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Pain that is "unanatomical" in distribution, for which there is no recent history of trauma, no evidence of a peripheral lesion and that resists traditional treatment, should be considered to be of psychogenic origin. The term *complex psychogenic pain* can be used when autonomic changes such as temperature abnormalities and physical findings such as tenderness accompany the pain. It is proposed that complex psychogenic pain is co-encoded centrally during a traumatizing event where the person experiences rage or fear with concomitant pain but is constrained from responding to the circumstances. Complex psychogenic pain is encoded as dissociated from the event. However, subsequent subconscious stimuli that recreate similar emotional, somatosensory, or cognitive states can activate a re-perception of the traumatic pain and engage various vasomotor processes. It is speculated that complex psychogenic pain is generated from amygdala efferents and is encoded in such a manner that precludes simple forgetting. Therapy consists of either delinkng the amygdala-based connection between the memory of the event and the emotional/somatosensory response or directly inhibiting amygdala outflow. Successful therapy extinguishes the pain.

Keywords: complex psychogenic pain; amygdala; rage; fear; dissociation; subconscious stimuli; prefrontal cortex; traumatization; serotonin; reconsolidation

The causative role of subconscious stimuli in the experience of chronic pain was first explored by Charcot, Janet, Freud, and Breuer (Tallis, 2002). They believed that the origin of the pain was a psychological trauma dissociated from conscious awareness. Accordingly, pain relief would occur only when the trauma could be brought to conscious awareness and treated. Walters (1961) defined psychogenic regional pain (PRP) as chronic pain that was associated with an emotional or psychological stimulus. The diagnosis is made when the patient experiences pain for which the clinician cannot find a physical lesion or peripheral cause. The most common pain locations described in his report on 430 cases were the back, neck, head, and upper limbs. Walters commented that the distribution of PRP was "unanatomical" and therefore probably was not generated by classical ascending pain pathways. Walters found that the pain could be associated with somatic abnormalities such as motor deficits, tenderness, sensory deficits, and autonomic dysfunction and that it was poorly responsive to treatment. We currently call this *complex regional pain syndrome*.

Tension myositis syndrome (TMS), another descriptor for chronic pain, was described by Sarno (1984, 1991, 1998, 2006), who argued that the pain reflects subconscious rage and is produced by vasoconstriction and ischemia in tendons and nerves. Sarno summarized the magnetic resonance imaging (MRI) data on lower back pain and concluded that MRI examination of the lower back does not correlate with either pain or treatment outcome. If there is no correlation, then the findings on the MRI do not correspond to the origin of the pain. He speculated that the pain arises from centrally encoded subconscious rage and that the pain prevents the person from experiencing that rage. Sarno stated that pain appears to occur at sites of a previous injury and observed sensory changes in the body, making the pain psychosomatic psychogenic in origin. Indeed, patients who suffer with lateralizing

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back pain that Sarno believed to be TMS have abnormal thermograms (Thomas, Cullum, Siahamis, & Langlois, 1990). Sarno recommended treatment that encourages the person to understand the benign origin of the pain. This process diminishes the fear associated with the pain. However, if the pain returns or is not extinguished, psychotherapy to identify and neutralize the subconscious rage is offered.

Scaer (2001), building on the work of Levine (1997), pointed out that chronic pain can be the result of a previous painful injury during a fearful episode where escape was impossible and a state of tonic immobility was produced. Scaer, like Sarno, believes that the pain and the observed autonomic changes, including skin temperature alterations, tenderness, and so on, are encoded centrally. This encoding occurs during the event in what Scaer calls the procedural memory system.

We prefer the term *complex psychogenic pain* (CPP) to describe the painful condition for which there is no peripheral cause. *Complex psychogenic pain* reflects and refers to a puzzling picture that is not explained by standard pain models. It is often unanatomical in distribution, associated with somatosensory changes, comorbid with psychological problems, and difficult to treat. For both patients and health care professionals, CPP appears to arise from peripheral sites. Thus, much therapeutic effort is directed to these areas, including treatments with opioid analgesia, surgery, and physical therapy. Unfortunately, these meet with little success. Because the pain does not have a peripheral cause and does not conform to known pathways, an understanding of how this pain is encoded centrally is essential to formulating an effective treatment.

