

Encoding States: A Model for the Origin and Treatment of Complex Psychogenic Pain

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Pain that is ‘un-anatomical’ in distribution, for which there is no peripheral lesion and that resists traditional treatment should be considered to be of psychogenic origin.

The term Complex Psychogenic Pain (CPP) can be used when autonomic changes, such as temperature abnormalities, and physical findings such as tenderness, accompany the pain. It is proposed that CPP is co-encoded centrally during a traumatizing event where the individual experiences rage or fear with concomitant pain but is constrained from responding to the circumstances. CPP 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. We speculate that CPP is generated from amygdala efferents and is encoded in such a manner that precludes simple forgetting. Therapy consists of either disrupting re-consolidation of the amygdala based linkage between the memory and the emotional/somatosensory response or directly inhibiting amygdala outflow. Successful therapy extinguishes the pain.

Key Words Complex psychogenic pain (CPP), amygdala, rage, fear, dissociation, subconscious stimuli, prefrontal cortex, traumatization, serotonin, reconsolidation.

THE ROLE OF PSYCHOLOGICAL PROCESSES IN CHRONIC PAIN

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 found to be 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 comments that the distribution of PRP was 'un-anatomical' and therefore probably 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). He argues that the pain reflects subconscious rage and is produced by vasoconstriction and ischemia in tendons and nerves. Sarno summarizes the MRI data on low back pain and he concludes that MRI examination of the lower back does not correlate either with 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 speculates that the pain arises from centrally encoded subconscious rage and that the pain prevents the individual from experiencing that rage. He states that the

location of pain appears to occur at sites of a previous injury and observes sensory changes in the body making the pain psychosomatic psychogenic in origin. Indeed, patients who suffer with lateralizing back pain felt by Sarno to be TMS, have abnormal thermograms (Thomas & Cullum & Siahamis & Langlois 1990). Sarno recommends treatment that encourages the individual 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) points 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 . The term ‘complex psychogenic’ reflects and refers to a puzzling picture that is not explained by standard pain models. It is often ‘un-anatomical’ in distribution, associated with somatosensory changes, co-morbid with psychological problems and is 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. As 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 individual cannot behaviorally express the emotional content of the event (Levine 1997, Scaer 2001, Sarno 2006). This encoding occurs during what Cannon (1929) refers 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. It is the sense of helplessness and powerlessness and the inability to take responsive action that is necessary for traumatization, and if the event is perceived by an individual 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. While the exact neurobiology remains 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 individual has no idea where it came 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. If CPP is encoded during events that produce the emotions of fear and defensive rage, we also speculate that the related emotions of anxiety and anger that occur as part of everyday stress have enough of a subconscious associative overlap as 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. As pain normally follows peripheral ascending pathways and is neuroanatomical in distribution, CPP appears to be strange. How can pain be 'un-anatomical'? The answer is that the CPP is encoded centrally. Scaer (2001) argues 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, since they can symmetrically involve both sides of the body, are related to the distribution of classic ascending pain pathways and hence permit the pain to be ‘un-anatomical’. For example, a pain that crosses the midline would be considered un-anatomical 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? While the subconscious cannot be touched or mapped, it can be explored and the sign-posts are the emotions and behavior of the individual. For this paper we use the word ‘subconscious’ as meaning mental content, often generated by internal or external cues not consciously registered but may nonetheless stimulate somatic symptoms and affective arousal.

As mentioned above, the individual 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, in the disorder prosopagnosia (Milders&Perrett 1993) individuals are unable to recognize a familiar face, but have physiological reactions as if they consciously knew the individual. Further, neuroimaging research reveals that subliminal presentation of threatening faces activate 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 fear produce CPP by activating the amygdala. The ability to reexperience the somatic pain requires dissociation of the painful stimulus **and** co-encoding with the traumatization that immortalizes the event. Thus, depending on the somatosensory systems activated by the event, individuals present with different disorders arising from similar events. The types of disorders that can be considered under CPP include (Fig. 1)

