Harnessing Electroceuticals to Treat Disorders Arising From Traumatic Stress: Theoretical Considerations Using A Psychosensory Model

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Traumatically encoded memories can last a lifetime. These memories, either by purposeful or inadvertent re-activation, cause the release of stress hormones and generate a persistent and inescapable allostatic load on the body, brain and mind. This leads to a maladaptive response, as the ability to return to pre-event homeostasis is no longer possible. The consequence of this response is that it increases risk for further traumatization and other disorders. Remarkably, recent research has shown that these memories become labile and subject to disruption upon recall. In this paper we outline conditions needed for an event to be encoded as a trauma and describe a method that abrogates the release stress hormones when cued by these memories of the event. Critical to this process is the AMPA receptor (so named for its specific agonist, AMPA, α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid, a compound that acts as glutamate, its natural substrate). It is hypothesized that traumatic encoding requires increasing the number and permanence of AMPA receptors on the lateral nucleus of the amygdala by a process called synaptic potentiation. Depotentiation, that is removal of these AMPA receptors, is required for de-encoding. We speculate that the generation of oscillatory intracellular calcium waves is necessary for this to occur. Electromagnetic fields, acting as electroceuticals, interact with voltage-gated calcium channels on depolarized post-synaptic membranes to produce these intracellular calcium oscillations of varying frequency. These oscillatory calcium waves are decoded by intracellular calmodulin which, depending on the frequency, either act to potentiate or depotentiate AMPA receptors. This article describes the theory and practical application of a psychosensory approach called Event Havening that generates an electromagnetic field to synaptically depotentiate these encoded AMPA receptors and eliminate the effects of traumatic encoding.

Keywords: Calcium oscillations, Voltage-gated calcium channels, Psychosensory techniques, Synaptic depotentiation, Event Havening, AMPA receptors

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INTRODUCTION

Symptoms arising from a traumatically encoded event are produced by the release of stress hormones from continual/intermittent activation of an encoded memory (either by conscious or inadvertent recall).[1] These hormones generate an allostatic load[2] that drives an attempt to a restore the pre-event state. This adaptation ultimately becomes maladaptive as the consequences of the chronic, inescapable stress arising from traumatic encoding dysregulate homeostatic processes causing a wide range of both physical and emotional problems. These include: immune system dysfunction, chronic pain, substance abuse, depression, anxiety, heart disease, asthma, increased risk for further traumatization (especially if the traumatic events were experienced in childhood) and other disorders.[3] The current treatment of symptoms arising from traumatic stress disorders are often incorrectly addressed. For example, if one has back pain, it is the perceived back problem that is addressed. However, the data on the surgical or medical treatment of chronic back pain often reveals poor outcomes.[4] Using the mind/brain/body model, the back pain may in fact be the result of a traumatically encoded event during which the individual had experienced this pain.[5] Alternatively, chronic unresolved anger appears to also be a cause of back pain.[6] Surgery or drugs will not ameliorate the symptoms because they do not arise from the back; they arise in the mind/brain and treatment must be so targeted.

Recall of an event encoded as a trauma will bring to conscious awareness components of the trauma and cause it to be re-lived as if for the first time. Without intervention, these events are re-encoded as traumatic in a process called reconsolidation.[7] However, recent research has shown that upon reactivation, these traumatic memories may become labile, their reconsolidation disrupted and the traumatic memory de-encoded. In the experimental rat model for inhibiting reconsolidation, a conditioned stimulus (CS), light, is coupled with an unconditional stimulus (foot shock). After conditioning the animal freezes in response to the light. Once conditioning is complete, the animal is exposed to the CS and a pharmaceutical that inhibits protein synthesis is injected into the brain. Hours later, subsequent attempts at reactivation no longer produce a response.[8] The protein synthesis inhibitor prevented memory reconsolidation. In this paper I would like to offer an approach using brain waves in

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place of a pharmaceutical to accomplish a similar de-encoding but by a different mechanism. These therapeutically active brain waves can be called electroceuticals.[9] This paper outlines a mechanism for the efficacy of electroceuticals and its application in a therapeutic modality called Havening.

For an event to be encoded as traumatic, specific conditions must co-exist that produces vulnerability for this special type of encoding. We speculate that there are four requirements for an event to be encoded as a trauma and furthermore, if all requirements are not met upon reactivation, the re-encoding of the event as a trauma may be disrupted. What then are the requirements for traumatization?[10]

REQUIREMENTS FOR TRAUMATIZATION

First, an event must occur (Fig. 1). We can experience this event either first, second or third hand. That is, we can be part of it, we can be a witness to it or we can be told of it. Indeed, one's ability to imagine the event while listening to the stories of trauma places those who seek to aid these individuals at risk for vicarious traumatization.

