The Neurobiology of Pain, Affect and Hypnosis

Jeffrey B. Feldman
Wake Forest School of Medicine

Recent neuroimaging studies have used hypnotic suggestion to distinguish the brain structures most associated with the sensory and affective dimensions of pain. This paper reviews studies that delineate the overlapping brain circuits involved in the processing of pain and emotions, and their relationship to autonomic arousal. Also examined are the replicated findings of reliable changes in the activation of specific brain structures and the deactivation of others associated with the induction of hypnosis. These differ from those parts of the brain involved in response to hypnotic suggestions. It is proposed that the activation of a portion of the prefrontal cortex in response to both hypnotic suggestions for decreased pain and to positive emotional experience might indicate a more general underlying mechanism. Great potential exists for further research to clarify the relationships among individual differences in reactivity to pain, emotion, and stress, and the possible role of such differences in the development of chronic pain.

Keywords: Affect, hypnosis, neuroimaging, neuroscience, pain, stress, trauma

Introduction

Pain is the second most frequent reason that individuals consult physicians, with pain disorders estimated to cost over $100 billion annually in the U.S. (Gallagher, 1997; Aronoff & DuPuy, 1997). Though pain is universally experienced, research and clinical efforts to investigate and manage pain have historically been handicapped by the fact that it is impossible to fully know another’s pain. The private, multidimensional
nature of pain is recognized in the International Association for the Study of Pain (IASP) definition of pain. The official IASP definition of pain, as noted by Chen (2001), identifies three elements:

1. Pain is associated with injury and “threat” of injury;
2. It is an “unpleasant” and “emotional” experience; and
3. It is “subjective.”

Despite this consensus definition that pain is a subjective emotional experience, it is only recently that the affective dimension of pain has gained salience in pain research (Chapman, 2001). The affective dimension of pain includes both the immediate feelings of unpleasantness associated with the painful sensation and negative emotions evoked by the pain. These have been termed primary and secondary affect, respectively (Price, 2000). Secondary affect, also referred to as “suffering”, is largely generated by one’s understanding of the pain and its long-term implications. The experience of “threat” is also inherent in the definition of pain. Whether inherent in the stimulus itself or in one’s assessment of the meaning of the pain, threat involves the activation of the “alarm reaction” of the sympathetic nervous system. This was noted by Melzack (1999), who wrote, “We are so accustomed to considering pain as a purely perceptual phenomenon that we have ignored the obvious fact that injury also disrupts the body’s homeostatic regulation systems, thereby producing stress and initiating complex programs to restore homeostasis” (p.89). Pain evokes and involves not only emotions, but also the stress response system. Individual differences in pain threshold and reactivity might therefore reflect differences in affective and sympathetic nervous system responsivity.

This article will review recent research that points to individual differences in pain reactivity, and brain-imaging studies that elucidate the processing of pain. Studies that clarify the affective versus sensory aspects of pain will be a major focus. The role of hypnosis in these studies will be highlighted. The relevance of a large body of research on individual differences in emotional reactivity to the further investigation of the affective dimension of pain will be discussed. Additionally, the possible role of prior traumatic experiences in the determination of affective and pain responsivity will be proposed. The potential use of hypnosis in further neuroscience research and the implications of basic research to date on the clinical use of hypnosis for pain management will be discussed.