We propose that CPP is encoded during a traumatizing event where the person cannot behaviorally express the emotional content of the event (Levine, 1997; Sarno, 2006; Scaer, 2001). This encoding occurs during what Cannon (1929) referred to as the emotional experience of defensive fury or fear. When fight or flight, the behavioral response to these emotions, cannot be carried out, the situation is perceived as inescapable and hopeless. The sense of helplessness and powerlessness and the inability to take responsive action are necessary for traumatization, and if the event is perceived by a person with an appropriate neurobiological landscape, this becomes an encoding moment for traumatization to occur.

The characteristics of this encoding moment are also critical to understanding CPP as well as its treatment. Although the exact neurobiology is unclear, we do know that the requisite landscape is the conjoined product of prior traumatic experiences and current events on an inherently predisposed brain. This means that not everyone who has a traumatic experience develops CPP.

The experience of chronic psychogenic pain must be the product of stimuli that cannot be consciously connected to the event but, like the accessible memories that produce emotional responses, they must have access to efferent pathways that can modulate peripheral sensation and autonomic activity as well as have global effects on cognitive functioning. Thinking about an event where pain is produced never reproduces the pain. Painful somatosensory responses are therefore dissociated from conscious awareness and have the potential to intermix the past with the present. A subconscious stimulus, one that is not consciously associated with the pain, can bring a dissociated pain to conscious awareness. The person has no idea where the pain comes from as the present becomes confused with a physical sensation from the past. The idea that pain arises from a subconscious dissociated stimulus is consistent with the everyday observation that pain cannot be experienced under normal conditions merely by thinking about a physically painful event. We also speculate that if CPP is encoded during events that produce the emotions of fear and defensive rage, the related emotions of anxiety and anger that occur as part of everyday stress have enough of a subconscious associative overlap to reproduce the pain response. This overlap causes confusion as to the encoding moment but helps explain how pain can be experienced without being traceable to the original event.

Pain From the Subconscious Mind

We do not think of somatic pain as arising from the mind because thinking about a painful event never reproduces the pain. The ability to re-experience pain requires subconscious stimuli activating a dissociated painful memory that was co-encoded with a traumatizing event. Usually, when pain occurs it is mistakenly believed to be of peripheral origin. Because pain normally follows peripheral ascending pathways and is neuroanatomical in distribution, CPP appears to be strange. How can pain be unanatomical? The answer is that the CPP is encoded centrally. Scaer (2001) argued that part of the nondeclarative

memory system, called procedural memory, encodes this type of pain. This occurs as a result of proprioceptive input from affected parts of the body during the traumatic event. Neither traumatic injury nor the muscles involved with a rage response (because they can symmetrically involve both sides of the body) are related to the distribution of classic ascending pain pathways, and hence this permits the pain to be unanatomical. For example, a pain that crosses the midline would be considered unanatomical if experienced without a peripheral lesion. As subconscious stimuli reactivate tissue that was conditioned during the event, for the clinician, this is puzzling.

Can subconscious stimuli activate the feelings in the body? Although the subconscious cannot be touched or mapped, it can be explored, and the sign posts are the emotions and behavior of the person. For this article we use the word *subconscious* to mean mental content, often generated by internal or external cues not consciously registered but that may nonetheless stimulate somatic symptoms and affective arousal.

As mentioned above, the person is routinely unaware of the causal role of any stimulus in the production of pain. The literature is replete with case histories that document this observation. Furthermore, research has shown that subconscious stimuli can indeed activate physiological processes. For example, people who have the disorder prosopagnosia (Milders & Perrett, 1993) are unable to recognize a familiar face but have physiological reactions as if they consciously knew the person. Furthermore, neuroimaging research reveals that subliminal presentation of threatening faces activates the right amygdala and the prefrontal cortex (Williams et al., 2006).

A human response to fear is fleeing, and the body prepares for flight with an increased heart rate and peripheral vasoconstriction. Fear, however, can occur at the moment of a motor vehicle accident or it can occur with an injury that is perceived as life threatening. When a child is subjected to sexual abuse, a normal response would be defensive rage. However, under these circumstances, actions that lead to escape or battle may not be possible. We speculate that activation by subconscious stimuli that were associated with the traumatizing event produce CPP by activating the amygdala. The ability to re-experience the somatic pain requires dissociation of the painful stimulus *and* co-encoding with the traumatization that immortalizes the event.