Second, the event has to have **meaning, that is, be sensed as a threat** for the individual and **generate an emotional response**. Meaning can be learned or innate, such as fear of a gun (learned) or fear of heights (innate). In the final analysis meaning derives its power from threatened loss of attachment. We are attached to living, our bodies, our friends, and our sense of who we are in the community. When our attachments are threatened we fear their loss, and a powerful emotional response is produced. There is no emotional response without prior attachment and traumatization cannot occur without an intense emotional response.

All threats are connected to loss of attachment, some are hardwired, and some are culturally based, but all provide meaning and produce a strong emotional response. Those that are hardwired are called unconditional threat stimuli (UTS) (Table 1). Some personal and public based threats are listed in Table 2. These threats and the emotions they generate are necessary but not sufficient for an event to be encoded as a traumatic memory. Two other requirements are needed, a vulnerable landscape and perceived sense of inescapability.

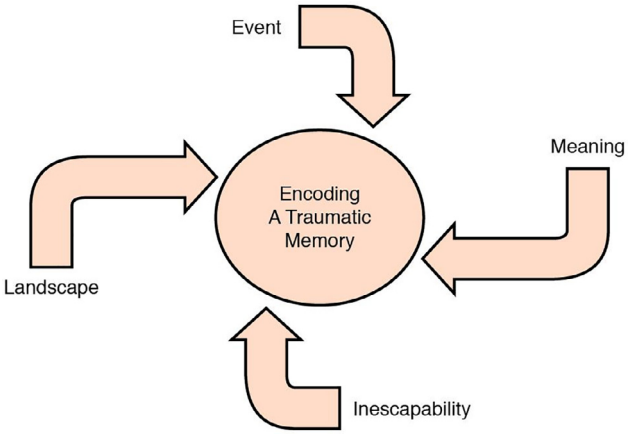

Fig. 1. An encoding moment.

Table 1. Unconditional threat stimuli.

Unconditional threat stimuli	
Abandonment	Darkness and night
Somatic pain	Air based predators
Heights	Ground based predators
Suffocation	Being killed by a predator
Being trapped	Smell, sight, and sound of
Open spaces	A predator

Table 2. Culturally based threats.

Personal and public based threats	
Loss of reputation	Excommunication
Loss of status	Loss of culture
Betrayal	Shame, guilt

A **vulnerable landscape** of the brain is the third element required for traumatic encoding. The landscape is the electromagnetic chemical state at the time of the event and is the sum of our inherent temperament, our sensitivity to stressors[11] and previous and current experiences. For example, preschoolers who witnessed the September 11 attack on the World Trade Center were at high risk for developing lingering emotional and behavioral problems only if they had had a previous frightening experience, like seeing a parent fall ill. It is unclear from this study whether these earlier frightening experiences resulted in traumatization. Nonetheless, it was found that 40% of those who had an earlier trauma suffered from depression, emotional outbursts, and poor sleep three years after 9/11. By contrast, children who saw the attack or its victims but had no such earlier trauma showed few, if any, psychological scars.[12] Remarkably, a previous traumatic event could be anything from an embarrassing moment to a serious accident. This simple but powerful illustration broadens our understanding about what can alter the landscape and sensitize an individual to traumatization.

What clinical features guide us to know who is more susceptible? Vulnerability is amplified by being overly empathetic, having low self-esteem, and in general, experiencing difficulty in regulating the level of emotional responsiveness. Individual traits such as obsessive-compulsive tendencies, anxiety, introversion, and addictive tendencies, also increase risk. The stressors caused by poverty and low education levels independently increase vulnerability for traumatization as does early childhood trauma.[13] In general, previous trauma predisposes to further traumatization. Thus, in the treatment of a current trauma, one should also seek earlier events that predispose the landscape towards vulnerability. If we can remove these early traumas, we can alter the landscape and make it more resilient to future distressing events.

The fourth requirement is perceived **inescapability**. In the natural non-human world one either escapes or is killed; there is no such thing as perceived inescapability, which requires a sense of outcome. However, for humans, there is a possibility of different outcomes. For example, in a car accident where we are tumbling out of control, there can be perceived inescapability and

the potential for traumatization. Returning to driving after surviving such an accident, if encoded as a trauma, can produce fear. This is protective but sometimes can be so amplified as to preclude the ability to ever drive comfortably again.