**Individual Differences in Pain Reactivity**

Individual differences in pain reactivity have long been understood to be a factor to consider in psychophysiological studies of pain. The nature of these differences was clearly demonstrated in an elegantly simple study by Coghill, McHaffie, and Yen (2001). Seventeen normal volunteer subjects (nine male, eight female) were subjected to a thermal stimulus of 49 degrees centigrade for 30 seconds “on” and 30 seconds “off” for a period of 5.5 minutes. As is often done in a clinical context, the subjects were asked to rate their degree of pain from zero to 10, with zero being no pain and 10 being the worst imaginable pain. Coghill et al. (2001) found a nearly perfect linear distribution of ratings from 1 to 9. In other words, individuals varied in their response to the same physical stimulus, with some indicating that it was only mildly bothersome.
(a rating of “1”) and others indicating that it was extremely painful (a rating of “9”). Furthermore, functional imaging indicated highly significant differences in brain activation between the high and low perceived intensity subjects. There was significantly greater activation in the primary somatosensory cortex (SS1), anterior cingulate cortex (ACC) and prefrontal cortex (PFC) among the high perceived-intensity subjects relative to their low perceived-intensity counterparts. In other words, the differences in cortical activation reflected the differences in the individual’s reported pain response. Notably, these differences occurred not only in the somatosensory cortex, where they might simply be considered the neural counterpart of the reported physical sensation, but also in frontal areas which might be considered somehow involved in the interpretation of those sensations.

**Pain Processing in the Central Nervous System**

Clinical efforts to manage pain have generally focused upon lessening the peripheral source of pain (nociceptive input), or at blocking pain at the spinal level. However, as stated by Casey (1999), “The size of the human forebrain in relation to the spinal cord gives anatomical emphasis to forebrain control over nociceptive input” (p. 7668). The most influential effort to account for the influence of affect, motivation and cognition upon the experience of pain to date has been the model proposed by Melzack and Casey (1968) over 30 years ago. They proposed that sensory and emotional components of pain are processed in parallel by distinct brain structures. Sensory-discriminative aspects of pain such as quality, location, and intensity were viewed as mediated by lateral thalamic nuclei and the somatosensory cortex. Affective and motivational dimensions of pain were proposed as mediated by medial thalamic nuclei, the prefrontal cortex, and the limbic system. Subsequently, a far more complex picture has emerged. In a previous study, Coghill, Sang, Maisog, and Iadarola (1999) convincingly demonstrated, using positron emission tomography (PET) with psychophysical assessment of graded painful stimuli, that pain intensity processing involves bilateral mechanisms of broader distribution in brain space than envisioned by Melzack and Casey. Pain intensity-related activation occurred bilaterally in the cerebellum, putamen, thalamus, insula, anterior cingulate cortex, and secondary somatosensory cortex. Contralateral activation was noted in the primary somatosensory cortex and supplementary motor area, and ipsilaterally in the ventral premotor area. In other words, the processing of pain intensity is far more complex, and involves more structures on both sides of the brain, than was posited in the traditional view that sensory-discriminative processing of pain is limited to the somatosensory cortex.

Multiple pathways entering the brain from the spinal cord further complicate the processing of pain. A and C fibers enter the spinal cord at the level of the dorsal horn, travel up the spine, and enter through a number of pathways in addition to the well-known anterior and lateral spinothalamic pathways. These include spinoreticular, spinohypothalamic, and spinopontoamygdaloid pathways. (Casey, 2000; Price, 2000). Spinoreticular pathways associated with arousal activate the nucleus raphe magnus (NRM), periaqueductal gray matter (PAG), lateral reticular nucleus (LRN), and locus ceruleus (LC). Electrical stimulation of these structures can produce powerful antinoceptive effects, probably serving the function of blocking pain under conditions of extreme stress such as a bullet wound not noticed in the heat of battle (Fitzgerald,
The locus ceruleus is also the brain structure most associated with the release of norepinephrine as part of the fight or flight response. The hypothalamus is the part of the brain most associated with the regulation of bodily processes and the initiator of the key aspects of the stress response involving the hypothalamic-pituitary-adrenal axis. The amygdala, as will be further discussed, is highly involved in emotional processing, especially of fear. Thus there are multiple pathways that function in parallel, directly activating parts of the brain associated with fear and the stress response. These pathways are in parallel with spinothalamic pathways that go from lateral thalamic nuclei to the SSI and other thalamic nuclei that connect to the cortex and the ACC. The ACC appears to be a central player in the process, receiving input from the thalamus, amygdala and insula and from the orbitofrontal and motor cortex (Price, 2000).