Migraine headaches
Low Back Pain
Neck and Upper Back Pain
Sciatica
Reflex sympathetic dystrophy
Somatization Disorders
Radiculopathies
Phantom Limb Pain

Figure 1. Categories of complex psychogenic pain.

Thus, depending on the somatosensory systems activated by the event, people present with different disorders arising from similar events. The types of disorders that can be considered under CPP are shown in Figure 1.

The person with CPP responds to stimuli that are subconscious and therefore does not relate the experience of pain to the encoding event. In fact, the memory of the original encoding event or its emotional component may also be dissociated, further compounding the confusion as to the origin of the pain.

A Hypothetical Model for the Mechanism of Traumatization

Cannon first described the physiology of defensive fury and fear in 1929. The term *fight or flight* accurately evokes the behaviors related to these highly emotional states. The behaviors are driven by the emotional states. Once a threat is perceived, the person is motivated to act. The mind engages and fear is generated. If escape is not possible, defensive fury may be activated and the person sends a signal to the predator that he or she is not afraid to do battle. The neurophysiological consequences of these emotional states are critical to the encoding of CPP.

Bracha and coworkers' (Bracha, Ralston, Matsukawa, Williams, & Bracha, 2004) reformulation of the fear survival response begins with a *freeze response* or *freezing*. This is what all animals do when they are exposed to danger, as in feeding in an open field where survival depends on vigilance. If a stimulus, either conscious or subconscious, alerts the animal to a potential predator, a freeze response

occurs. Here, the animal pays attention to the location of the stimulus and begins assessment. From an evolutionary point of view, because a predator scans for movement, freezing is advantageous for the prey. Salience and focus occur when the prey's brain releases dopamine. Higher levels of dopamine in the prefrontal cortex increase attention, focus the senses, and produce a state of arousal. If a predator or a stimulus that is highly suggestive of a predator appears, it causes fear or defensive rage in preparation for flight or battle. Just prior to the onset of action, the animal no longer needs to focus on the whether this is a predator; the animal knows it is a predator and also that the animal needs to either escape or fight. Serotonin is released, diminishing the effect of dopamine, while epinephrine, norepinephrine, adrenocorticotropic hormone, and endorphins are also released to prepare the person for action. These neurochemicals are critical for encoding of the event. In particular, the high levels of epinephrine and norepinephrine are critical (Cahill, 2003). As defensive rage follows fear, it produces the same neurobiological state but with different muscular activity. If escape is accomplished and a safe place is found or the predator is frightened or killed in battle, further serotonin is released. It is necessary and sufficient that serotonin be released when a safe place is found to avoid traumatization. Recent speculation (Ruden, 2007) postulates that rising serotonin levels activate γ-aminobutyric acid (GABA) neurons that inhibit the reconnection of the link between memory and amygdala outflow. It can be speculated that the repetition compulsion seen in victims of trauma may be driven by a desire to change the outcome and "find a safe place" (van der Kolk, 1989).

If, in the final stage of this sequence, escape or victory is not possible, the animal may enter into a state of tonic immobility, playing dead. Bracha defined this behavior as *fright*. Tonic immobility may enhance survival when a predator, thinking that its prey is dead, temporarily loosens its grip, providing the prey an opportunity to escape. These four moments have four different neurobiological states. Thus, freeze is different from rage or fear, flight or fight, and fright. We speculate that during the emotional state, defensive rage and fear provide the requisite neurobiological moment for encoding of CPP. As mentioned earlier, the appropriate neurobiological landscape must also be present. If there is no resolution for these emotions, that is, fighting or fleeing and finding safety are not possible, a traumatization is permanently encoded.

Defensive rage and fear are characterized by a number of physiologic signs of sympathetic activation, such as piloerection, increased heart rate, increased blood flow to muscles, and vasoconstriction. A clenched jaw with the aggressive baring of teeth, tightening of neck muscles and back muscles, and arching of the lower back characterize defensive rage. The biology of this process also explains the vasoconstriction as postulated by Sarno. This posturing, meant to frighten the predator, is also the location of most of the PRP and tension myositis. It is different from predatory rage, where the back is bent forward in an attack position. Defensive rage becomes an encoding moment when people are forced against their desire and feel helpless against a much more powerful adversary, or when a perceived injustice occurs with no resort. As a human response to fear, fleeing is essential and the body prepares for flight with an increased heart rate and peripheral vasoconstriction. Fear, without the ability to flee and find safety, can occur at the moment of a motor vehicle accident or it can occur with an injury that is perceived as life threatening. These are encoding moments, and the treatment of the resultant CPP requires a delinking of the emotional components (e.g., fear and rage) from the memory of the event.