LANDSCAPE AND SYMPTOMATOLOGY

We have found that the landscape at the time of encoding is different from that which produces symptoms. Thus, it is critical to note that symptomatology derived from traumatic encoding requires a permissive neurobiological landscape. This is of *paramount importance* for understanding the consequences of traumatization. An event may occur and be encoded as a traumatic memory, but symptoms may be delayed or occur not at all depending on the landscape. Thus, the expression of symptoms is not always directly related to a particular traumatic event, but rather from the cumulative effect of unrelated stressors and the maladaptive responses that create a permissive landscape for symptom generation. It is not uncommon to see the delay in onset of symptoms be so long as to make the connection between the event and symptoms difficult at best.

HYPOTHESIS

Our premise is that on recall of a traumatic event, if all four requirements remain unchanged, the event will be reconsolidated as traumatic. However, if at the time of recall we can provide the biological experience of safety (escape) only three requirements are met and the memory will not be re-encoded. Subsequent attempts at retrieval will produce a memory without emotion and without stress. In order to accomplish this, we need to understand the underlying neurobiology of encoding a traumatic memory. Since the memory of the event is connected to the system that generates emotions, we look to the limbic system and specifically the thalamo-amygdala pathway as the entry point.

COMPONENTS OF A TRAUMATIZED MEMORY

To underscore what composes a traumatic memory, we consider that the memory contains everything that is present at the time of encoding.[14] All cognitive components enter the brain through their respective sensory organs and are converted into an electrochemical signal. These signals contain the **threatening content** which creates meaning. The **complex content** surrounds the threatening content and is associated with the threat, and the context, which is everything else (Table 3). The encoding also includes what happens after these stimuli are perceived and produce the visceral components (Table 4) such as the **somatosensory** (e.g. pain, temperature, position), **autonomic** (e.g. blushing, sweating) and the all important **emotional**.

Table 3. Aspects of the encoded event.

Cognitive components of a traumatic event
Threatening content
Complex content
Context

Table 4. Components of a traumatic event.

Visceral components of a traumatic event
Emotional
Somatosensory
Autonomic

ENCODING A TRAUMATIC MEMORY

Traumatic encoding starts with a threatening event. The receiving sensory organ(s) converts this into an electrochemical signal, which is relayed to the thalamus.[14] The thalamus sends projections to various parts of the brain including the lateral amygdala (Fig. 2). The membrane surface of lateral amygdala has many types of receptors, but one specific type of glutamate receptor, the AMPA receptor, which derives its name from the man-made agonist, AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid) is critical to maintaining the traumatic memory.[15]

When a threatening event is perceived, a thalamo-amygdala neuron releases glutamate and activates NMDA receptors on the post-synaptic amygdala neuron. (The N-methyl-D-aspartate receptor, NMDA receptor or NMDAR, is also a glutamate receptor and ion channel protein found in nerve cells.) Activation of NMDA receptors depolarizes the post-synaptic membrane via calcium influx. With the membrane depolarized, voltage-gated calcium channels (VGCC's) can now respond to low energy electromagnetic fields.[16] A high frequency signal, which interacts with altered voltage-gated calcium channels,[17] produces a synchronous intracellular calcium oscillation.[18] This oscillation is then decoded by calmodulin and other proteins that aide in bringing AMPA receptors to the membrane surface. Under traumatizing conditions when the requirements are met a unique phosphokinase called PKMzeta,[19] which lacks a shut off domain is activated. PKMzeta phosphorylates the AMPA receptor on the intracellular side of the lateral nucleus of the amygdala and maintains and permanently anchors it at the synaptic interface (potentiation). Meanwhile, the complex content and the context enter the basolateral (BLA) nucleus of the amygdala through the cortex and the hippocampus where they are bound with the signal generated by the UTS.[20] Outflow from the BLA are then bound to the visceral connected components located in different parts of the brain. This, then permanently encodes the entire event. (Fig. 2).[21] Once potentiated, these AMPA receptors make the **encoding immutable** and can be reactivated by cues that reflect the original event.

As an illustration we look at simple phobias. A bridge phobia is generated when the requirements for traumatization are met on a bridge. We perceive that we are way above the ground/water (the UTS is height, the **event**). This unconditional threat stimulus generates fear (because the bridge is covered with ice) and meaning (we may lose control and fall off bridge and die), and we subconsciously associate the fear with the bridge. If there is **meaning** (you could drive off the bridge and die) and your landscape is **vulnerable** (you are under stress for other reasons) at that moment and **inescapability** is perceived (no way off the bridge at that moment), AMPA receptors are trafficked to the cell surface and anchored by PKMzeta. Milliseconds later, the complex content (e.g. the color of the bridge) and the context (e.g. the sky was cloudy, ice on the bridge), which are the non-