Migraine headaches

Low Back Pain

Neck and Upper Back Pain

Sciatica

Reflex sympathetic dystrophy

Somatization Disorders

Radiculopathies

Phantom Limb Pain

Categories of Complex Psychogenic Pain

Fig. 1

The individual 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 individual is motivated to action. The mind engages and fear is generated. If escape is not possible, defensive fury may be activated and the individual 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 stimuli, 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, since a predator scans for movement, freezing is advantageous for the prey. Saliency and focus occurs when the prey's brain releases dopamine. Higher levels of dopamine in the prefrontal cortex increase attention, focuses the senses and produce a state of arousal. If a predator, or stimuli that are 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, also that it needs to either escape or fight. Serotonin is released diminishing the effect of dopamine while epinephrine, norepinephrine, ACTH

and endorphins are also released that prepares the individual for action. It is these neurochemicals that are critical for encoding of the event. In particular, it is the high levels of epinephrine and nor-epinephrine that 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 GABA neurons that inhibit the reconsolidation 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 defines this behavior as *fright*. Tonic immobility may enhance survival when a predator, thinking that his 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 than rage or fear and is different from flight or fight that is different than fright. We speculate it is during the emotional state, defensive rage and fear, that 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 is 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 and increased blood flow to muscles and vasoconstriction. A clenched jaw with the aggressive bearing 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 individuals 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 de-linking of the emotional components, eg. fear and rage, from the memory of the event.

THE 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 coeruleus (LC) efferents. A specific and as yet unknown neurobiological landscape is also required since 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 non-dissociated memory pathways (McIntyre 2003). The BLA sends signals to the central nucleus of the amygdala (Ce) that activates extensive excitatory efferent neurons to the sympathetic nervous system, the hippocampus, the nucleus accumbens and the hypothalamic/pituitary axis (HPA). These efferent neurons are involved with stress responses. In addition, Neugebauer et al. (2004) have shown 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).

Based on recent research, after memory consolidation of the original traumatizing event, retrieval of the event places the intra-amygdala linkage between stimulus and pain into a protein synthetic dependent state. Reconsolidation is required for subsequent retrieval. (Nader 2004). We speculate the CPP is the result of a conditioned release of norepinephrine into the LA that sets in motion a reconsolidation of the linkage between the LA and BLA, and its connection to Ce efferents. One approach to eliminating CPP would be by disrupting reconsolidation. This would prevent future activation of the LA→BLA pathway involved in the traumatization, and 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 (Fig.2). This approach, however, would not necessarily eliminate all of the stimuli that can activate CPP.

Subconscious stimuli → LA → BLA of the Amygdala → Central Nucleus → CPP

After CPP is encoded, various subconscious stimuli enter the LA nuclei of the amygdala. There they activate the linkage between the BLA and the Central Nucleus and through polymodal pathways produce pain. 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 subconscious dissociated stimuli to produce pain. Blocking the BLA → Ce pathway can also prevent the experience of CPP

Fig. 2

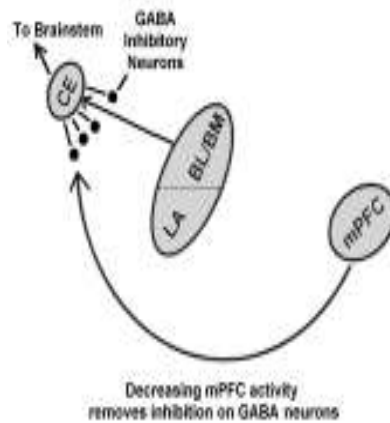
The basolateral portions of the amygdala are not the storage part of the memory, rather they coordinate emotions, somatosensory processes and motoric action. The BLA appears to be the location for the linkage between thought, emotion and somatosensory aspects of the event. If the flight or fight cannot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

Traumatic memories will auto-reconsolidate unless an inhibitory 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 since 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 three months duration. There were no obvious peripheral lesions nor was any recent history of trauma present. 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 where she could not escape. Three months ago, when she had made the decision 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 upon, that is, 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. This retrieval also causes the linkage that generates Ce efferent activity to become labile, requiring reconsolidation so that it remains intact. 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 treats patients by decreasing the fear by suggesting that the pain is not of sinister nature. Interactions between the medial prefrontal cortex 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 et al. 2003) (Fig 3).



Modified from Quirk et. al. (2003)
As prefrontal activity decreases, GABA neurons exert more of an inhibitory response on the outflow of the BLA

Fig. 3

This may be similar to the mechanism used by the cortex to calm fear generated by the hardwired thalamic → amygdala pathway that generates a fear response for unconditioned fear stimuli. (Ruden 2005). By removing the fear, the pain often remits. 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 (2004) calls the ‘fright’ phase of the survival response, what Scaer calls ‘freezing’. The different terms for this state lead to confusion. This paper chooses to use the word ‘freezing’ to denote the first awareness of a threat and ‘tonic immobility’ as 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 individual develops an emotional response, either fear or defensive rage, and is unable to respond. It is 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 rise allows for unopposed norepinephrine release by subconscious stimuli to initiate reconsolidation. This, it is hypothesized is the mechanism of traumatization.