Role of the Amygdala

A minimum of two neurobiological elements are necessary for traumatization to occur: the secretion of epinephrine and norepinephrine and the amygdala (Cahill 2003). This process is initiated in the lateral nucleus (LA) of the amygdala by norepinephrine sent via locus ceruleus (LC) efferents. A specific and as yet unknown neurobiological landscape is also required because not all those who experience a trauma develop a traumatization. From the LA, a signal is then sent to the basolateral nucleus (BLA). The BLA of the amygdala appears to have the necessary connections to be a suitable site for encoding both dissociated and nondissociated memory pathways (McIntyre, Power, Roozendaal, & McGaugh, 2003). The BLA sends signals to the central nucleus of the amygdala (Ce), which activates extensive excitatory efferent neurons to the sympathetic nervous system, the hippocampus, the nucleus accumbens, and the hypothalamic–pituitary axis. These efferent neurons are involved with stress

Figure 2. After complex psychogenic pain (CPP) is encoded, various subconscious stimuli enter the lateral nuclei (LA) of the amygdala. There they activate the linkage between the basolateral nucleus (BLA) and the central nucleus of the amygdala (Ce) and produce pain through polymodal pathways. If, as we speculate, the linkage between the LA and BLA can be activated by subconscious stimuli, then disrupting this pathway will also extinguish the ability of subconsciously dissociated stimuli to produce pain. Blocking the BLA→Ce pathway can also prevent CPP.

responses. In addition, Neugebauer, Li, Bird, and Han (2004) showed that the central nucleus has a nociceptive area that may be important in the experience of CPP. Importantly, the activity of the central nucleus, the final common pathway for fear generation, can be modulated by the prefrontal cortex (Berretta, 2005).

We speculate that CPP is the result of pain being encoded in the procedural memory system during the traumatizing event. This pain is linked to the cognitive and emotional components of the traumatic event stored elsewhere in the brain. Subconscious associated stimuli release norepinephrine into the LA and the BLA nuclei that activates the linkage between the components of the traumatic event and sends efferent signals that produce pain. One approach to eliminating CPP would be by disrupting this linkage. This would prevent future activation of the LA→BLA pathway involved in the traumatization and would prevent outflow from all associative stimuli sharing the common path. This disruption would eliminate the effect of the subconscious memory and the subsequent pain. Another approach would be to inhibit the BLA→Ce pathway (Figure 2). This approach, however, would not necessarily eliminate all of the stimuli that can activate CPP.

The basolateral portions of the amygdala are not the storage part of the memory; rather they coordinate emotions, somatosensory processes, and motoric action. The BLA appears to be the location for the linkage between thought, emotion, and somatosensory aspects of the event. If flight or fight cannot be carried out, this moment of defensive rage or fear has the potential to produce a traumatization.

Traumatic memories will remain intact unless a delinking process is initiated. Thus, subsequent subconscious stimuli associated with the event have the potential to reactivate the pain. They can be physical, emotional, or somatosensory and, because the pain is dissociated, will have no cognitive relationship to the encoding event. To clarify this, an example is instructive. In this case a young woman presented with severe hand pain of 3 months duration. There were no obvious peripheral lesions or recent history of trauma. A detailed history revealed, however, that 15 years prior she had been involved in a taxi accident in London where her hand, in the exact location where she currently experienced pain, swung around and hit her face, breaking her nose. The encoding of the traumatizing event occurred during a life-threatening accident that she could not escape. When she decided to return to that city, the pain appeared. She was completely unaware of any connection between the accident and the current hand pain.