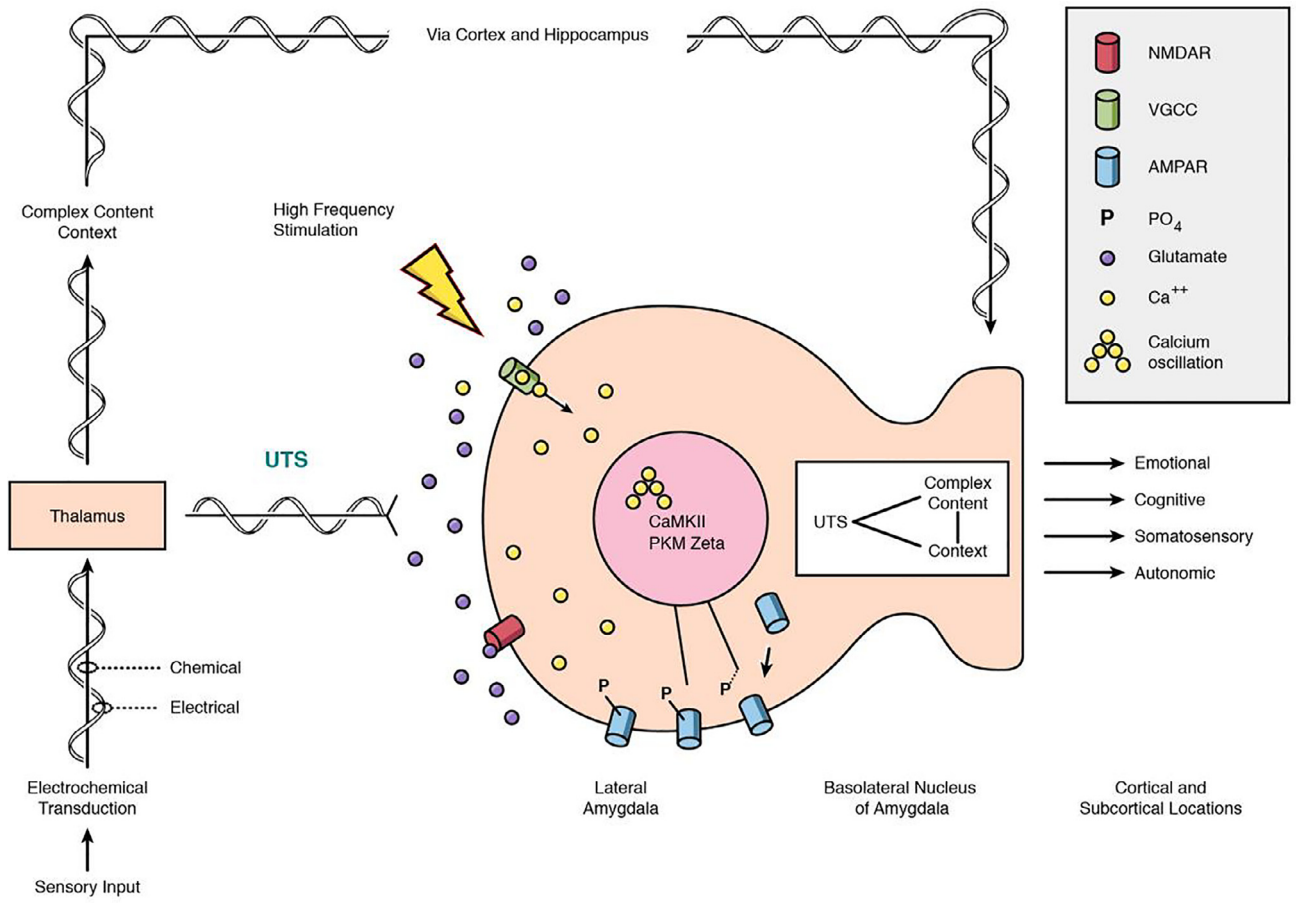

Fig. 2. Sensory input arising from an event is transduced to an electrochemical (EC) signal, which enters the thalamus. The UTS aspect is sent via thalamo-amygdala pathway to the surface of the lateral nucleus of the amygdala. The complex content and the context aspects of the event are sent to the cortex. The thalamo-amygdala neuron releases glutamate activating the NMDA receptor allowing Ca^{++} to enter the cell, depolarizing the membrane. A high frequency wave is generated which interacts with voltage-gated calcium channels (VGCCs). Membrane depolarization changes the VGCC's allowing for a calcium influx that is dependent on the wave frequency. This produces an intracellular calcium oscillation. This oscillation is decoded and brings AMPA receptors to the cell surface. Traumatic encoding conditions also activate a kinase called PKMzeta which is brought to the membrane surface where AMPA receptors are phosphorylated, permanently anchoring them to the surface (potentiation). Meanwhile, the complex content and the context enter the basolateral (BLA) nucleus of the amygdala where they are bound with the signal generated by the UTS. Outflow from the BLA is then sent throughout the brain where they are bound to areas that produce visceral responses. Reactivation by purposeful or inadvertent recall can reproduce some or part of the event as it was encoded in the brain.

emotional aspects of the event, enter the amygdala via the cortex and hippocampus and are co-encoded with the activated visceral components (mostly fear). One should consider these traumatically placed AMPA receptors on the lateral nucleus of the amygdala to be the on-ramp to re-living the emotional, cognitive, somatosensory and autonomic components stored in the brain.