**Hypnosis and Pain**

In a series of studies, Rainville and his associates demonstrated the role of the ACC in processing the affective component of pain. In the first of these studies, Rainville, Duncan, Price, Carrier, and Bushnell (1997) used hypnotic suggestion to create a perceptual dissociation between pain unpleasantness and intensity. Hypnotic suggestions to both increase and decrease pain unpleasantness were given without changing the perceived intensity of the pain sensation. PET monitoring revealed significant changes in pain-evoked activity within the anterior cingulate cortex while there were no changes in the primary somatosensory cortex. In other words, the activation of the ACC varied depending upon hypnotic suggestion for the degree of unpleasantness of the stimulus, despite the degree of activation of the somatosensory cortex remaining constant.

Rainville, Carrier, Hofbauer, Bushnell, and Duncan (1999) further demonstrated in a series of experiments that hypnotic modulation of pain unpleasantness could be achieved independent of variations in perceived pain intensity. In the second experiment there was a significant correlation between stimulus-evoked heart rate increase and ratings of pain unpleasantness, but not pain intensity, suggesting a direct functional interaction between pain affect and autonomic activation. In the third experiment, suggestions were given to increase and decrease the sensory rather than the affective dimension of pain. Significant modulation of pain intensity was achieved with secondary changes in pain unpleasantness ratings. In other words, hypnotic suggestions to increase or decrease pain unpleasantness did not change perception of pain sensation. On the other hand, changes in pain unpleasantness closely paralleled the modulation of pain intensity, in response to hypnotic suggestions directed at the sensory qualities of pain sensation. This combination of results was viewed as consistent with a successive stage model of pain processing proposed by Wade, Dougherty, Archer, and Price (1996) and more fully articulated by Price (2000). Price (2000) reviewed pain affect mechanisms that involve a central network of brain structures and pathways that contain both serial and parallel connections. In their model, the ACC plays a pivotal role, receiving multiple inputs and thereby integrating somatosensory input with other sensory modalities, memory, and prefrontal cortical areas that attach significance and long-term implications to the sensation of pain.

**Hypnosis and Pain Modulation**

Rainville, Carrier, Hofbauer et al. (1999) noted that hypnotic susceptibility (Stanford Hypnotic Susceptibility Scale: Form A; Weitzenhoffer & Hilgard, 1959) was
specifically correlated to the degree of pain unpleasantness modulation in the experiment that targeted suggestions to that dimension, and to the degree of pain intensity modulation in the experiment that worded suggestions to increase or decrease that dimension. This is in contrast to a prior study by Price and Barber (1987) that seemed to indicate that hypnotic susceptibility was a good predictor of hypnotic sensory analgesia but a poor predictor of affective analgesia. Review of those results in light of the results of Rainville, Carrier Hofbauer et al. (1999) appear to indicate that those prior results were a function of the content of the suggestions that primarily involved the sensory dimension of pain. Rainville, Carrier, Hofbauer et al. (1999) concluded that the specific pain dimension on which hypnotic suggestions act depends on the content of the instructions and is not a characteristic of hypnosis itself. The power of hypnosis to differentially modulate sensory vs. affective dimensions of pain is even more impressive given recent findings by Chapman and his associates. Chapman, Carrier, Hofbauer, et al. (2001) found that the sensory and affective dimensions of phasic (brief duration) pain were indistinguishable in the self-report and psychophysiology of normal laboratory subjects. In other words, individuals could not reliably make a distinction between those two dimensions of pain in a non-hypnotic state. In contrast, Rainville, Carrier, Hofbauer, et al. (1999) found that individuals in an hypnotic state could not only make a distinction, but could differentially modulate sensory and affective dimensions of pain (with corresponding differential activation of brain structures) based on the nature of the hypnotic suggestion.