The process of traumatization requires consolidation of the event throughout the brain. If this memory is reactivated by subconscious stimuli, it is the amygdala portion of the memory pathway that generates the somatosensory experience and requires protein synthesis to sustain the ability to retrieve the encoded memory. (Nader 2000). This synthetic activity reconsolidates the amygdala portion of the memory and sets the stage for future reactivation. Thus, traumatization promotes a memory that auto-reconsolidates the somatosensory component. Traumatization also promotes auto-reconsolidation for both the emotional components and cognitive components.

The process of reconsolidation is fundamentally different from consolidation of the original traumatizing event. That is, re-consolidation does not recapitulate consolidation (Bahar & Dorfman & Dudai 2004). If reconsolidation involves a final common pathway, then disrupting this pathway could potentially prevent all associative stimuli, including dissociated stimuli, from activating the BLA efferents and subsequent activation of the central nucleus that produce biological effects. We speculate that inhibition of the re-consolidation can be accomplished

by various maneuvers that release serotonin. Such maneuvers have an analogy to massage (Field 2005). That is, we need to induce a rise of serotonin after activation of the memory when the linkage between the LA and BLA is in a protein synthetic dependent 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 coeruleus. These GABA receptors inhibit glutamate directed protein synthesis as well as glutamate activated release of norepinephrine from the locus coeruleus.

Levine has shown that if one can physically and/or imaginatively replicate the fleeing part and be guided to an imagined safe place, protein synthesis may be inhibited and the encoding disrupted. This is the concept that Peter Levine 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 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, thus removing the encoded trauma. It is speculated that imaginatively running and finding a safe place raises serotonin and disrupts protein synthesis. It is extremely helpful in situations where the cognitive and emotional components are dissociated.

If, after the locus coeruleus and the LA → BLA pathway are activated, causing a rise in serotonin levels should disrupt the initiation of the reconsolidation of the LA → BLA

pathway where the relationship between memory and pain is stored. The first step in this process is to activate the emotional affect, either fear or rage, of the traumatic memory. This serves to release glutamate in areas of the brain associated with the event, in particular, the amygdala and the locus coeruleus. Glutamate activates the local synthetic protein machinery in the BLA and releases norepinephrine from the locus coeruleus. 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 (count backward from 5, for example) and eye movements. (Craig 2007) (Grant & Threflo 2002).

Specifically, for this therapeutic effect, we postulate that these procedures increase serotonin levels that stimulate GABA release in the BLA (Rainnie 1999) and directly inhibit glutamate activated pathways that cause the release of norepinephrine in the locus coeruleus (Ashton-Jones 1991). This two-pronged approach inhibits both LA initiation of reconsolidation and BLA protein synthesis necessary for reconsolidation of the amygdala based link between the retrieved memory and the pain. A schema for these approaches is seen below (Fig. 4)

Activation of traumatic memory by subconscious stimuli that have been brought to conscious awareness → Release of glutamate → Activation of BLA protein synthesis and norepinephrine release → EMDR or Tapping → Global release of serotonin from dorsal raphe → GABA released in Locus Coeruleus and BLA → Inhibition of release of norepinephrine and inhibition of LA initiation of protein synthesis in the BLA → Reconsolidation inhibited → //Ce → NO PAIN

Serotonin induced GABA release on glutamate activated locus coeruleus inhibits norepinephrine release and inhibition of glutamate driven protein synthesis disrupts reconsolidation and diminishes the Ce outflow. The consequence of this is to remove the pain. (See Text)

Fig 4

The consequences of inhibiting the reconsolidation are to de-link the relationship between the memory of the event and the somatosensory component. Thus, there is a loss of the ability for stimuli to activate pain.

CONCLUSION

CPP is fundamentally an amygdala based disorder. It can be defined as pain that is ‘un-anatomical’, associated with autonomic dysfunction without current evidence of 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 coeruleus and activates the lateral nucleus of the amygdala.

If the individual 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 permits the lateral nucleus to initiate reconsolidation of the linkage between the retrieved memory and the outflow from the BLA to reproduce the pain.

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 tapping can disrupt reconsolidation. In addition, medial prefrontal cortical inhibition of the outflow from the BLA is also possible. (Fig 5).

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)**

A pathway that can be interrupted produces CPP
At three sites

EFT and EMDR block re-consolidation of the emotional response to conscious stimuli. SE allows for completion of the event using non-cognitive stimuli. Cognitive and psychotherapy can alter the prefrontal signals to the amygdala and can re-frame the event, producing an extinction of the pain.

Fig. 5

The young woman whose hand was injured, was able to recall in vivid detail the accident where she was clearly traumatized. We applied the tapping procedure to this memory and the hand pain instantly disappeared when memory of the trauma was de-linked from the emotional response.