APPLYING A PSYCHOSENSORY TECHNIQUE TO DEPOTENTIATE AMPA RECEPTORS

We define a psychosensory therapy as one that uses non-specific sensory input to alter mood, thoughts and behavior. This technique represents a third pillar (psychotherapy and pharmacotherapy are the other two) as it works by different mechanisms. The outcome of a particular psychosensory therapy can be divided into two major divisions, one in which the mind is activated by the memory of the event or a component of the event just prior to sensory input (exposure) and operates through synaptic depotentiation and one in which the mind is at rest prior to sensory input (non-exposure). The first group addresses life-specific events and their consequences. The second group can be used to downregulate various emotional states. For psychosensory approaches we propose that brain waves generated by sensory input therapeutically act as electroceuticals. While the mechanisms by which sensory input produces these electroceuticals are not well understood, it must somehow involve the "meaning," learned or innate, of the sensory input for that individual. A partial list of the psychosensory therapies is given below (Table 5):

Recalling a traumatic event is often experienced as if it were happening for the first time. This suggests that the downstream neurobiology involved with recall may be congruent with encoding. As mentioned above, we speculate that if one could produce

a response at the moment of recall that signals safety, a perceived escapability, one of the requirements for encoding of the emotional event would be lost.[10] If correct, experiencing safety, a haven, at that moment may disrupt the path that leads to the maintenance and re-encoding of the event and its attendant components. What mechanism would allow this to happen?

THE NEUROBIOLOGY OF TRAUMATIC MEMORY DEPOTENTIATION

Imaginal recall of the memory activates the thalamo-amygdala neuron and causes release of glutamate at the thalamo-amygdala synapse. This activates the post-synaptic NMDA and the AMPA receptors on the lateral nucleus of the amygdala. NMDA receptor activation permits calcium entry into the cell and depolarizes the neuronal membrane. The binding of glutamate to the AMPA receptor exposes the anchoring phosphate. We speculate that applying a soothing, gentle, comforting touch (we call Havening Touch) signals safety (escapability) and produces a low frequency delta wave[22] as measured by EEG. This low frequency delta wave interacts with the altered voltage-gated calcium channels (VGCCs) on the depolarized post-synaptic membrane of the lateral amygdala producing a slow intracellular calcium oscillation. It has been shown that this oscillation arises as a consequence of calcium influx from extracellular calcium and not from intracellular calcium stores.[23] This sets up a cascade of events which cause depotentiation of the AMPA receptor. Why does gentle touch signal safety? The answer lies in evolutionary biology and begins at birth. The newborn, unable to either speak or understand language, has an innate fear of abandonment that causes it to cry out. The mother, whose brain is suffused in the hormone oxytocin (the labor-inducing and bonding hormone), hears this cry. The sound drives the mother to hold, stroke and comfort her newborn child. The instinctive touch is indeed Havening Touch. It causes the infant to experience a sense of safety and feel it is not abandoned. As touch is continued, crying abates and both newborn and mother are comforted. This relationship between soothing touch and sense of safety lasts a lifetime. Thus, Havening Touch, applied under therapeutic conditions, tells the brain that we are safe and the event is escapable. In addition, as in potentiation, this glutamate release by binding to the AMPA receptor also exposes the internal phosphorus site that anchors the AMPA receptor to the membrane surface. This low frequency calcium oscillation is decoded by calmodulin and leads to the activation of an enzyme called calcineurin, a phosphatase. Calcineurin removes the anchoring phosphorous molecule from the AMPA receptor. Dynamin, along with clathrin, then causes endocytosis (the bringing into the cell) of AMPA receptors thus removing them from the surface membrane.[24] Once inside the cell the AMPA receptors are either degraded or recycled (Fig. 3). Only depolarized membranes participate in this process thus speaking to the specificity of the process.[25]

The disappearance[26] of the event-specific AMPA receptors from the surface of the post-synaptic neuron is the neurobiological equivalent to de-linking the recalled memory (the threat) from its co-encoded components. In essence, we are removing the on-ramp to the amygdala pathways that bring the cognitive and visceral components to conscious awareness.[27] The previous emotional response no longer can be produced and the release of stress hormones due to reactivation on recall does not occur (Fig. 4). The rapid time course for this process (seconds to minutes) suggests that the process does not involve protein synthesis but is electrochemical in nature.