Therapeutic Approaches to CPP

It is worthwhile to review the process that causes CPP and then determine where and how we can intervene to remove the pain. An event is perceived on a suitable landscape and produces a rage or fear response. If the emotional state can be acted on, that is, producing a behavioral response to fight or flee and find safety, then the event is inhibited from being encoded as traumatic. However, if the event is perceived as life threatening and inescapable, the potential for traumatization exists. Once encoded, retrieval of the memory releases norepinephrine and activates the LA and BLA. There are two locations where intervention appears to be possible. One is the pathway that connects the BLA→Ce. The medial prefrontal cortex can modulate this pathway through activation of intercalated neurons located between the BLA and the Ce. Indeed, Sarno, who believes that chronic pain is due to unexpressed anger, tries to decrease the patient's fear by suggesting that the pain is not sinister in nature. Interactions between the medial prefrontal cortex

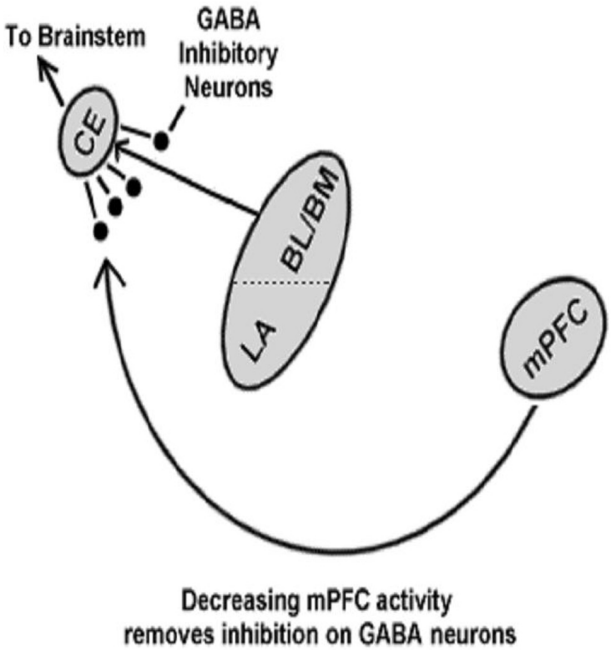

Figure 3. As prefrontal activity decreases, γ-aminobutyric acid (GABA) neurons exert more of an inhibitory response on the outflow of the basolateral nucleus (BLA). CE, central nucleus of the amygdala; LA, lateral nucleus of the amygdala; mPFC, medial prefrontal cortex. Modified from Quirk et al. (2003).

and the amygdala play a crucial role in modulating fear (Berretta, 2005). The medial prefrontal cortex, realizing that there is nothing to fear, decreases the Ce outflow by disinhibiting the inhibitory GABA interneurons (Quirk, Likhtik, Pelletier, & Pare, 2003) (Figure 3). However, if one addresses only the fear component, the underlying anger remains and the pain may recur or appear elsewhere in the body. If this occurs, Sarno then recommends psychotherapy to uncover and treat the source of subconscious rage. Levine (1997) and Scaer (2001) believe that the traumatizing event is encoded in what Bracha et al. (2004) called the "fright" phase of the survival response, what Scaer calls "freezing." The different terms for this state lead to confusion, so in this article we use the word *freezing* to denote the first awareness of a threat and the term *tonic immobility* to indicate the last part of the fight or flight response, or Bracha's term *fright*. Our analysis hypothesizes that the encoding moment is the stage of the process where the person develops an emotional response, either fear or defensive rage, and is unable to respond. This occurs at the height of norepinephrine release. The inability to escape and find a safe place prevents the global release of serotonin from the dorsal raphe. This lack of serotonin increase allows for unopposed norepinephrine release by subconscious stimuli. It is hypothesized that this is part of the mechanism of traumatization.

The process of traumatization requires consolidation of the event throughout the brain. If this memory is reactivated by subconscious stimuli, the amygdala portion of the memory pathway generates the somatosensory experience.

We speculate that delinking of the emotional, cognitive, and somatosensory components can be accomplished by various maneuvers that release serotonin. Such maneuvers have an analogy to massage (Field, Hernadez-Reif, Diego, Schanberg, & Kuhn, 2005). That is, we need to induce an increase of serotonin after bringing the memory to conscious awareness when the linkage between the LA and BLA is in an activated state. We speculate that serotonin disrupts this pathway by activating GABA inhibitory receptors that cause release of GABA (Ruden, 2007; Stutzman & LeDoux, 1999) in the BLA and locus ceruleus. GABA then inhibits the glutamate driven release of norepinephrine from the locus coeruleus and the pathways that lead to efferent neurons in the BLA.