Rainville, Hofbauer, Paus, et al. (1999) further examined the cerebral mechanisms underlying hypnotic induction and suggestion. The effects of hypnosis and suggestions to alter pain unpleasantness were measured using both positron emission tomography (PET) to measure changes in regional cerebral blood flow, and electroencephalography (EEG). The experimental conditions included a baseline restful state followed by hypnotic relaxation alone and by hypnotic relaxation with suggestions for altered pain unpleasantness. During each scan, the left hand was immersed in neutral (35 degrees C) or painfully hot (47 degrees C) water in the first two conditions and in painfully hot water in the last condition. Hypnosis alone resulted in significant increases in both occipital rCBF and delta EEG activity that were highly correlated with each other. Peak increases in rCBF were also observed in the caudal part of the right anterior cingulate sulcus and bilaterally in the inferior frontal gyri. Hypnosis–related decreases in rCBF were found in the right inferior parietal lobule, the left precuneus and the posterior cingulate gyrus. Hypnosis with suggestions produced additional widespread increases in rCBF in the frontal cortices, predominantly on the left side. In addition, the medial and lateral posterior parietal cortices showed suggestion-related increases overlapping partly with regions of hypnosis-related decreases.

In other words, compared with the activation when individuals went into an hypnotic state of generalized relaxation, different areas of the brain were activated when they responded to specific suggestions to reduce the unpleasantness of the pain. Rainville, Hofbauer, Paus, et al. viewed their results as supporting a state theory of hypnosis in which occipital increases in rCBF and delta activity reflected the alteration of consciousness associated with decreased arousal and possible facilitation of visual imagery. Frontal increases in rCBF that accompanied suggestions for altered unpleasantness were proposed as reflecting the verbal mediation of the suggestions,
working memory and other “top down processes” involved in the reinterpretation of the perceptual experience.

Rainville, Hofbauer, Bushnell, Duncan, and Price (2002) recently replicated and expanded upon their prior findings. Using PET scans of 10 normal volunteers, they did 4 scans immediately before and 4 scans after the induction of hypnosis, asking subjects to rate their perceived level of mental relaxation and mental absorption. They once again found that hypnotic relaxation was associated with rCBF increases in both occipital lobes. In addition, the right Sylvan region, the left insula and the right ACC continued to evidence activation associated with relaxation. Decreases in rCBF observed in the prior study were replicated in the right inferior parietal lobule, the precuneus, and the left posterior temporal cortices. By contrast, reduced activation in the mesencephalic brainstem and the thalamus was noted to be exclusively associated with mental relaxation. Increased rCBF in the upper pons, the thalamus and the mid-ACC appeared to be exclusively associated with mental absorption. Furthermore, increases in prefrontal rCBF were positively correlated primarily with mental absorption. Overall, the study replicated prior findings that there are reliable changes in activation of certain brain structures and deactivation of others associated with the induction of hypnosis (mental relaxation), and that these differ from those involved in hypnotic absorption. Rainville, Hofbauer, Bushnell, et al. (2002) conclude by stating that, “These findings are consistent with the notion that hypnotic states are achieved through the modulation of activity within a distributed network of cerebral structures involved in the regulation of consciousness states” (p. 898).

Further support for a state theory of hypnosis was recently reported by Freiderich et al. (2001) using different psychophysiological measures. They recorded event-related electrical brain potentials to noxious laser-heat stimuli and pain reports during hypnotic analgesia, distraction of attention, and a control condition for highly hypnotically suggestible individuals. They found that pain reports were significantly lower in the hypnotic analgesia and distraction of attention conditions relative to controls. Nevertheless, based on amplitudes of laser-evoked brain potential (LEP), they found that hypnotic analgesia and nonhypnotic distraction of attention involve different brain mechanisms.

A full discussion of the important implications of the findings of the Rainville, Hofbauer et al. (1999), Rainville, Hofbauer, Paus, et al. (2002) and Freiderich et al. (2001) studies on the long-standing debate on the nature of hypnosis is beyond the scope of this paper. Some of the specific findings of Rainville, Hofbauer, Paus, et al. (1999) and Rainville et al. (2002) are, however, particularly relevant to the connection between pain and affect. Their observed changes in frontal cortices, predominantly on the left side, in response to hypnotic suggestions for decreased pain unpleasantness, provide a link to the literature implicating specific brain regions in the processing of affect.

