

The Neurophysiology of Pain Perception and Hypnotic Analgesia: Implications for Clinical Practice

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Abstract

Although there remains much to be learned, a great deal is now known about the neurophysiological processes involved in the experience of pain. Research confirms that there is no single focal “center” in the brain responsible for the experience of pain. Rather, pain is the end product of a number of integrated networks that involve activity at multiple cortical and subcortical sites. Our current knowledge about the neurophysiological mechanisms of pain has important implications for understanding the mechanisms underlying the effects of hypnotic analgesia treatments, as well as for improving clinical practice. This article is written for the clinician who uses hypnotic interventions for pain management. It begins with an overview of what is known about the neurophysiological basis of pain and hypnotic analgesia, and then discusses how clinicians can use this knowledge for (1) organizing the types of suggestions that can be used when providing hypnotic treatment, and (2) maximizing the efficacy of hypnotic interventions in clients presenting with pain problems.

Keywords: Hypnotic analgesia, pain, hypnosis, neurophysiology.

A preponderance of evidence indicates that hypnotic analgesia can significantly and substantially reduce pain associated with both acute and chronic pain conditions (Jensen & Patterson, 2006; Montgomery, DuHamel & Redd, 2000; Patterson & Jensen, 2003). However, clinicians who use hypnotic interventions to treat individuals with pain use a large variety of hypnotic approaches and suggestions, varying from quite focused suggestions for reducing or blocking the experience of pain sensations (e.g., Crasilneck, 1979) to more general and diffuse suggestions to have experiences inconsistent with pain and suffering, such as the vivid reliving of pleasurable autobiographical experiences (Faymonville, M. E., et al., 2000). Often clinicians report that with any one patient they provide just one or two of the many

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possible suggestions that might be effective for pain management (Evans, 1989; Gainer, 1992; James, Large, & Beale, 1989), although it is not always clear why the particular suggestions used were chosen over others. There is also a tendency for clinicians to focus on suggestions that target the sensory components of pain, as opposed to using or including suggestions that address pain's emotional, motivational, cognitive, and behavioral components (Crasilneck, 1995; Evans, 1989; Gainer, 1992; Jack, 1999; Lu, Lu, & Kleinman, 2001; Simon & Lewis, 2000; Williamson, 2004; see Abrahamsen, Baad-Hansen, & Svensson, in press, and Sachs, Feuerstein, & Vitale, 1977, for descriptions of more comprehensive hypnotic analgesia treatment approaches).

During the past decade, there has been an explosion of knowledge about the neurophysiological basis of pain and hypnotic analgesia. This knowledge has important implications for understanding how hypnosis might be most effectively applied to clinical pain problems. The purpose of this review article is to provide a brief overview of the current state-of-the-science knowledge concerning the neurophysiological basis of pain and hypnotic pain management, and to then discuss the implications for this knowledge for organizing and expanding the types of hypnotic suggestions that might be considered when providing hypnotic interventions for pain management.

Mechanisms Involved in Pain Perception and Regulation

In the 1600s, the French philosopher René Descartes argued that pain was a simple reflexive response to physical damage. In his model, information about physical damage detected by sensors in the skin is transmitted directly through a single channel to a “pain center” in the brain. Descartes viewed the brain as a passive recipient of sensory information; “real” pain was thought to be entirely, or at least mostly, related to the amount of physical damage that existed outside of the nervous system in the peripheral tissue. Descartes model of pain (called the ‘specificity theory’) remained the generally accepted view of scientists and health care providers for the next 300 years.

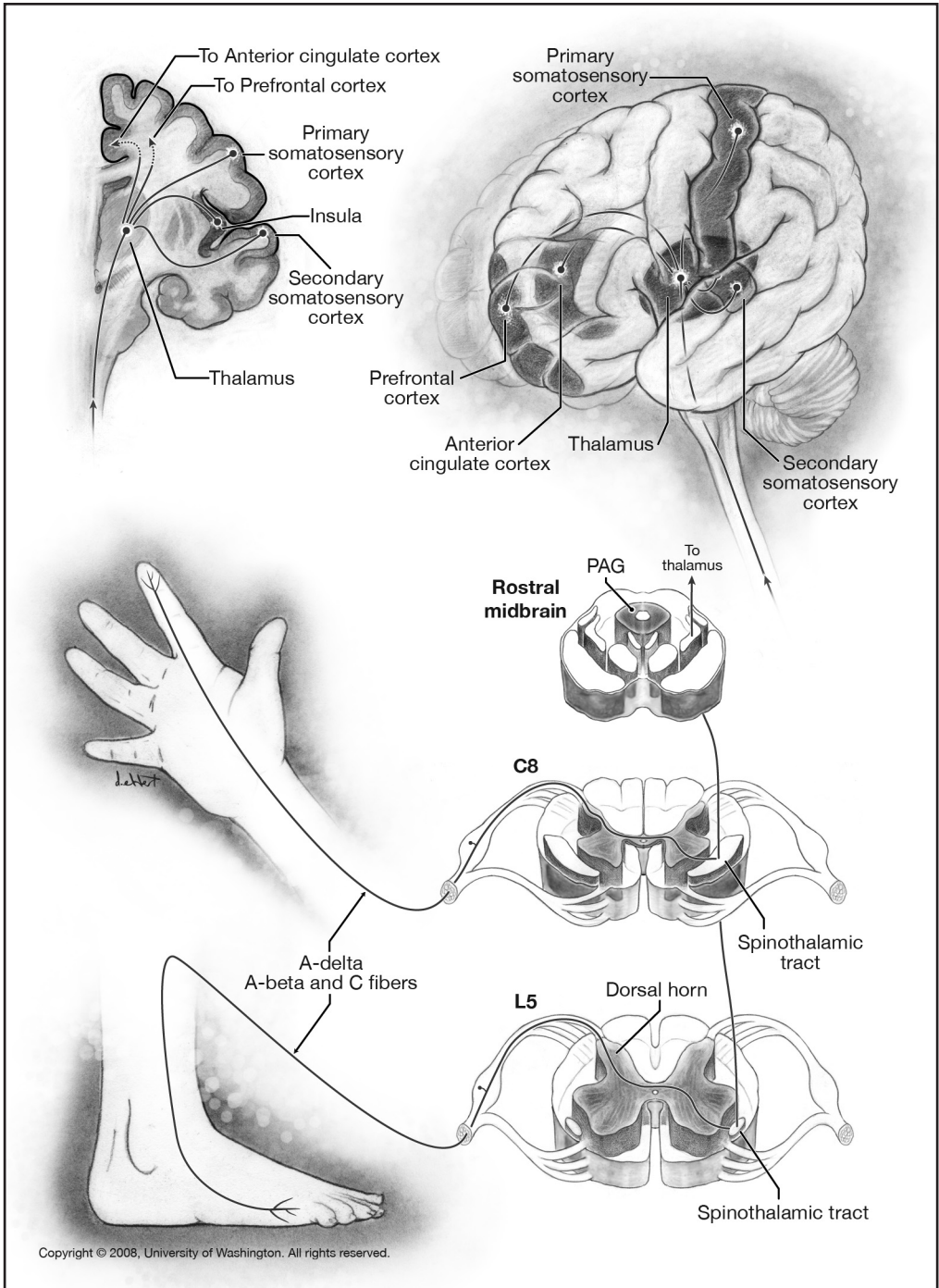
Beginning, perhaps with the publication of the gate control theory of pain in 1965 (Melzack & Wall, 1965), but particularly during the past decade, pain researchers have shifted at least some of their attention away from the periphery and have focused more on activity in the spinal cord and the brain. As a result, we now know that the brain is *not* merely a passive recipient of the nociceptive information it receives. Rather, the spinal cord and brain actively process and modulate that information in multiple areas. There is no “pain center” in the brain. Instead, multiple integrated pain networks work together to contribute to the global experience of pain.