REFERENCES

- Ashton-Jones, G., Akaijam H., Charlety, P., Chouvet, G. (1991). Serotonin selectively attenuates glutamate-evoked activation of noradrenergic locus coeruleus neurons. *J. Neurosci.* 11:760-769.
- Bahar, A., Dorfamn, N., Dudai, Y. (2004). Amygdalar circuits required for either consolidation or extinction of taste aversion memory are not required for reconsolidation. *Eur. J. Neurosci.* 19(4):1115-8.
- Berretta, S. (2005). Cortico-amygdala circuits: role in the conditioned stress response. *Stress* 8(4) :221-32.
- Bracha, S.H., Ralston, T.C. Matsukawa, J.M. Williams, A.E., Bracha, A.S. (2004). Does “Fight or Flight” need updating? *Psychosomatics* 45:448-449.

- Cahill, L. (2003). The neurobiology of emotionally influenced memory. *Ann. NY Acad. Sci.* 985: 238-46.
- Cannon, W.B. (1929). *Changes in pain, hunger, fear and rage: An account of recent research into the function of emotional excitement*, 2nd ed. Appleton-Century-Crofts. New York, N.Y.
- Craig (2007) www.emofree.com
- Field, T., Hernandez-Reif, M., Diego, M., Schanberg, S., Kuhn, C. (2005). Cortisol decreases and serotonin and dopamine increase following massage therapy. *Int. J. Neurosci.* 115(10):1397-1413.
- Grant, M., Threflo, C. (2002) EMDR in the treatment of chronic pain. *Journal of Clinical Psychology* 58(12):1505-20.
- Levine, P.A. (1997). *Waking The Tiger*. North Atlantic Books. Berkeley, California.
- McIntyre, C.K., Power, A.E., Roozendaal, B., McGaugh, J.L. (2003). Role of basolateral amygdala in memory consolidation. *Ann. N.Y. Acad. Sci.* 985:273-293.
- Milders, M.V., Perrett, D.I. (1993). Recent developments in the neuropsychology and physiology of face processing. *Baillieres Clin Neurol.* 2(2):361-88.
- Nader, K., Schafe, G.E., LeDoux, J.E. (2000). Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature* 406:722-6.
- Neugebauer, V., Li, W., Bird, G.C. Han, J.S. (2004). The amygdala and persistent pain. *Neuroscientist* 10(3):221-234.
- Quirk, G.J., Likhtik, E., Pelletier, J.G., Pare, D. (2003). Stimulation of medial prefrontal cortex decreases the responsiveness of central amygdala output neurons. *J Neuroscience* 23(25):8800-7.
- Rainnie, D.G. (1999). Serotonergic modulation of neurotransmission in the rat basolateral amygdala. *J. Neurophysiol* 82(1):69-85.
- Ruden, R.A. (2005). A neurological basis for the observed peripheral modulation of emotional responses. *Traumatology* 11:145-158.
- Ruden, R.A. (2007). A model for disrupting an encoded traumatic memory. *Traumatology* 13:71-75.
- Sarno, J.E. (1984). *Mind Over Back Pain*. William Morrow & Co. New York, N.Y.
- Sarno, J.E. (1991). *Healing Back Pain*. Warner Books. New York, N.Y.
- Sarno, J.E. (1998). *The Mindbody Prescription*. Warner Books. New York, N.Y.
- Sarno, J.E. (2006). *The Divided Mind*. Regan Books, HarperCollins. New York, N.Y.
- Scaer, R.C. (2001). *The Body Bears The Burden*. Haworth Medical Press. Binghampton, N.Y.
- Stutzman, G.E., LeDoux, J. E., (1999) GABAergic antagonists block the inhibitory effects of serotonin in the lateral amygdala: A mechanism for modulation of sensory inputs related to fear conditioning. *J. Neurosci.* 19(11)RC8.
- Thomas, D., Cullum, D., Siahhamis, G., Langlois, S. (1990). Infrared thermographic imaging, magnetic resonance imaging, CT scan and myelography in low back pain. *British Journal of Rheumatology* 29(4):268-73.
- Tallis, F. (2002). *Hidden Minds*. Arcade Publishing, New York, N.Y.
- Van der Kolk, B. (1989). The compulsion to repeat trauma. *Psychiatric Clinics of North America.* 12(2):389-411.
- Walters, A. (1961). Psychogenic regional pain alias hysterical pain. *Brain* 84: 1-18.
- Williams, L.M., Liddell, B.J., Kemp, A.H., Bryant, R.A., Meares, R.A., Peduto, A.S., Gordon, E. (2006). Amygdala-prefrontal dissociation of subliminal and supraliminal fear. *Human Brain Mapping* 27