**Pain, Affect and Autonomic Arousal**

Chapman, Nakamura, Donaldson, Jacobson, Bradshaw, Flores, and Chapman (2001) succinctly framed one of the key points of the present paper when they stated, “If pain has an affective dimension then it should lend itself to study suitable for emotion research” (p. 291). They went on to state that, “viewed broadly, some relationship should exist between autonomic arousal and negative affect, and this should
extend to the affective dimension of pain” (p. 291). They argued from their review of brain imaging studies that the perception of pain emerges not only from activity in the somatosensory cortex, but also from activity in emotion-related limbic areas of the cortex, including the insular and cingulate cortices. Furthermore, the involvement of spinohypothalamic and spinoreticular pathways, in conjunction with activation of the locus coeruleus with its projections into limbic structures (i.e., dorsal and ventral noradrenergic bundles) strongly suggests the simultaneous emotional processing of nociceptive signals, rather than it being consequent to higher order sensory processing. In their study they had 56 men and 44 women repeatedly experience varied painful electrical fingertip stimuli at low, medium and high intensities. In half of the trial blocks, subjects made sensory judgments; in the rest they made affective judgments. Physiological measures were monitored including pupil dilation, heart rate, respiration rate, skin conductance response (SCR) and late near-field evoked potentials. As previously mentioned, in contrast to Rainville, Carrier, Hofbauer, et al.’s (1999) hypnotic subjects, these participants’ pain ratings did not significantly differ when they were making sensory versus affective judgments. Also, the psychophysiologic measures did not differentiate affective versus sensory judgment conditions. Psychophysiologic measures, primarily the SCR, did reflect variability in the pain ratings. Congruent with the findings of Coghill et al. (2001) noted earlier, individual differences were noted among subjects in their baseline pain ratings. Chapman et al. (2001) further noted statistically significant individual differences in the relationship between pain ratings and SCR responses, indicating probable differences in the degree to which individuals detect and therefore emotionally react to visceral activity. In other words, while overall there was a significant relationship between pain ratings and measures that reflect sympathetic nervous system arousal, individuals differed in the degree to which autonomic arousal was associated with increased report of pain. Such autonomic arousal is generally associated with the experience of emotion, with variations among individuals in their emotional reactions to pain evident.

The Neurobiology of Emotions and Affective Style

In a recent meta-analysis of 55 neuroimaging studies of the functional anatomy of emotions, Phan, Wager, Taylor and Liberzon (2002) concluded the following:

1. The medial prefrontal cortex appeared to have a general role in emotional processing;
2. Fear specifically engaged the amygdala;
3. Sadness was associated with activity in the subcallosal cingulate;
4. Induction of emotional responses by visual stimuli activated the occipital cortex and the amygdala;
5. Induction by emotional recall/imagery recruited the anterior cingulate and insula; and
6. Emotional tasks with cognitive demand particularly involved the anterior cingulate and insula.

Other brain structures noted to be involved in the processing of emotions include the hippocampus and the dorsal lateral prefrontal cortex (Davidson, 2000, 2001, 2002). Damasio et al. (2000) found that the recall of all emotions they studied (happiness,
sadness, fear, and disgust) activated the cingulate, insular cortex, and brainstem. They noted that their findings are consistent with anatomic evidence that these regions are direct and indirect recipients of signals from the internal milieu and viscera, and are therefore important in the regulation of homeostasis. Phan et al. (2002) point out that Reiman et al. (1997) posited that the insula may function as an “alarm center” for internally-sensed dangers or homeostatic changes. Such a hypothesis would be congruent with brain imaging studies that consistently report the activation of the insula, as well as the ACC in response to pain. Indeed, given the affective dimension of pain, it is not surprising that there is a great deal of overlap between those structures identified as being activated by pain and those associated with emotional responding.