Although it can be argued that the brain is the final common pathway to the experience of pain (“no brain – no pain”), the neurophysiological processes that underlie the experience of pain have peripheral (outside of the spinal cord; for example, in the limbs), spinal, and supraspinal (above the spinal cord; that is, in the brain stem and brain) nervous system components. This section provides a brief overview of the primary neurophysiological structures and mechanisms involved in pain perception. However, it should be kept in mind that what follows is a very general overview; the reader interested in greater detail should read several of the many excellent reviews and texts on this topic that were used as primary sources for the material presented in this section (Apkarian, Bushnell, Treede, Zubieta, 2005; Byers & Bonica, 2001; Craig, 2003; DeLeo, 2006; Rainville, 2002; Terman & Bonica, 2001).

Peripheral Mechanisms

All bodily tissues are innervated by receptors that respond to physical injury. These receptors are classified as a function of the type of nerve fibers they connect to. The nerve fibers that transmit most of the information about physical damage are the thin (thin because they do not have a myelin sheath covering them, which also makes them transmit information more slowly) C fibers and thicker (myelinated, therefore faster) A-delta fibers. A third type of fiber, A-beta, that normally carry information related to touch, can also transmit information that contributes to the experience of pain (see Figure 1).

Figure 1: The primary nervous system structures involved in the processing and experience of pain



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The stimulation or damage that excites these receptors, and the information about this stimulation transmitted along the C, A-delta, and A-beta fibers, is not pain. Pain does not occur until structures in the brain become activated and involved. Rather, the information from nerve receptors that communicate physical damage or the potential for physical damage is called *nociception* (from the Latin, *nocere*, which means harm or injury); the receptors that trigger this information are called *nociceptors*. Nociceptors can be more or less sensitized, and therefore respond differently at different times to the same amount of stimulation or damage. The mechanical and chemical changes that sensitize or inhibit nociceptors are exceedingly complex, and can change as a function of many factors.

Spinal Mechanisms

The fibers that communicate nociceptive information from the periphery to the central nervous system, including C, A-delta, and A-beta fibers, enter the spine at the dorsal horn (see Figure 1). Here, they terminate and then synapse with the dendrites and neurons of the spinothalamic tract (STT). The STT is the most important (but not only) pathway for the transmission of nociception to the brain. The relative responsiveness of STT cells is influenced by activity coming down to the dorsal horn from supraspinal sites. Some of the first clear evidence for descending inhibition came from research demonstrating that electrical stimulation of the periaqueductal gray (PAG) area in the midbrain resulted in significant analgesia at different sites in the body. Moreover, the PAG itself receives significant input from various sites in the brain, including the insula, the anterior cingulate cortex, and the sensory cortex; all areas known to be involved in the processing of pain (see next section).

Supraspinal (Above the Spine) Mechanisms

Although the discussion so far has focused on peripheral and spinal mechanisms of nociception, it is important to keep in mind that the activation of these mechanisms is neither necessary nor sufficient to produce the perception of pain. *Pain is perceived when complex integrated cortical (supraspinal) systems are engaged* with or without the presence of nociception; and pain can be relieved when these same systems are disengaged or interrupted.

A number of supraspinal sites have been shown to be involved in the perception of pain, but the most consistent areas that have been identified across different imaging studies are the thalamus, the primary and secondary somatosensory cortex (S1 and S2), the anterior cingulate cortex, the insula, and the prefrontal cortex (see Figure 1; see also Apkarian, Bushnell, Treede, & Zubieta, 2005). These brain areas and structures work closely together and with other CNS structures in an integrated fashion to produce the experience we label as pain.

Thalamus

The thalamus is located just above the brain stem. It can be considered the primary relay center for transmitting sensory information from the periphery and spinal cord to various sites in the cortex. The STT neurons that originate in the dorsal horns of the spinal cord terminate in a number of different areas of the thalamus, each of which then projects further to various cortical structures. Neurons from one of these areas project to the primary sensory cortex (S1), the posterior parietal cortex, and the secondary sensory cortex (S2). Neurons from other areas project, separately, to (1) the insula, (2) the anterior cingulate cortex, and (3) the prefrontal cortex. Yet another area in the thalamus where STT cells terminate projects diffusely to a number of sites of the brain *other* than the cerebral cortex. Electrical stimulation of these nuclei has clear (indirect) cortical effects, primarily producing a general activation. In short, cells in the thalamus that receive nociception information via the STT project directly too many different areas in the cortex.