Once the AMPA receptors are depotentiated, the UTS pathway that produces the emotional component is now gone. Once this is removed, the autonomic, somatosensory components are also irretrievable as they are generated through the amygdala. Parts of the cognitive component, which goes through the cortex as complex content and context may remain so that some of the recalled memory can be visualized, but this depends on how much of the event entered the amygdala via the cortex and hippocampus.

Table 5. Types of psychosensory therapy.

GROUP 1	
Havening	
Emotional Freedom Techniques (EFT)	
Callahan Techniques-Thought Field Therapy (CT-TFT)	
Eye Movement Desensitization and Reprocessing (EMDR)	
Others	
GROUP 2	
Yoga	Aromatherapy
Acupuncture	Massage
Biofeedback / Neurofeedback	Reiki
Exercise and related activities	Rolfing
Music	Others
Light	

EVENT HAVENING (EH) AS A PSYCHOSENSORY TECHNIQUE[9]

Havening is a technique that uses the sensory aspects of gentle touch to generate an electroceutical effect. The specific electroceutical generated by touch is called a delta wave and has a frequency of 0.5−4 Hz. We call Havening a psychosensory technique because it uses the application of non-specific sensory input, touch, to change the psyche. Event Havening deals with specific events. There are other forms of Havening, which constitute the system called Havening Techniques. These can be found at www.Havening.org.

Overview

Event Havening has five elements. **First** is the retrieval of the event by imaginal recall. This releases glutamate from the presynaptic neuron. This activates the NMDA receptor and Ca^{++} influx depolarizes the post-synaptic membrane. In addition, glutamate attaches to the AMPA receptor, and the anchoring phosphate becomes exposed.

Second, once the memory is reactivated, the client is asked to generate a Subjective Units of Distress (SUD).[28] The SUD is an eleven-point scale from 0-10 where zero reflects calmness and no distress and 10 is extreme distress.

Third, once SUD is stated, the client empties their mind and Havening Touch is applied (see Fig. 5) either by the client