Most relevant to our discussion of individual differences in pain responsivity, Davidson (2000, 2001, 2002) and his associates at the University of Wisconsin have found consistent individual differences in emotional reactivity and regulation that they have termed affective style. Davidson and his associates have repeatedly found large individual differences in both infants and adults in baseline electrophysiological measures of prefrontal activation. Such individual variations are associated with differences in affective reactivity termed by Davidson (2000, 2001, 2002) as dispositional affect. In other words, some individuals are more prone to respond with negative emotions, while others with positive emotions. Individual differences in asymmetry of activation of the prefrontal cortex and amygdala are two key components of affective style. Among normal subjects, film-induced negative affect increases relative right-sided prefrontal and anterior temporal activation, while induced positive affect elicits an opposite pattern of asymmetric activation. Individual differences in baseline levels of asymmetric activation in these brain regions are lawfully related to variations in dispositional affective style. Left-frontally activated participants reported more positive and less negative affect on the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988) than their right-frontally activated counterparts. Similarly, participants with greater glucose metabolism in the right amygdala report greater dispositional negative affect on the PANAS, as well as other measures of negative emotional responsivity. Furthermore, a number of studies have shown that baseline levels of activation in the amygdala are associated with dispositional negative affect and depression. The amygdala has been noted by Davidson (2002) to be crucial for the learning of new stimulus-threat contingencies and important in the expression of cue-specific fear. Davidson has suggested that the connections between the PFC and amygdala play an important role in the regulation of emotions. Davidson (2002) has further emphasized that “affective chronometry”, or individual differences in tonic activation and phasic reactivity in this circuit, play an important role in governing different aspects of anxiety. Individuals who report greater dispositional negative affect and who show increased reactivity to stressful events may be those individuals who have difficulty regulating negative affect and, specifically, in modulating the intensity of negative affect once it has been activated (Davidson, 2000, 2001, 2002).

The relevance of dispositional affect and affective chronometry to individual differences in pain responsivity seems clear. If there are individuals who more quickly and negatively react to emotional stimuli, what source of negative affective stimuli could be more fundamental than painful stimuli? Furthermore, Rainville, Hoffbauer, Paus et al.’s (1999) hypnotic subjects demonstrated increases in rCBF in the frontal
cortices, predominantly on the left side (i.e. associated with positive affect) in response to suggestions for decreased pain unpleasantness.

Davidson (2001) also noted that the hippocampus plays a key role in the context modulation of emotional behavior. He proposed that what might be abnormal in disorders such as PTSD and depression is not the display of abnormal emotion, but rather the display of perfectly normal emotion in an inappropriate context. He observed that patients with PTSD behave similarly to an animal with a hippocampal lesion in failing to modulate emotional responses in a context-appropriate manner. Such a defect in response modulation appears relevant to the fact that a high percentage of individuals suffering from chronic pain have a history of prior trauma.

**Pain, Stress and Posttraumatic Stress Disorder (PTSD)**

Davidson has stated that, “the model we have developed over the past several years features individual differences in prefrontal activation asymmetry as a reflection of a diathesis that modulates reactivity to emotionally significant events” (2000, pp.1202-1203). He further explained that their studies support the idea that individual differences in electrophysiological measures of prefrontal and amygdala asymmetry mark some aspect of vulnerability to positive and negative emotion elicitors. (Davidson, 2000, 2001, 2002). What would be a more powerful or evolutionarily relevant emotional elicitor than pain? Further, implicit in the concept of the expression of a diathesis is an interaction between a genetic predisposition and stressful life experience in the development of such a vulnerability. Individuals who have experienced severe trauma would therefore be more likely to be more reactive to pain. While this has not directly been studied to date, there are many studies demonstrating a strong association between chronic pain and prior physical and/or sexual abuse. Studies indicate a history of trauma in 50-75% of individuals suffering from chronic pain whether due to an accident (Geisser et al., 1996), low back pain (Schofferman, Anderson, Hines et al.,1993; Blair, Blair, & Rueckert, 1994), headache (Domino & Haber, 1987), pelvic pain (Badura, Reiter, Altmair et al., 1997), Walker, Katon, Hansom et al., 1995), or general chronic pain (Wurtele, Kaplan, & Keaimes, 1997). Beckham, Crawford, Feldman et al. (1997) found that among Vietnam veterans presenting for treatment of PTSD, 80 % suffered from a chronic pain condition. Further, Sherman, Turk, and Ojifuji (2000) found a high prevalence of PTSD symptoms in fibromyalgia patients. Van der Kolk wrote, “Ever since people’s responses to overwhelming experiences have been systematically explored, researchers have noted that a trauma is stored in somatic memory and expressed in changes in the biological stress response” (1994, p. 253). Linking the stress response system to pain processing, Melzack stated:

> The limbic system, which receives projections of the medial sensory transmission pathways, is the neural substrate of the affective-motivational dimension of pain and a portion of the system, including the hypothalamus, is an integral part of the stress system. The two systems are so interdependent that they should be considered as components of a single system (1999, p. 97).

Whether the pain processing and stress response systems should be viewed as one system or closely interrelated systems, the studies currently reviewed demonstrate
a good degree of overlap among pain processing, affective reactivity, and the stress response system. Genetic makeup and prior life experiences can generate the neurophysiological differences underlying individual variations in pain, emotional and stress reactivity. Furthermore, the interrelated nature of these systems creates the likelihood that activating one system can trigger reactions in the others. For instance, pain may trigger psychophysiological and affective reactions, including memories associated with prior traumatic experiences. This was dramatically communicated to me by a patient with chronic back pain, who, following a good deal of therapeutic work, said, “It’s not the pain itself, Doc, that’s so hard to deal with. It’s all these memories [of early life trauma] and feelings that the pain triggers, with them flashing back to me like tapes playing in my mind.” In this author’s experience, it is very rare for a patient consciously to experience and articulate a connection between pain and prior strongly negative emotional experience. Nevertheless, might not pain generally trigger a high degree of affective and psychophysiological reactivity in individuals who have had a history of trauma and associate pain with overwhelmingly negative experience(s)? Rossi reminded us of such a process of “state-dependent learning” (Overton, 1978) as key to the development of psychophysiological symptoms, and to their resolution through mind-body healing. Rossi (1986) stated, “State-dependent memory, learning, and behavior processes encoded in the limbic-hypothalamic and closely related systems are the major information transducers that bridge the Cartesian dichotomy between mind and body” (p. 203). Rossi fundamentally argued that hypnosis is a process that can change the neurophysiological reaction patterns developed through state dependent learning. We now know that the processing of pain and emotion is more complex, involving more of the brain than traditionally termed “the limbic system.” Nevertheless, Rainville, Carrier, Hofbauer, et al. (1999) and Rainville et al. (1997, 2002) demonstrated that hypnotic suggestion can change an individual’s experience of the sensation and/or the affective dimension of pain, with corresponding differences in brain activation. There is thus a great potential for further use of hypnosis for neuroscientific investigations of the relationship between pain, affect, and trauma, as well as a therapeutic tool to alleviate associated suffering.

Summary and Directions for Future Research: Pain, Affect and Stress

Pain has been defined by the IASP as a subjective, unpleasant and emotional experience associated with injury or the threat of injury. The fact that pain involves affective and stress response systems is implicit in the definition. Neuroimaging studies demonstrate that pain involves highly distributed processing in multiple cortical and subcortical regions (Coghill et al., 1999). Information is processed in series and in parallel concerning the location, intensity, unpleasantness (primary affect) and the individual’s emotional reaction to the pain (secondary affect). Not surprisingly, many of the cortical structures associated with the processing of pain overlap with those associated with emotion and responses to stress. Foremost among these are parts of the ACC, insula, and prefrontal cortex. Additionally, the amygdala, which is the key brain structure associated with the fear response, has been noted to be part of a spinopontoamygdaloid pathway activated in the processing of pain (Price, 2000). One study indicated that it is the affective response to nociceptive input rather than its
perceived intensity that best predicted psychophysiological measures of reactivity to pain (Rainville, Carrier, Hofbauer, et al., 1999).