Somatosensory cortex

The somatosensory cortex is divided into primary (S1) and secondary (S2) areas. S1 cortex lies in strip on the surface of the brain just behind the motor cortex, and the neurons in the S1 cortex are organized in such a way that each area of the body represented in distinct and well-defined loci within the S1. The S2 cortex lies at the base of the S1 cortex in the parietal lobe. The number of S2 neurons that respond to noxious stimulation via the thalamus appears to be relatively small. Nevertheless, S2 neurons (along with neurons in the insula) are among the first in the cortex to receive nociceptive input (Apkarian, Bushnell, Treede, & Zubieta, 2005). It is thought that S1 and S2 cortex both encode spatial information about nociception (that is, they help to tell us *where* on the body damage has or might have occurred), and that the S2 cortex, perhaps more than S1, is involved in encoding the severity and quality of the stimulus/nociception (Chudler & Bonica, 2001; May, 2007; Miltner & Weiss, 1998).

Anterior cingulate cortex

The anterior cingulate cortex (ACC) lies in the front part of the cingulate cortex, which itself lies just above the corpus colossus in the fissure of cortical tissue that separates the brain's two hemispheres. The ACC is one of the structures of the limbic system, and is related to a large number of processes and activities. Evidence supports the conclusion that pain-related activity in the ACC is related to the affective/emotional component of pain (Apkarian, Bushnell, Treede, & Zubieta, 2005). Findings also suggest that pain-related ACC activity subserves the motivational-motor aspects of pain (i.e., getting ready to do something about the pain), including the facilitation of cognitive, behavioral and emotional coping efforts (Rainville, 2002; Craig, 2003).

Insula

Although the insula is part of the neocortex (the surface of the brain), it lies deeply inside a fold (the Sylvian fissure) of the brain, near the sensory cortex. Like the ACC, the insula is a component of the limbic system. Craig (in press) has argued that the insula is primarily the sensory component of the limbic system, and is responsible for encoding a person's sense of his or her physical condition across a number of domains as they relate to motivation (the extent to which we "feel" thirsty, hungry, pain, or itch, versus feel satisfied and physically content). When discrepancies exist between (1) what the brain is hardwired to know the body needs for survival (oxygen, food, physical integrity) and (2) what the brain perceives (a lack of oxygen, low blood sugar, pain), alarm bells ring; and the insula may be largely responsible for determining when (and how loud) those bells should ring.

Prefrontal cortex

The prefrontal cortex lies on the front of the frontal lobes (see Figure 1). This area of the brain is generally thought to be involved in the planning of complex cognitive responses and in moderating social behavior, among other executive functions. As it relates to pain, the prefrontal cortex is thought to encode the cognitive aspects of pain, such as memory for pain, evaluation of the meaning of pain, and executive decisions concerning what to do about pain (Apkarian, Bushnell, Treede, & Zubieta, 2005), which are then initiated with the help of the ACC and motor cortex. Also, research shows that activity in the prefrontal cortex is *negatively* associated with the severity of pain, consistent with the view of the frontal cortex as serving a generally inhibitory function, and a specific role for this area in the modulation of pain (Lorenz, Minoshima, & Casey, 2003).

Plasticity

In addition to causing both specific (i.e., targeting specific areas) and diffuse (across the whole brain) activity in the nervous system, nociception can produce physical changes in the brain; changes that affect the processing of future nociceptive input. For example, nociception can sensitize some of the cortical areas involved in pain processing so that they are more likely to become active in response to future nociception (Tinazzi, Fiaschi, Rosso, T., Faccioli, Grosslercher, & Aglioti, 2000; see review by Melzack,Coderre, Katz, & Vaccarino, 2001).

Nociception can also produce shifts in the relative importance of structures in the brain as they respond to and process future nociception. For example, activity in the prefrontal cortex becomes increasingly engaged and associated with the experience of pain as pain becomes more chronic (Apkarian, Bushnell, Treede, & Zubieta, 2005). This finding provides a neurophysiological rationale for something that clinicians have long known; for the adequate treatment of chronic pain, processes related to the prefrontal cortex, such as memories and the meaning of the pain in the context of the patient's life goals, must be addressed. Plastic changes in the brain have also been found in patients with pain associated with amputation (Flor, et al., 1995; also see review by Elbert & Rockstroh, 2004), complex regional pain syndrome, type 1 (Maihöfner, Handwerker, Neundörfer, & Birklein, 2003; Pleger, Tegenthoff, Ragert, Förster, Dinse, Schwenkreis, et al., 2006), back pain (Apkarian, Sosa, Sonty, Levy, Harden, Parrish, et al., 2004; Flor, Braun, Elbert, & Birbaumer, 1997; see also Schmidt-Wilcke, Leinisch, Gänsbauer, Draganski, Bogdahn, Altmepfen, et al., 2006), and fibromyalgia (Kuchinad, Schweinhardt, Seminowicz, Wood, Chizh, & Bushnell, 2007).

As a group, these findings show that ongoing nociception and pain alters brain physiology and future pain processing. Unfortunately for persons with chronic pain, the changes that have been observed appear to lead to increases, rather than decreases, in pain and suffering. On the other hand, the fact that the nervous system shows changes in response to nociception and pain also opens the door to a number of treatment options such as hypnotic analgesia that might be able to reverse, or at least influence, CNS (dys)function.

Understanding the Neurophysiological Basis of Hypnosis and Hypnotic Analgesia

The improvements in neuroimaging technology have also facilitated research into the neurophysiological effects of hypnosis and hypnotic analgesia. One of the most important findings from this research is that the neurophysiological effects of hypnosis depends on the specific suggestions used. For example, effective hypnotic suggestions for decreased pain unpleasantness, but not pain intensity, have been shown to be associated with decreased activity in the ACC, but not S1 or S2 cortex (Rainville, Duncan, Price, Carrier, & Bushnell, 1997; see also Wik, Fischer, Brag e, Finer, & Fedrickson, 1999, who also found a decrease in activity in the cingulate cortex with hypnotic analgesia suggestions). On the other hand, effective suggestions for decreased pain intensity have been shown to be associated with decreased activity in the S1 cortex (and produce a similar trend in S2), but not the ACC (Hofbauer, Rainville, Duncan, & Bushnell, 2001).

Faymonville, et al. (2000) found that during the hypnosis, and relative to the other conditions, participants in a hypnosis condition undergoing painful stimulation had greater activity in the midcingulate portion of the ACC. Although the findings of Faymonville et al. (2000) differ to some extent from those of Rainville, Duncan, Price, Carrier, and Bushnell, M.C. (1997) and Wik, Fischer, Brag e, Finer, and Fredrikson (1999), cited above, who both found *decreased* activity in the ACC following hypnotic analgesia suggestions, Faymonville et al. (2000) argue that the findings as a group support the conclusion that the ACC plays an active role in pain modulation in hypnotic

analgesia; a conclusion consistent with the imaging research on the neurophysiological correlates of pain experience, cited above.