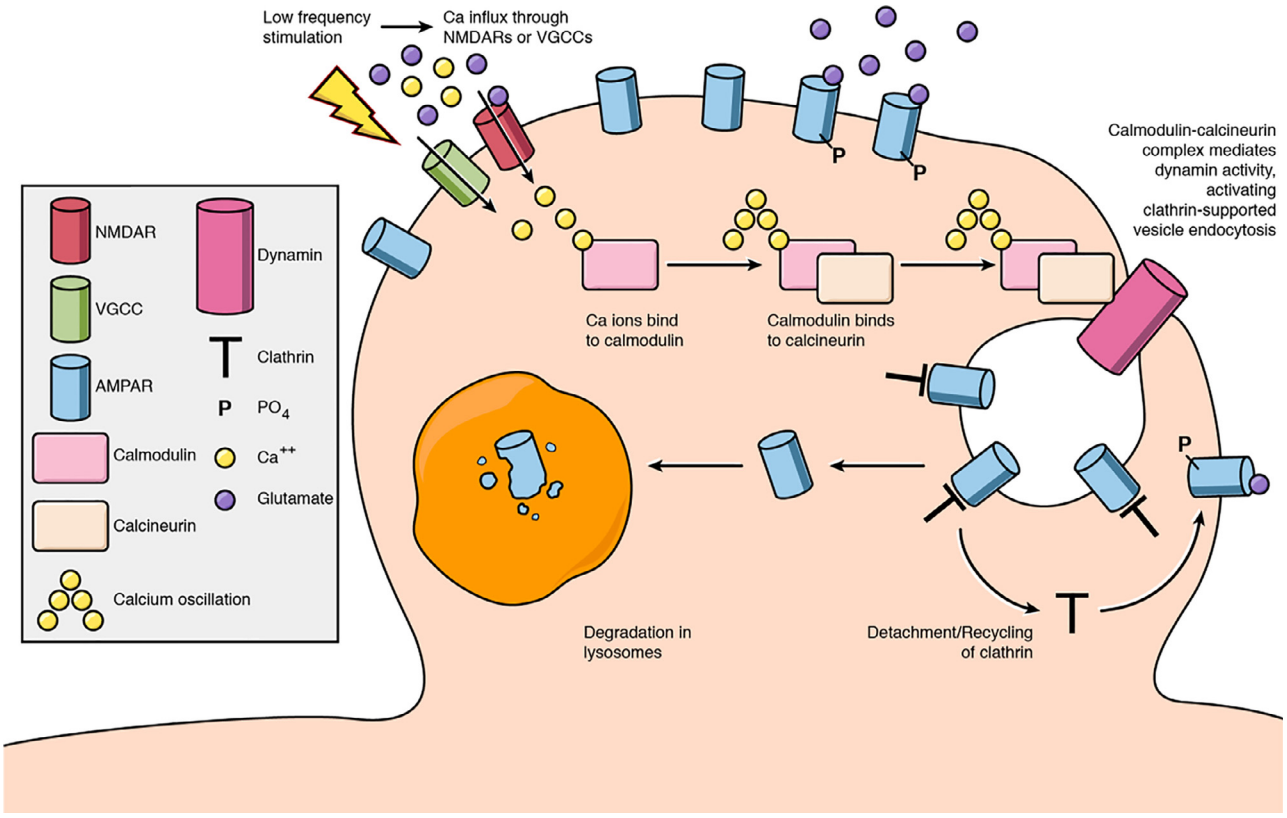

Fig. 3. Activation of the encoded pathway by recall (or inadvertently) via working memory releases glutamate at the surface of the lateral nucleus of the amygdala. This depolarizes the membrane via NMDA receptor mediated calcium influx and activates the AMPA receptors, exposing the anchoring phosphate. The VGCC channel can now respond to the low frequency wave generated by Havening Touch to produce a calcium oscillation that is decoded by calmodulin. This transforms the calmodulin into a protein that activates the phosphatase calcineurin. Calcineurin then dephosphorylates the AMPA receptor making it susceptible to depotentiation by proteins dynamin and clathrin. Once removed from the surface, the AMPA receptor is degraded by lysosomes and no longer available for transmission of information to the BLA.

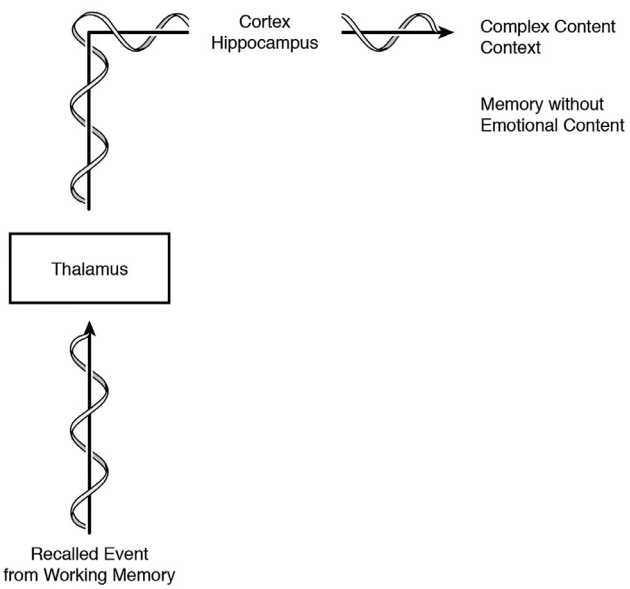

Fig. 4. Post-Havening.

or practitioner. Havening touch is a gentle and soothing touch, applied to the upper arms, palms and around the eyes.

Fourth and concurrently with Havening Touch is distraction. Distraction displaces the recalled event from working memory and prevents it from continuously activating the amygdala. Generally, this distraction also prevents other distressing thoughts from entering awareness, but not always. Distraction techniques can be visual, auditory or cognitive, such as imagining and describing walking on a beach, climbing stairs, chanting, humming a tune or counting backward. While the entire process might seem curious, its effects are almost immediate and profound.

The **fifth** element is a debrief. After the emotion-producing AMPA pathway is depotentiated (SUD=0) the practitioner has the client close their eyes again and asks them to try to recall the memory.

A FACILITATED EVENT SELF-HAVENING (EH) SESSION

The practitioner should rehearse the Havening Touch and distraction sequence before attempting to teach the client. As mentioned above, the touch component can also be applied by the practitioner and is called facilitated Havening. Videos on how to

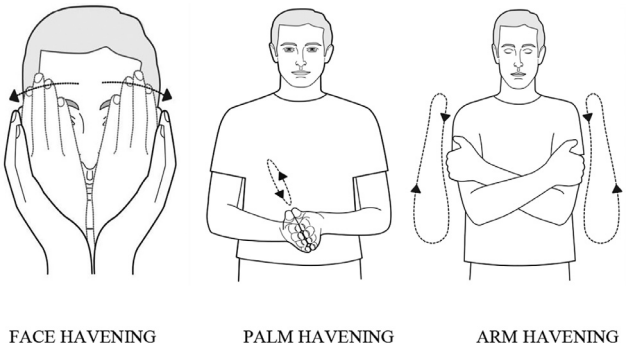

Fig. 5. Types of Havening touch applied by self (Self-Havening touch).