Investigations of the neurophysiology of pain and emotions have developed along separate and parallel tracks. It is proposed that a cross-fertilization of work in these areas begin. Researchers have consistently found significant individual differences in response to painful (Coghill et al., 2001) and emotional stimuli (Davidson, 2000, 2001, 2002). Investigation of the relationship between pain responsivity and “dispositional affect” (Davidson, 2000, 2001, 2002) is a logical beginning. Are individuals who are more predisposed to respond to negative affect more sensitive to pain? Further research might profitably investigate the relationships among dispositional affect, reactivity to painful, emotional and/or other stressful stimuli, and the development of chronic pain. The role of prior trauma in the development of such vulnerability might provide a key to understanding more general underlying mechanisms. Specifically, research investigation of the relative psychophysiological reactivity and cortical activation patterns of trauma survivors with and without chronic pain, other chronic pain patients and “normal” subjects to painful and other emotional stimuli is suggested.

Summary and Clinical Implications: Hypnosis and Pain

A number of findings concerning the nature of hypnosis and clinical implications for pain management appear worthy of emphasis. Though generally the perception of pain is an integrated process, Rainville (Rainville, Carrier, Hofbauer, et al., 1999; Rainville et al., 1997) used hypnotic suggestion to enable subjects to distinguish sensory and affective components of pain. Further, the degree of modulation of sensory or affective response to pain was correlated with hypnotic suggestibility. Chapman et al. (2001), who did not use hypnosis in their experiment, found that individuals were not able to reliably distinguish sensory and affective components of pain. This suggests that hypnosis may enable hypnotically responsive individuals to do what they cannot do in a non-hypnotic state. Hypnosis therefore appears to be a potentially more potent clinical tool for pain management than approaches that do not use it (i.e. relaxation, cognitive-behavioral).

It also is important for clinicians using hypnosis in pain treatment to not make the common error of thinking of pain as a purely sensory experience. Traditionally, hypnotists have focused upon techniques to enable individuals to dissociate from the sensation of pain. Neodissociation theory has proposed that hypnotic analgesia involves “…the disruption—the dissociation—of sensory information on the way to conscious awareness” (Barber, 1996, p. 6). A new door opens to potential hypnotic techniques when we also think in terms of facilitating dissociation from, or moderating, the affective components of pain (Price, 1996, p. 83). It is quite possible that individuals who cannot dissociate from the sensation of pain might respond to suggestions that diminish the affective dimension of pain. In this way, as demonstrated by Rainville and his associates (Rainville, Carrier, Hofbauer, et al., 1999; Rainville et al., 2002), individuals might report the same degree of pain sensation but be less distressed by it.

Of further clinical importance are Rainville, Carrier, Hofbauer, et al. (1999), and Rainville et al.’s (2002) findings that different brain areas are activated by hypnotic induction and hypnotic suggestion. The implication of these findings is that hypnotic induction alone is not as effective as hypnosis with specific suggestions. In other
words, the message matters. The crafting of suggestions relevant to the individual and the nature of his/her pain appears to be important, and creates an avenue for further research investigation. Rainville and his colleagues (Rainville, Carrier, Hofbauer, et al., 1999; Rainville et al., 2002) also found that the left prefrontal cortex was activated by suggestions for pain reduction. Davidson (2000, 2001, 2002) summarized the body of research from his lab indicating that this area corresponds to the elicitation of positive emotional affect. Further research is needed to clarify whether this is a result of the verbal processing of the hypnotic message as suggested by Rainville, Carrier, Hofbauer, et al. (1999), or the activation through hypnosis of a more general mechanism associated with positive emotional experience.

Conclusion

Brain imaging techniques have generated an explosion of neuroscience research investigating the processing of pain, emotion and stress. Hypnosis has been a tool used effectively in conjunction with brain imaging to distinguish the brain activity underlying the processing of the sensory and affective dimensions of pain. In the studies reported, researchers have elucidated distinct processes underlying hypnotic induction and suggestion. The long established clinical use of hypnosis in modulating pain, emotion, and stress creates the potential for further research on these interrelated aspects of experience. Great potential remains for the further use of hypnosis as a tool in neuroscience investigation, which in turn can expand our understanding of hypnosis and its clinical applications.

References