Using the design (and some of the subjects) used in their 2000 study, Faymonville and colleagues (Faymonville et al., 2003) next examined the functional connectivity between the midcingulate portion of the ACC and other brain areas during hypnosis. Connectivity was assessed as a correlation in activity (assessed by positron emission tomography) between the ACC and other areas of the brain physiologically connected to the ACC. In this study, they replicated the significant effects of their hypnotic intervention (relative to rest and to distraction) on pain. They also found an increase in connectivity with hypnosis in activity between the midcingulate ACC and the bilateral insula, the pregenual cingulate cortex, pre-supplementary motor area, right prefrontal cortex and striatum, thalamus, and brain stem. In other words, their hypnotic suggestion to re-live a pleasurable autobiographical event appeared to enlist activity in, and connectivity between, many of the areas that make up a critical cortical pain processing network.

Fingelkurts, Fingelkurts, Kallio, and Revonsuo (2007) compared cortical functional connectivity (assessed via correlations in EEG activity assessed over different scalp sites across a variety of frequency bandwidths) in a single highly hypnotizable individual between baseline and hypnotized conditions in two hypnosis sessions separated by a year. In apparent contrast to the findings of Faymonville et al. (2003), they found significant *decreases* in connectivity in activity (assessed as correlations in EEG activity across different bandwidths) assessed over a number of scalp sites following the hypnotic induction in both hypnosis sessions. Along these lines, Egner, Jamieson, and Gruzelier (2005) reported a decrease in functional connectivity (assessed via correlations in EEG gamma band activity) between the frontal midline and left lateral scalp sites in highly susceptible subjects after hypnosis during a Stroop task.

There is also evidence that hypnotic analgesia may be effective, at least in part, through its influence on activity at the level of the spinal cord. Support for this possibility comes from a variety of studies that demonstrate hypnotically induced reductions in skin reflex on the arm (Hernandez-Peon, Dittborn, Borlone, & Davidovich, 1960), nerve response in the jaw (Sharav & Tal, 1989), and muscle response in the ankle (Kiernan, Dane, Phillips, & Price, 1995). The study by Kiernan and colleagues (1995) may be particularly informative, as it demonstrated that suggestions for analgesia were correlated with the R-III (spinal nociceptive) reflex, a response that is not subject to voluntary control. Using a methodology similar to that of Kiernan, Dane, Phillips, and Price (1995), Danziger and colleagues (Danziger et al., 1998) later found two patterns of R-III reflex associated with hypnotic analgesia in 18 individuals with high levels of hypnotizability: eleven of their study participants showed clear inhibition and 7 showed facilitation of the spinal nociceptive reflex following hypnotic analgesia suggestions. Although the reasons for the differences in response are not easily explained, they do indicate that highly suggestible individuals can show changes in spinal nociceptive reflex when given hypnotic analgesia suggestions.

One additional study is of relevance for this section, and for helping to understand the effects of hypnosis on sensations. In this study, Derbyshire, Whalley, Stenger, and Oakley (2004) identified eight individuals who had high (>8) scores on the Harvard Group Scale of Hypnotic Susceptibility and who had been screened for their ability to experience pain, in response to hypnotic suggestions, without any noxious stimulation. fMRI images of brain activity were made in three conditions: (1) during actual noxious thermal stimulation, (2) following hypnotic suggestions for this same pain experience without stimulation, and (3) following a request that they imagine this pain without any hypnotic induction. Average pain ratings (on 0-10 scales) during noxious stimulation was 5.7, and the average pain ratings

in response to hypnotic suggestion was 2.8 (none of the participants reported that they experienced pain during the imagined pain condition). Similar brain areas became active following both noxious stimulation and hypnotic suggestions for pain, including the thalamus, ACC, midanterior insula, and parietal and prefrontal cortex. This research is striking in that it shows that hypnotic suggestions without physical stimulation can create reports of sensations (in this case, pain), and that the brain responds as if the sensations were real (see also Kosslyn, Thompson, Costantini-Ferrando, Alpert, & Spiegel, 2000, for similar findings concerning the perception of color). Of course, if people can create the sensation of pain, they can create other sensations as well, including sensations that are much more comfortable.

Does hypnosis and hypnotic analgesia increase or decrease cortical connectivity? Does it increase or decrease activity in the ACC? Does it inhibit or facilitate spinal reflexes? If the research findings that have been reported to date are replicated in future studies, then a reasonable conclusion is that hypnotic analgesia can do all of these things, depending, perhaps, on the specific suggestions used, the characteristics of the individuals being studied, and the specific neurophysiological effects and sites being examined. Concerning connectivity, for example, it is possible that responses to some hypnotic suggestions (e.g., to re-live a pleasurable experience, as suggested by Faymonville and colleagues) require more connectivity between some brain areas in order to effectively reduce pain, while responses to other suggestions (e.g., suggestions to “let go,” which is a common part of many hypnotic inductions) require decreased connectivity between some cortical areas in order to be effective. Similarly, some hypnotic suggestions (e.g., to decrease the affective or bothersomeness quality of pain) may require a global decrease in ACC activity to be effective, while others (e.g., to experience more pleasure related to memories of positive experiences) may require an increase in ACC activity.

One conclusion from this research is clear: “hypnotic analgesia” is not a single treatment that affects pain via a single underlying physiological mechanism. Instead, different hypnotic suggestions appear to affect pain experience via their effects on different neurophysiological processes and sites. This observation raises the intriguing possibility that *the efficacy of hypnotic analgesia interventions might be maximized by including suggestions that address multiple underlying physiological areas and processes*; not just, for example, those areas related to the sensory (somatosensory cortex) or affective (ACC) domains of pain.

Implications for Clinical Applications of Hypnotic Analgesia

Given the strong evidence for the efficacy of hypnotic approaches (Jensen & Patterson, 2006; Montgomery, DuHamel, & Redd, 2000; Patterson & Jensen, 2003), as well as evidence that the “side effects” of hypnotic analgesia are overwhelmingly positive (Jensen & Patterson, 2006), clinicians would do well to consider providing hypnotic treatments for any individual suffering from acute or chronic pain who expresses an interest in this approach. However, there is not yet a strong research base for understanding which hypnotic interventions or suggestions are most effective overall, or if some suggestions are more effective for some conditions than others. Until such research is performed, clinicians could use what is known about pain neurophysiology to help guide their use of hypnotic analgesia.

