low back pain. On the Stanford Hypnotic Susceptibility Scale Form C (SHSS:C; Weitzenhoffer and Hilgard, 1962) the participants were found to be all moderately to highly hypnotizable except for one low hypnotizable man.

Three experimental sessions occurred approximately one week apart. The first session involved the assessment of hypnotic susceptibility and pain control training, the second involved SERP assessment to noxious electrical stimuli during attention and hypnotic analgesia conditions, the third involved EEG assessment to cold pressor pain during attention and hypnotic analgesia conditions. At the first session, following the SHSS:C, participants were taught hypnotic analgesia techniques to control experimentally produced pain. Participants dipped their left hand into water and ice for 60 s immersions ('cold pressor test'). By the third hypnotic analgesia dip, 60% had completely eliminated all pain perception and 80% had completely eliminated all distress perception. In comparison to unselected college students, these results are of a surprising magnitude and raise important research questions whether adults with chronic pain are more hypnotizable than the general population. If they are hypnotizable, then hypnotic analgesia techniques should be quite suitable for assisting in the control of their own pain (Crawford et al., 1998).

The development of self-efficacy was documented across the sessions through the successful transfer of newly learned skills from the reduction of experimental pain to the reduction of their own chronic pain. Overall the participants reported significant chronic pain reduction (McGill Pain Questionnaire), increased psychological well-being (Beck Depression Inventory; SCL-90-R) and increased sleep quality (fewer minutes to fall asleep).

In the second experimental session, the participants had their SERPs recorded at the scalp while 30 noxious electrical stimuli were administered to the left middle finger during conditions of waking and hypnosis. Reported below are the results of the within-subjects, A-B-A design following a hypnotic induction in which blocks of stimuli were presented in conditions of attend, hypnotic analgesia, attend. The two attend conditions were averaged and compared to the hypnotic analgesia condition.

For the adults with chronic low back pain, during hypnotic analgesia, hypothesized inhibitory processing was implicated by an enhanced N140 in the anterior frontal (Fp1, Fp2) region. This component is thought to reflect the 'complex reciprocal interactions between posterior and prefrontal [anterior frontal] cortex and subcortical structures' that play 'a key role in governing sequential attention processes' (Desmedt and Tomberg, 1989, p. 343). Additionally, during hypnotic analgesia, a greater N250 negativity was observed in the fronto-central region. These findings suggest an 'active disattention during hypnotic analgesia, rather than the normally increasing spotlighted attention towards relevant incoming sensory signals' (Crawford et al., 1998, p. 111).

Reductions of perceived intensity of pain during hypnotic analgesia were accompanied by reduced P200 amplitudes in the midfrontal, central and left parietal regions, and by reduced P300 amplitudes in the right midfrontal and central regions. These findings may be interpreted as suggesting the reduced involvement of the parietal cortex. One of the functions of the parietal cortex is to determine positions of body parts by organizing sensory inputs received from the somatosensory and anterior frontal cortex (Desmedt and Tomberg, 1989).

Our laboratory is the first to examine changes in the contingent variations, which are related to arousal and attention (Tecce, 1972; Tecce and Cattanach, 1982) in the pre-stimulus SERP period prior to the regularly occurring electrical stimulus that occurred every three seconds for 30 trials. Much work has centred around the CNV (contingent negative variation), hypothesized to reveal enhanced cortical excitability enabling a preparatory state. Additionally, there is a positive-moving contingent variation that may result from inhibitory processing and 'a "disfacilitation" in cortical neuronal networks' (Rockstroh et al., 1993, p. 236; for review, see Birbaumer et al., 1990). If hypnotic analgesia involves inhibitory processing, then such a positive-moving contingent variation may be present.

Among the adults with chronic low back pain, we observed additional evidence for the occurrence of inhibitory processing during hypnotic analgesia: a positive-going slope of the prestimulus contingent cortical potential only in the left anterior frontal region. This may reflect a lowering of cortical activity (Birbaumer et al., 1990; Rockstroh et al., 1993) and increased inhibitory processing (Tecce, 1972; Tecce and Cattanach, 1972). The asymmetry is strong evidence against eye movement artefact. Gruzelier (Gruzelier, 1988; Crawford and Gruzelier, 1992) suggests a decreased involvement of the left anterior region during hypnotic induction and certain hypnotic phenomena.