do this can be found on www.Havening.org. Once learned the following sequence should be followed:

Practitioner begins by saying to the client, "I would like you to close your eyes and bring to mind the memory that causes you distress along with its visual, emotional and other aspects of the event, as many as you can remember. Try to put yourself back in that moment." After a brief time, Practitioner asks the client to provide a SUD from 0 to 10 where 10 is extreme distress and 0 is the absence of distress, calmness. The client gives a number. Practitioner then asks the client to open and close their eyes. Then in a voice that is unhurried, supportive and friendly practitioner asks client to begin to apply Havening touch (Fig. 5). Then practitioner asks the client to visualize him or herself at the bottom of a staircase, and walk up the 20 steps, and with each step to count out loud, in normal speaking voice, the steps as they ascend. Practitioner states to the client, "Each step you take causes the distress to diminish and vanish and causes you to feel safe, peaceful and calm." The client should use Face, Arm and Palm Havening Touch.

After 20 is reached, client continues to apply Havening Touch and practitioner asks the client, "Please hum a familiar tune such as *Take Me Out to the Ball Game* or any other tune that you know." When the humming is completed, Arm Havening is continued and practitioner says, "Open and close your eyes and look at the back of your eyelids." The client is told to take a deep inhale and gently exhale. Practitioner then asks "What is the current state of the distress from 0 to 10 without thinking about anything other than my voice?" This sequence is repeated with different distractions until the SUD reaches 0 or remains stuck at a number. When the number reaches 0, have the client sit quietly. Practitioner says, "When you are ready you may open your eyes. You should feel calm and relaxed, you also may feel lighter." If the client is unable to bring the number down to zero, have the client close their eyes and ask them to reactivate the memory and look to see what is still lingering and repeat the process. The number should go to 0. If it does not, then further investigation into history is needed. This is often most difficult part as the event that produced the emotionally valenced pathway may not be the presenting concern.

Post-Havening discussions often uncover other events and feelings that require treatment. Careful and creative listening is essential. Usually, once changed, the recalled memory cannot change back to the original. However, if on debriefing distress remains, finding what is left and performing Havening on that aspect is necessary. A discussion of what is now imagined on recall is always useful and interesting:

POST-HAVENING OUTCOMES

A relaxed state is always seen after successful Havening, and retrieval of the memory is reportedly altered in one of six ways:

1. The UTS is no longer accessible by imaginal recall. The emotional content is changed or gone.
2. The memory is blocked and is inaccessible. This occurs when the entire event is included in the emotion producing content.
3. The memory is fuzzy and incomplete. This occurs when aspects are stored as complex content and context.
4. The memory is viewed from a distance and as if by a detached observer. The experience is of being emotionally detached, literally removed from the event. As time goes on, the ability to recall the moment fades into the distance.
5. The memory is richer in peripheral detail. With the emotional component gone, the complex content and context become clearer.
6. The memory is resolved metaphorically. This is the most remarkable of all resolutions where the mind solves the problem.

Other feelings may arise after Havening, for example, fatigue, spaciness, sadness (this occurs most often after Havening anger) and a feeling of lightness. These feeling can last from minutes to days. Once depotentiated, the emotion-producing memory and its co-encoded components are lost and cannot be reconstituted. This removes the allostatic load and alters the landscape. The client's facial expressions and posture are altered. Clients appear younger as the stress is removed from their bodies. This observation reflects the removal of internal distress that had been writ large on their physiognomy.

CONCLUSION

The evolutionary advantage of permanently encoding a threat is obvious. It allows us to become rapidly aware of potential danger through the mechanism of pattern recognition as the brain seeks similarity in novel circumstances. If a perceived escape or a safe place is not found at the time of a threatening event, the event may be encoded along with its components as a traumatic memory. This encoding has a unique biology that can produce life-long suffering. This paper describes a possible mechanism by which this encoding occurs. In addition, it proposes a technique called Event Havening that is speculated to generate a delta brain wave which has an electroceutical effect and leads to de-encoding of the event by synaptic depotentiation. Viewing the mind/brain/body as an electrical system with dysfunctional circuitry and rhythms suggests new approaches to treatment. The success of techniques listed in Group 1 (Table 5) most likely occurs by a similar mechanism. Event Havening holds promise to repair and restore pre-event homeostatic functioning. The biological and clinical expressions of traumatization are complex and incompletely understood, but once an encoded pathway is severed, both the memory and its consequences appear to be forever altered. Psychosensory Therapy, such as Havening has the potential to break the chains of remembrance so the past is no longer always present.

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