As described above, the experience of pain is associated with activity in the periphery, the spinal cord, a number of specific supraspinal areas, a general increase in cortical activity, and plasticity (changes in cortical structures). Changes in cortical connectivity have also been shown to be associated with hypnosis and hypnotic analgesia. The variety of neurophysiological processes associated with the experience of pain may explain, at least in

part, why so many psychological interventions effectively reduce pain for so many people; the target (of brain areas and processes associated with pain experience) is rather large, and hard to miss. The knowledgeable pain clinician can take advantage of the complexity of these processes when helping his or her clients learn to better manage pain.

Table 1 lists nine nervous system sites and neurophysiological processes that can contribute to the experience of pain, along with types of hypnotic suggestions that could potentially influence activity at those sites or within those processes (see also chapter on pain management in Hammond [1990] for a large number of additional hypnotic suggestions and strategies for pain control). Most, but not all, of the types of suggestions listed have been described in hypnotic analgesia case reports and clinical trials, although the suggestions described in these studies show a tendency to focus on just one or two types of suggestions with any one client or in a clinical trial.

Conclusions concerning the actual neurophysiological mechanisms affected by the suggestions listed in Table 1 must await research that addresses the site(s) of activity of these different suggestions; we cannot assume that any reductions in pain resulting from the suggestions listed in Table 1 always and necessarily have their effects via the one specific site or process that they are listed next to. For example, it is possible that suggestions designed to target pain intensity could influence pain via: (1) decreases in activity in the somatosensory cortex; (2) activation of the PAG, which could then produce a decrease in the responsivity of the STT cells in the dorsal horn via descending inhibition; (3) decreases in activity in the ACC and insula; (4) decreases in connectivity; or (5) some combination of these or other mechanisms. It is also possible that the specific mechanisms that underlie the same suggestion differ from one individual to another. However, even though we cannot conclude at this point that the suggestions listed in Table 1 necessarily have their analgesic effects by causing changes in the sites or processes they are listed with in Table 1, the sites and processes listed do provide the clinician with a way of organizing the kinds of hypnotic analgesia suggestions that they might consider when working with a client with pain.

Suggestions related to a goal of decreasing diffuse cortical activity

One primary effect of nociception is a general increase in cortical activity. To address this effect, it can be helpful to provide patients with suggestions for being able to create for themselves a sense of global calm. This can be accomplished by providing direct hypnotic suggestions for relaxation or other commonly used suggestions that contribute to a sense of general comfort. Many, if not most, clinicians who use hypnosis for pain problems provide suggestions for relaxation, often as a part of the initial induction (e.g., Abrahamsen, Baad-Hansen, Svensson, in press; Crasilneck, 1995; Jack, 1999; Lu, Lu, & Kleinman, 2001; Montgomery, Bovbjerg, Schnur, David, Goldfarb, Weltz, et al., 2007; Simon & Lewis, 2000; Spinhoven & Linssen, 1989).

A suggestion to create and then experience being in a “special place” (that is, a place that is beautiful, safe, relaxing, and calming to the client) can also achieve this goal (see Abrahamsen et al., in press, and Lang, et al., 2006, for examples of use of “safe place” suggestions as a component of hypnotic pain management). Almost all individuals, regardless of their global hypnotizability, report that they experience relaxation inductions as comforting and calming, and some (although not the majority), report substantial decreases in their experience of pain that can then last for hours following this induction alone. The ability to achieve a sense of general calm, through these or other suggestions, should be considered a key skill that should be taught to any individual who experiences pain.

Suggestions related to a goal of decreasing peripheral activity

There is some evidence that hypnotic suggestions can influence the chemical, inflammatory and other peripheral physiological processes that affect the responsivity of nociceptors. In one early study, Chapman, Goodell, and Wolff (1959) gave subjects hypnotic suggestions for one arm being “normal” or “anesthetic” (“numb” and “wooden”), or suggestions for one arm being “normal” and the other being “vulnerable” (“painful,” “burning,” “damaged,” and “sensitive”). They then exposed each arm to noxious thermal stimulation. Of the 12 times they compared responses between arms in the “normal” versus “anesthetic” condition, they found a greater increase in Bradykinn (a vasodilator associated with increased pain) content in the perfusate from a “vulnerable” arm than an “anesthetic” arm.

Also, the fact that hypnotic suggestions can affect spinal reflexes (Danziger, et al., 1998; Kiernan, Dane, Phillips, & Price, 1995), peripheral vascular activity (Casiglia, et al., 1997; Klapow, Patterson, & Edwards, 1996) and objective measures of wound healing (Ginandes, Brooks, Sando, Jones & Aker, 2003) can be taken as additional support for the conclusions that peripheral processes can be influenced by hypnotic suggestions. Thus, suggestions to decrease nociceptor responsivity in the periphery (see Table 1 for examples) should at least be considered and tried; and of course maintained in future treatment sessions and included in practice recordings for those patients who respond well to such suggestions.

Suggestions related to a goal of decreasing STT (dorsal horn and thalamus) activity.

The STT cells in the dorsal horn are affected by both ascending and descending factors. To stimulate memories of the effects of ascending factors, it could be suggested that the patient experience any one of a number of peripheral sensations that are known to modulate STT cells via ascending inhibitions (see Table 1 for examples). Other suggestions could be used to stimulate possible descending inhibitions, such as suggestions to “turn down the volume” on any experiences of discomfort (Table 1).

Suggestions related to a goal of decreasing activity in the somatosensory cortex activity.

The S1 and S2 cortexes encode information about pain intensity, location, and quality. Suggestions associated with these domains of pain are perhaps the ones most commonly described in hypnotic analgesia case reports and clinical trials, and include direct and indirect suggestions to affect each of these three pain domains (see Table 1).

Suggestions related to a goal of decreasing activity in the insula.

The insula has been suggested as the area of the limbic system responsible for encoding how a person feels physically with respect to homeostasis, including the overall presence and severity of pain, thirst, hunger, and air hunger, as well as more positive physical experiences such as sensual touch (Craig, 2003, in press). The goal with hypnotic suggestions related to these processes would be to “fill the insula” with pleasurable and calming physical sensations. When successful, such suggestions would be accompanied by a sense, not only of reduced pain, but of significant feelings of “relief,” which could then contribute to an overall sense of comfort and calm.