Kropotov et al. (1997) ) are the first to demonstrate the possible involvement of the anterior cingulate and the anterior temporal cortex in hypnotically suggested

analgesia. They recorded intracranial SERPs to 30 painful cutaneous stimuli in conditions of attend, hypnotic analgesia, and attend in two female patients with obsessive-compulsive disorder bearing multiple temporarily implanted intracranial electrodes. These electrodes were located in the anterior cingulate cortex, amygdala, temporal cortex, and parietal cortex. Hypnotically suggested analgesia led to a successful reduction of self-reported pain perception and accompanying SERP intracranial changes in the hypnotically responsive patient, but not in the non-hypnotically responsive patient. As shown in Figure 1, reduced pain perception to noxious electrical stimuli to the right middle finger was accompanied by (a) a significant reduction of the positive SERP component within the range of 140–160 msec poststimulus recorded in the left anterior cingulate cortex, (b) a significant enhancement of the negative SERP component 200–260 msec poststimulus recorded in the left anterior temporal cortex. No significant changes were observed in other cortical regions or at the scalp-recorded Fz.

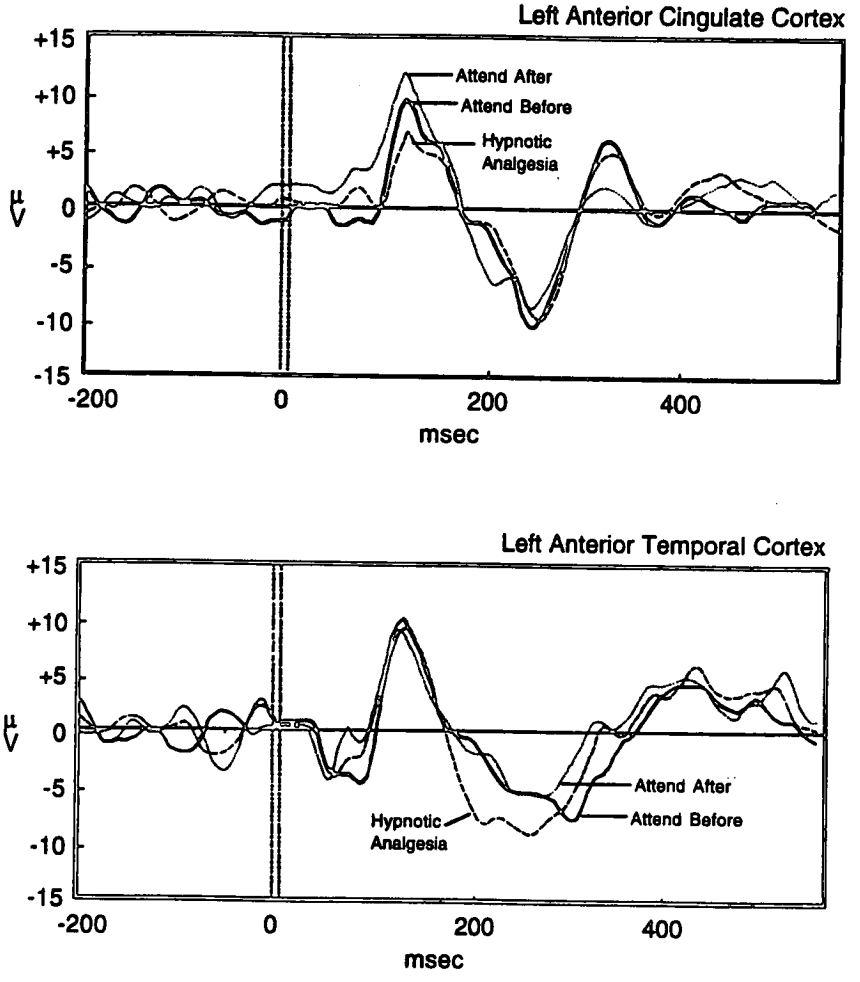


Figure 1. In a hypnotizable patient, somatosensory event-related potentials at two intracranial sites, averaged across 30 trials during conditions of attend, hypnotically suggested analgesia and attend. Note the reduction in the positive component at 140–160 msec recorded in the left anterior cingulate (upper) and the increase in the negative component at 200–260 msec recorded in the left anterior temporal cortex (lower). With permission, reprinted from Kropotov et al. (1997, p. 5).

These findings are consistent with recent research (e.g. Bromm and Chen, 1995; Derbyshire et al., 1996; Talbot et al., 1991), which implicates the anterior cingulate cortex's involvement in pain processing and the organization of responses to emotional and noxious stimuli (Devinsky et al., 1995). We also interpret these findings to be 'indicative of increased active and controlled inhibitory processing of the incoming painful stimuli . . . [and] further evidence that hypnotic analgesia is analogous to a No-Go condition of response inhibition' (Kropotov et al., 1997, p. 6).

In sum, somatosensory event-related research strongly demonstrates that active inhibitory processes in the brain are involved in hypnotic analgesia. Future work will include evaluating earlier SERP components as well as localizing their sources in cortical and subcortical structures.

*EEG activity*

Further evidence of inhibitory processes occurring during hypnotic analgesia may be gleaned from studies of EEG activity. Most hypnosis studies have evaluated EEG activity during the administration of ongoing tonic pain, usually cold pressor pain. Stimulus-bound EEG activity following phasic painful stimuli has yet to be addressed.