Peter Levine has shown that if one can physically and/or imaginatively replicate the fleeing part and be guided to an imagined safe place, traumatization can be disrupted. This is the concept he uses in a method called somatic experiencing (SE). SE explores the "body memory," what Levine calls a "felt sense." This felt sense represents the somatosensory memory of a traumatizing event and may activate the release of glutamate in the LC and BLA. Patients will try to bring this felt sense to consciousness and then will be guided to an imaginary safe place. Dr. Levine's approach uses the physical aspects of the event and helps the patient complete the behavioral response to the traumatizing event, finding safety and thus removing the encoded trauma. It is speculated that imaginatively running and finding a safe place raise serotonin. This technique is extremely helpful in situations where the cognitive and emotional components are dissociated.

An increase in serotonin levels, occurring after the locus ceruleus and the LA→BLA pathway are activated, should delink the LA→BLA pathway where the relationship between memory of the event, pain, and emotion are stored. The first step in this process is to activate the emotional affect, either fear or rage, of the traumatic memory. This releases glutamate in

Activation of traumatic memory by subconscious stimuli that have been brought to conscious awareness → Release of glutamate → Norepinephrine release into the amygdala → EMDR or Tapping → Global release of serotonin from dorsal raphe → GABA released in Locus Coeruleus and BLA → Inhibition of release of norepinephrine → inhibition of BLA activation → NO PAIN

Figure 4. Serotonin induced GABA release on glutamate activated locus coeruleus inhibits norepinephrine release thus inhibiting BLA activation. The consequence is to diminish Ce outflow and remove the pain.

Somatic Experiencing works here
Event→ Rage or Fear→ // Fight or Flight
EFT and EMDR work here

Cognitive therapy and psychotherapy work here
→ **Encoded Pain (CPP)**

Figure 5. Emotional Freedom Technique (EFT) and eye movement desensitization and reprocessing (EMDR) delink the relationship between the somatic, cognitive and emotional components of a traumatizing event. Somatic Experiencing (SE) allows for completion of the event and the finding of a safe haven. Cognitive therapy and psychotherapy can alter prefrontal signals to the amygdala and can reframe the event, extinguishing the pain. CPP, complex psychogenic pain.

areas of the brain associated with the event, in particular the amygdala and the locus ceruleus. We speculate that subsequent sensory stimulation produces a global release of serotonin from the dorsal raphe. These sensory stimulation procedures involve soothing physical stimulation on various parts of the body, cognitive tasks (e.g., count backward from 5), and eye movements (Craig, 2007; Grant &Threflo, 2002).

In addition, we postulate that these procedures increase serotonin levels that stimulate GABA release in the BLA (see Figure 4).

Serotonin-induced γ-aminobutyric acid (GABA) release on glutamate-activated locus ceruleus and BLA inhibits norepinephrine release and diminishes outflow from the Ce. The consequence of this is to inhibit pain and which is accomplished by delinking the relationship between the memory of the event and the somatosensory component.

Conclusion

The hypothesis presented here ties together the work of Sarno, Levine, and Scaer. CPP should be considered an amygdala-based disorder. It can be defined as pain that is chronic and unanatomical in distribution, associated with autonomic dysfunction, without a history of recent trauma or peripheral lesions and unusually resistant to medical treatment. It is speculated that encoding CPP occurs at the time of defensive rage and fear when norepinephrine is released from the locus ceruleus and activates the lateral nucleus of the amygdala. If the person is unable to take action to either fight or flee and find safety, serotonin is not released. The lack of serotonin release on the appropriate landscape allows for the unopposed norepinephrine to activate the pathway of LA→BLA when a subconscious stimulus retrieves the memory. This retrieval activates the amygdala and pain is produced.

Treatment first requires a diagnosis of trauma-related pain. If the pain is considered psychogenic in origin, a search for a traumatic event is often revealing. Treatment requires that the trauma be resolved. The technique used is dependent on the availability of event memory, emotional recall, and somatosensory changes. Increasing serotonin by imaginal means and sensory stimulation can disrupt outflow form the amygdala (see Figure 5).

In the previous example, the young woman whose hand was injured was able to recall in vivid detail the accident in which she was clearly traumatized. We applied sensory stimulation to this memory and the hand pain instantly disappeared when the memory of the trauma was delinked from the emotional response.

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