The relaxation suggestions discussed above, to encourage the patient to “feel” physically relaxed, and that are often a part of hypnotic inductions, achieve this goal. Other suggestions related to pleasurable physical feelings, such as feelings of a comfortable warmth or coolness (it is often a good idea to suggest that patients feel “just the right temperature” because sometimes people would feel more comfortable feeling cooler and sometimes feeling warmer) or memories of being physically comfortable can also achieve this. Such sensations, of course, are incompatible

with severe pain, so could contribute to a sense of homeostasis (perceived relief, comfort, and physical safety; see Table 1).

Suggestions related to a goal of decreasing activity in the ACC

The ACC has been proposed as being important for addressing and facilitating the motor aspects of the limbic system (Craig, 2003, in press). Suggestions to address this component of pain would include suggestions relating to a decreased need to *do anything* in response to feelings of discomfort (see Table 1). Alterations in this component of pain may explain, at least in part, the benefits of acceptance approaches to chronic pain treatment (McCracken & Vowles, 2006). However, suggestions to address the motor dimension of pain, or at least the compulsion to reduce pain, are rarely described in the in hypnotic analgesia literature.

Suggestions related to a goal of altering activity in the prefrontal cortex

Because nociception directly activates executive processes related to the meaning of pain, memories of pain, and the implications of pain for future functioning, excluding suggestions to address these processes limits the benefits that patients could potentially obtain from hypnotic analgesia treatments. Suggestions that the chronic (old, not any new) sensations have no meaning for the patient's physical well being (providing, of course, that the patient has been carefully evaluated from a medical perspective, with the finding that the chronic pain sensations can safely be ignored) can be very useful (see Table 1). When providing such suggestions, it might be useful to include suggestions that any new sensations, that might have implications for the patient's health and physical well being, can be noted, and appropriate action taken to "... address your health... whatever action is most appropriate for your wellness."

The clinician may also bring into the suggestions ideas about the patient's values and life goals, and link them to what is known about adaptive pain coping (e.g., maintaining an appropriate level of activity, focusing on life goals other than just pain reduction, increasing participation in distracting and healthy activities, appropriate activity pacing), and adaptive pain attributions (see Dane, 1996; Patterson & Jensen, 2003). To the extent that any experience of pain is associated with memories that contribute to pain and suffering, links to more comforting memories via age regression can be created (see, for example, Crasilneck, 1995; Lu, Lu, & Kleinman, 2001; and Abrahamsen, R., Baad-Hansen, L., & Svensson, P., in press). Age progression is yet another way to associate new, comforting images and feelings to sensations that had previously been interpreted as pain, but that are hopefully becoming, with treatment, increasingly associated with relaxation, comfort, and/or a sense of "an interesting challenge to be dealt with" (as opposed to something to panic about).

This aspect of pain is the primary target of traditional cognitive restructuring approaches to pain management (Turk, 2002; Turk, Meichenbaum, & Genest, 1983). However, cognitive restructuring is not usually applied following hypnotic inductions; this intervention seeks to engage patients directly, while they are in a non-hypnotic state, in the logical process of identifying and stopping maladaptive thoughts (which can be viewed as a type of self-suggestion) and developing more adaptive and realistic thoughts to replace the old ones (Ehde & Jensen, 2004). Cognitive restructuring has proven efficacy (Hoffman, Papas, Chatkoff, & Kerns, 2007), and should be considered as a part of treatment when working with an individual with pain. However, hypnosis can be used to help identify adaptive cognitive responses and attributions, as well as to experience, during hypnosis (i.e., not just imagine or think about) what it would be like to live with new adaptive thoughts.

Suggestions related to a goal of altering cortical connectivity

As was noted above, research suggests that connectivity – the functional association between different brain areas and processes – can be both enhanced and disrupted by hypnotic suggestions, perhaps as a function of the specific suggestion(s) made and the neurophysiological processes required to respond to those suggestions. To the extent that the perception of pain requires connectivity between different components of the cortical networks, then suggestions to disrupt this connectivity could presumably disrupt the experience of pain. A number of examples of such suggestions exist in the literature, including suggestions to dissociate from the experience of pain, almost as if it were in another person’s body (Botta, 1999), or to experience oneself as separating from one’s body (e.g., as “floating;” see Lang et al., 2006; see also Abrahamsen et al., in press; Jensen, et al., 2005; see Table 1).

Suggestions relating to plasticity

The cortical changes that are known to occur in persons with acquired amputation and CRPS-1, and that are related to chronic pain, can be reversed by interventions which increase activity and normal use of the S1 cortex associated with the painful area (Flor, Denke, Schaefer, & Grüsser, 2001). Hypnosis can be used to facilitate this, by suggesting to patients with phantom limb pain or CRPS-1 that they experience their affected limbs as moving comfortably and normally (Muraoka, Komiyama, Hosoi, Mine, & Kubo, 1996; Oakley, Whitman, and Haligan, 2002; Rosén, Willoch, Bartenstein, Berner, & Røsjø, 2001). Such suggestions can easily be incorporated into any hypnotic analgesia treatments that also include the other suggestions listed above. But such suggestions need not be limited to persons with phantom limb pain or CRPS-1 (see Dane, 1996, for an example of the use of this type of suggestion in a patient with MS). Asking patients to experience themselves as moving comfortably and easily presumably produces cortical activity and connections associated with pain-free movement. To the extent that such activity produces physiological changes that effect future cortical responses to movement, it is likely that those changes would be positive ones.

It is not clear whether the decreases in the density of gray matter in the dorsolateral prefrontal cortex that have been noted in persons with low back pain and fibromyalgia can be reversed via hypnosis or other means. The only explanation put forth so far for this decrease is that this area is chronically activated as a part of the brain’s attempt to modulate the pain, and this “overuse” leads to the release of toxic chemicals and subsequent gray matter loss (Apkarian, et al., 2004). One might speculate that a clinician could help create a “rest” for the prefrontal cortex—a decreased need for it to make ongoing efforts to modulate pain – by using one of the several suggestions listed above (for example, by altering the sensory qualities of pain or dissociating the sensory qualities of pain from the emotional responses to any pain experienced; or by encouraging the idea that the person need not “do anything” in response to pain), which might at least stop or slow the process of density loss. We do know that gray matter, at least in the parietal cortex, can be increased with learning (Draganski, et al., 2006). Whether or not some other similar mechanism, perhaps enhanced by hypnotic approaches, can be used to stop, slow, or reverse the cortical density loss in dorsolateral frontal cortex remains to be seen.