In a patient undergoing dental surgery with hypnosis as the sole anesthetic, total EEG power decreased with a greater diminution in the left hemisphere in alpha and theta EEG bands (Chen et al., 1981). Karlin, Morgan, Goldstein (1980) reported hemispheric shifts in total EEG power during hypnotic analgesia to cold pressor pain that were interpreted as greater overall right-hemisphere involvement at the bipolar parieto-occipital derivation.

Crawford (1990) evaluated the accompanying EEG during 60 s immersions of the left hand into ice cold water during conditions of attend and hypnotic analgesia. Of particular interest were interactions between condition and hypnotic level in the high theta (5.5–7.5 Hz) frequency band. First, as has been shown often in the literature (e.g. Sabourin et al., 1990; for a review, see Crawford and Gruzelier, 1992), highs generated significantly more high theta power than did lows at mid frontal (F3, F4), temporal (T3, T4), parietal (P3, P4) and occipital (O1, O2) sites. This theta activity is thought to possibly originate in the hippocampal region and be associated with focused attention, thus reflective of the more effective attention system observed in highly hypnotizable persons (for review, see Crawford and Gruzelier, 1992). The low hypnotizables showed no significant hemispheric asymmetries, while the high hypnotizables did. In the temporal region (T3, T4), when concentrating on the pain the highs were significantly more left hemisphere dominant in high theta power. By contrast, during hypnotic analgesia the highs showed a significant shift to less left hemispheric theta power and greater right hemisphere theta power.

Using phasic pain consisting of electric shocks to the left wrist, De Pascalis and Perrone (1996) assessed ongoing bilateral EEG activity recorded from mid-frontal, central and posterior regions during waking, hypnosis with no analgesia, hypnosis with analgesia in low and high hypnotizables. They did not evaluate the stimulus-bound EEG following each painful stimulus, although the SERPs were evaluated in a separate publication (De Pascalis et al., 1992). During hypnotic analgesia, highs showed reductions of total power, delta (0.5–3.75 Hz) and beta 1 (13.0–15.75 Hz) in the right hemisphere, which was accompanied by a decrease in sympathetic activity level as evidenced by heart period variability. De Pascalis and Perrone found no differences in high theta (6.0–7.5 Hz) at recorded sites, but it should be noted that they did not record from the temporal region where Crawford (1990) observed high theta hemispheric shifts. In both attend and analgesia conditions during hypnosis, highs

evidenced a reduction of low theta (4.0–5.75 Hz) with similar levels of activity in both hemispheres, whereas in waking there was greater low theta present in the left hemisphere. The reduction of low theta during hypnosis suggests greater cortical arousal or attention during hypnosis. This provides further support to Crawford et al.'s (1993) previously discussed proposal that hypnosis requires cognitive effort.

### *Other neurophysiological changes during hypnotic analgesia*

Hypnotic analgesia apparently has an inhibitory effect on peripheral spinal reflex activity (for review, see Price, 1996). Motor-neuron excitability, as measured by the Hoffman reflex amplitude, was decreased significantly during hypnosis in high but not low hypnotizables, yet suggested analgesia had no effect (Santarcangelo et al., 1989). Reductions in brief latency (Hagbarth and Finer, 1963) and R-III amplitude (Kiernan et al., 1995) of spinal reflexes were observed during hypnotic analgesia.

Since some inhibitory processes operate via endogenous opioid peptides, several studies assessed the potential effect of hypnotic analgesia on the opiate descending control mechanism. Unlike opiate analgesia, hypnotic analgesia has generally not been reversed by the opiate antagonist naloxone hydrochloride (Barber and Mayer, 1977; Goldstein and Hilgard, 1988; Spiegel and Albert, 1983; but see Stevenson, 1978) except under circumstances of stress (Frid and Singer, 1978). Preliminary studies (e.g. Domangue et al., 1985; Sternbach, 1982) suggest other neurochemical processes may be involved in hypnotic analgesia. The mean plasma level of beta-endorphin-immunoreactivity was enhanced after hypnotic analgesia in arthritic patients, while no changes in plasma levels of epinephrine, dopamine or serotonin occurred (Domangue et al., 1985). The surprising paucity of research in this area leaves it open to exciting explorations.

## Summary

The research reviewed here supports the view that hypnotic analgesia is an active inhibitory process that involves several attentional and pain systems in the brain. Even though the processes may be dissociated from conscious awareness and appear to be out of volitional control, we propose that hypnotic analgesia depends upon the activation of a supervisory, attention control system – involving the anterior frontal cortex – which then participates with other cortical and subcortical processes in the allocation of thalamocortical activities. Hypnotic analgesia is the result of changes which affect the active allocation of attention and disattention associated with the anterior frontal region, as well as spatio-temporal aspects of the perception of pain associated with the posterior cortical systems.

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