It is almost always the case that hypnotic analgesia treatment should include post-hypnotic suggestions to help make any and all benefits obtained with hypnosis permanent (Jensen & Patterson, in press). Given the evidence that nociception and the experience of pain can have detrimental effects on brain physiology and activity that are lasting, it is certainly possible that hypnotic interventions can have beneficial effects on brain physiology and activity, and that these changes can also be lasting. Thus, hypnotic analgesia sessions

should usually end with post-hypnotic suggestions that any benefits obtained from the session will last (see Table 1).

Summary and Conclusions

The complexity of the neurophysiological processing of pain means that there should be many ways and types of suggestions that will influence the experience of pain. Each of these types of suggestions should at least be considered, if not tried, with every patient. Through systematic application of these suggestions, and then continued use of those suggestions found to be most effective with any one patient, treatment efficacy for every patient can be maximized. More research will show which nervous system areas and processes are most responsive to hypnosis in general and to specific hypnotic analgesia suggestions in particular, and how these might differ as a function of patient characteristics (e.g., hypnotizability, pain diagnosis). The findings from this research will help guide future clinical work. But clinicians need not wait for the results of this research to practice state-of-the science clinical care. They can begin now, by expanding the suggestions they use to address all pain-related processes, in order to maximize the benefit their clients will achieve in getting control over pain and its effects on their lives.

Table 1: Hypnotic suggestions or approaches for pain management and possible site(s) of action (see text for discussion).

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
Diffuse cortical activation	Generalized calm	<p>“Allow your mind to be aware of the muscles and tendons in the [state body part or area], and allow those muscles and tendons to relax...let go...noticing whatever sensations that let you know that the muscles are relaxing. Perhaps a sense of heaviness...or warmth... or a sense of lightness... I don’t know what sensations you will experience, as the [body part or area] feels more and more relaxed...more and more comfortable...with every breath you take... and now, those feelings of relaxation move to the [next body part or area, until the entire body is covered].</p>
	The experience of being in a calming “safe place”	<p>“...and in your mind’s eye, you can picture yourself moving down a path, to a safe and comfortable place...a place you might have been before...or a place that you create for yourself... [continuing with suggestions that will allow the client to experience details of the place, including sensory experiences as well as references to safety and control].</p>
Periphery	Experience peripheral analgesia	<p>“The area of pain and discomfort is being engulfed in a psychological anesthesia...”(Crasilneck, 1995, p. 260), or glove anesthesia and transfer of sensations with glove anesthesia to peripheral areas.</p>
	Produce the experience of decreased nociceptor responsivity	<p>You can “... begin to reduce the oversensitivity by pour[ing] soothing, healing fluid down the affected nerves to help them.” (Williamson, 2004. p. 148).</p>

Note: STT=Spinal Thalamic Tract; ACC=Anterior Cingulate Cortex

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
STT (dorsal horn and thalamus)	Experience sensations that ascending STT cell inhibition such as warm or cool sensations.	“...your body, your spine, knows that when you rub a body part it overwhelms the spine with comfortable sensations, that displace any other sensations...and you can take advantage of that ability...imagining that someone is rubbing, massaging the part that is sometimes uncomfortable...feeling that relaxing massage...and noticing how it replaces any feelings of discomfort.”
	Experience or produce activities that reflect descending STT cell inhibition.	“Your brain is now sending messages to the gate-control stations to tune down the intensity and quality of the pain signals, so that you will feel less and less discomfort...” (Sacerdote, 1978, p. 20).
	Metaphors and images related to the inhibition of flow.	“...Picturing yourself in a room that is just too bright...so you go over to the dimmer switch, and turn the knob...dimming the lights” (see also “master control room” metaphor used by Gainer, 1992).
Somatosensory cortex	Decreasing pain intensity	“Now the pain in your right hand is beginning to lessen, the pain is subsiding; the pain is decreasing. With every breath you take, the pain in your right hand is diminishing; less pain... Lessening, decreasing, and almost gone” (Crasilneck, 1999, p.259), or “I can’t take away all of your pain...it is asking too much of your body...And if you lose 1%...you would still have 99% of it left, but it would still be a loss of 1%. You could lose 5 percent of that pain. You wouldn’t notice the loss of 5% because you would still have 95% of the pain; but you would still have a loss of 5%...You might even lose 80% of your pain, but I don’t think that is quite reasonable yet. I would be willing to settle for a loss of 75%...what is the difference between 75% and 80%, and sooner or later you can lose 80%, and maybe 85% but first, let us settle for 80%.”(Erickson, 1983, p.236).

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
	Alter the pain site	<p>“Now, you’ve got cancer pain. Why not have another kind of pain also? Why not have pain out here in your hand? You have cancer pain in your body. It is very, very troublesome; it is very, very threatening... You wouldn’t mind any amount of pain out here in your hand... And if you had pain out here you could stand any amount...” (Erickson, 1986, p. 80; Jack, 1999; Spinhoven and Linssen, 1989).</p>
	Alter pain extent	<p>“The area of any discomfort is shrinking, becoming smaller and smaller, actually shrinking, to the size of a hand... a palm... a band-aid... a button... a tiny tiny speck... so small... hardly noticeable at all.”</p>
	Alter pain quality	<p>“...substitute a different feeling, such as numbness or warmth, or tingling or pressure, for any unpleasant sensations... you may already be feeling different sensations – sensations that slowly and easily take the place of any uncomfortable feelings...” (Jensen et al., 2005, pp. 208-209), or “... This short, cutting, stabbing, blinding pain of yours, could you make that into a dull, heavy pain?” [and then transform this into a feeling of relaxation and weakness] (Erickson, 1980, p. 318), or “If you will just pay attention to that grinding pain you will notice that it is a <i>slow</i> grinding pain” (Erickson, 1983, p. 227; Abrahamsen, et al., in press).</p>
	Metaphors that alter the sensory aspect of pain	<p>“And you can picture the discomfort as a picture, a figure, or an image... that’s right... and now notice as the size, color, location, or other aspect of the image changes, or you might even imagine” or, “... You can picture putting these feelings in a box, then putting this box into another box, and then putting this box in yet another box, and placing that box in a room down a long hallway.” (Jensen et al., 2005, p. 206; Abrahamsen et al., in press; Erickson, 1967; Jack, 1999; Gainer, 1992; Spinhoven & Linssen, 1989).</p>

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
Insula	Experiencing comfortable bodily sensations (relaxation, warmth, lightness, etc.)	<p>“And in your special place, you can feel a breeze, it might be cool...or warm...it is just the right temperature...it just feels so good,” or lightness, etc.).</p> <p>“As you lower yourself into the healing water...you might feel a tingling...the feeling of the water around you...and the temperature...it is just right for you right now...pleasantly warm or cool...I don’t know what would feel the best to you now...but you do...and the water...so relaxing...filling your body with a sense of comfort... such relief... you might feel yourself wanting to sigh... a sign of relief...”, or</p> <p>“... now just feel the shawl and drape it around yourself...feel the snugness and how it is protecting you...feeling warm and snug trapping all the warm air...” (Jack, 1999, p. 235), or</p> <p>“Now as I talk and I can do so <i>comfortably</i>, I wish that you will <i>listen to me comfortably</i>... <i>it’s so comfortable</i> Joe to watch a plant grow...” (Erickson, 1966, pp. 203-204).</p> <p>“Remembering a time when you just felt so good, physically. Maybe you were getting a massage, maybe it was when you were running, comfortably, easily, and strongly...feeling so good... muscles relaxed, really feeling that way, right now.”</p>
ACC	Age regression to the experience of physical sensations incompatible with pain	<p>Experiencing a feeling of “not caring” about the pain - a feeling of not having to <i>do</i> anything about it.</p> <p>“... And know that you and your body are safe... the body knows...what to do to heal itself...there is no need for you to <i>do</i> anything more, other than what any person requires to maintain their health and comfort... you can just relax and accept that you will feel more and more comfortable.”</p>

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
Prefrontal cortex	Amnesia for pain to reduce recall of distress and dread of future pain	“The explanation was offered that, in amnesia for pain, one could experience pain throughout its duration, but would immediately forget it and thus would not look back upon the experience with a feeling of horror and distress, nor look forward to another similar pain experience with anticipatory dread and fear. (Erickson, 1959, p. 70)
	Altering the meaning of pain	“Pain...is no cause for undue alarm...it [can] reasonably be put into the background, much as noisy children are invited to play in a room with the door closed where they can be responded to if necessary, but otherwise ignored.” (Dane, 1996, p.233).
	Focus on valued goals other than pain reduction	“As you are able, more and more, to ignore feelings of discomfort, you are free to consider other things, and to move towards your own important life goals, perhaps goals related to what you want to do with with your family...your children... you are now free to grow...to leave discomfort behind,” or “While you’re thinking about this or that particular happy thing, you won’t have enough energy left over with which to feel the pain of your cancer because all of your energy is going to go into this matter of thinking over all the nice things...” (Erickson, 1983, p. 318).
	Focus on physical activity and fitness	“To create for yourself a healthy lifestyle...in the ways that are just right for you...being appropriately active... choosing and maintaining the exercises that fit with your goals and life...feeling so good about how in control you are... of your body...your health...your life.”

Neurophysiological site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
	Age regression to experience memories of comfort	<p>“And you can think back to a time when you felt so very comfortable... and remember what that felt like...in body... in mind...where are you? what are you doing?...you are there...really there... notice what is around you...and how you feel...really experience that comfort...in every nerve, every muscle, every tendon. Are you experiencing this now? Good...and now, remember this...allow the mind to bookmark this state. You can come back, now, to the here and now..., but bring this comfort with you.”</p>
	Age progression	<p>“And now... you can see yourself sometime in the future...maybe later today...tomorrow...next week...even months from now... feeling so much better then you do now. So confident in your ability to live the life you want, no matter what sensations you experience, to do what you want and need to do, comfortably, with a sense of relaxation and purpose...and thinking thoughts that reflect this confidence and comfort. Just filled with optimism and hope...a confidence that you can manage and move forward...you feel good physically... and emotionally. So relaxed...yet strong...so calm. I wonder what thoughts are going through your head that reflect these feelings? Positive, realistic, and reassuring thoughts...you may remember some of these thoughts...yes, that one.... and you will be able to tell it to me later...and now, as you come back to the here and now, you are bringing back with you some, or much, or all...you choose how much... of these feelings, these thoughts, with you... bring them right back with you as a part of your brain and body...so you can experience them here and now...and the rest of today, and tomorrow...” (Spinhoven & Linssen, 1989).</p>

Neurophysiological		
site(s) or problem	Goal(s) of suggestion(s)	Examples of suggestion
Cortical connectivity	Experiencing oneself as distant from one's body	<p>“The body is so relaxed...you even lose awareness of some parts.almost as if they were becoming thinner and thinner, or disappearing altogether...and as the body disappears, perhaps you experience yourself as a point of consciousness...floating...just floating in space...the body far below...you are just pure consciousness completely separate from the body.”</p>
	Disconnect sensations from an emotional response	<p>You may already be aware that any pain intensity, and the distress that pain can sometimes produce are two different things. They are processed in different parts of the brain. It is possible to be aware of a painful sensation, but not be bothered by it. Sometimes, it is even possible to experience significant pain...and still it does not seem to bother you...” (Jensen et al., 2005, p.208)</p>
Plasticity	Experiencing the painful area as able to move comfortably and easily	<p>“And as you experience yourself in this comfortable place, you can also observe your arm moving, naturally, and easily... the arm is bending...the fingers are moving...and now, lifting that weight... feeling the arm move...making it move... getting stronger and stronger...”</p>
	Post-hypnotic suggestions to make any benefits permanent	<p>All benefits that you have obtained from the session today, and your use of it for self-hypnosis in your daily life... those benefits can become... more and more... a permanent part of how your brain works... so that any time the brain can automatically and easily... without you even having to think about it... move itself into a state that allows for comfort and relaxation... just like the more you learn to do anything, driving, walking [insert appropriate examples from the patient's own life], the more automatic it becomes... automatic, freeing you up to do whatever else you want to do... to talk... to listen... to enjoy the moment... to really <i>be</i> with family and friends... what your brain is learning, and to the extent that it brings you comfort and a greater sense of control, then, is becoming more and more a permanent part of who you are... of how your brain works.”</p>

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