WHY TAPPING WORKS
Speculations from the Observable Brain

By

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ABSTRACT

A new therapy for phobias, PTSD, addictive behaviors and other psychological issues was first described by Dr. Roger Callahan and involves thought activation of the problem followed by tapping on certain acupoints in a specific sequence. In addition, a gamut procedure involving further tapping, eye movements and following simple commands is used. For most cases, the problems were reportedly cured in a matter of minutes. We speculate on a neuroanatomical and neurophysiological mechanism for this technique.

We propose that tapping and other sensory stimulation increase serotonin in both the prefrontal cortex and the amygdala. The success of this technique requires that glutamate be first increased in the circuit that involves the conditioning stimulus and the unconditioned stimulus. This analysis does not specify sequences for tapping and allows for other sensory stimulation to be used. We suggest the name 'Affect Activation/Sensory Stimulation' to encompass this general approach. AA/SS represents a paradigm shift for the treatment of these problems.

Key Words: Thought Field Therapy, Serotonin, Glutamate, Tapping, Amygdala, Prefrontal Cortex, Phobia, Post Traumatic Stress Disorder, Craving, Addictive Behavior

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INTRODUCTION

In 1986 Dr. Roger Callahan discovered that **tapping** under the eye of an individual with a water phobia immediately and permanently cured this problem *(Callahan 2001*). Dr. Callahan believes that activating a distressful thought produces a perturbation in the energy field that surrounds the body. His model is based on traditional Chinese medicine, that is, when energy flow is disturbed a person becomes ill. By tapping on specific traditional Chinese medicine acupoints in a specific sequence these 'Thought Fields' resume normal function and healing occurs. He calls his method Thought Field Therapy (TFT). Variations on this therapy have been developed and are available as web based documents. These therapies constitute a field called Energy Psychology (*www.energypysch.org).*

From an observational point of view, when TFT is applied, it literally appears that a switch has been thrown. After a successful treatment thoughts that had been clear were less so. Not only was the ability to generate a clear image diminished, the response to that thought was gone, and for good! Sometimes the individual felt euphoric, sometimes confused as to what happened, but always calmer.

A large study that involved over 29,000 patients was conducted using these procedures. The results *(Andrade & Feinstein 2003)* are remarkable. For a wide range of problems, such as specific phobias, panic disorders, post-traumatic stress disorders, acute stress disorders, and anxiety-depressive disorders this method was successful in 76% of the subjects. Also, in this category were a variety of painful emotional states including grief, guilt, anger shame, jealousy, rejection, painful memories and others. These techniques also seemed to help impulse control disorders and cravings. These researchers noted that most of the treatments did not require the special protocols developed by Dr. Callahan, rather they found that for most disorders one sequence sufficed.

Fear, anger, grief, depression, anxiety, aggression, cravings and other emotions represent a complex neurophysiological response that involves both cortical and subcortical systems. There are many ways to alter these systems. These methods include the psychotherapies, phamacotherapies, yoga, meditation, Electro-convulsive shock, acupuncture, hypnosis, psychosurgery, EMDR, stem cell implantation, biofeedback, systematic desensitization, neuroloinguistic programming and others. These methods variously affect the brain's electrical activity, the concentration of neurochemicals, the threshold to neuronal activation and the neural connections that are available. By its effects we judge that TFT must call forth similar responses.

A neurobiological model must explain several characteristics of this therapy. Firstly, why is it necessary to activate the distress before it can be treated? Secondly, why is the treatment specific, that is, if an individual has a snake phobia and an elevator phobia these problems need to treated separately? Thirdly, why does the same protocol work for many different problems? Fourthly, why does the distress appear to diminish during tapping (As measured by a decreasing SUD, Subjective Unit of Distress, a 0 to 10 scale where 0 is none and 10 extreme distress, as reported by patient) *(Wolpe 1958)?* Fifthly, what is the transduction event that converts tapping into a biological event in the brain? Lastly, how and why does this treatment produce a rapid and permanent change in an individual's response to the distressful thought?

THE AMYGDALA AND EMOTION

Neuroimaging *(Phan&Wager&Taylor&Liberzon, 2004),* lesional *(Cousens&Otto, 1998, LeDoux&Ciccheti&Xagoraris&Romanski ,1990 Blanchard&Blanchard, 1972)* and neuroanatomic *(Sah&Farber&Lopez De Armatntia &Powers ,2003)* studies point to the amygdala as the final common pathway for expression of emotions. The amygdala is well suited for this job. It receives input from the hippocampus, the prefrontal cortex, the thalamus, midbrain nuclei, and other cortical and subcortical areas *(Maren 2001)*. The amygdala is made of several nuclei; the basolateral (BL), the lateral (LA) and the basomedial (BM) make up the basolateral complex, the BLA *(Maren, 2001).* It is the lateral nucleus where the information from other areas are received. The associations between a conditioned stimulus and response are believed to be stored in the BLA and when appropriate a signal is sent to the Central (Ce) nucleus of the amygdala. Activation of the Ce is necessary to produce the behavioral, autonomic and endocrine components of an emotional response by activating other areas of the brain. (*Fig. 1*)

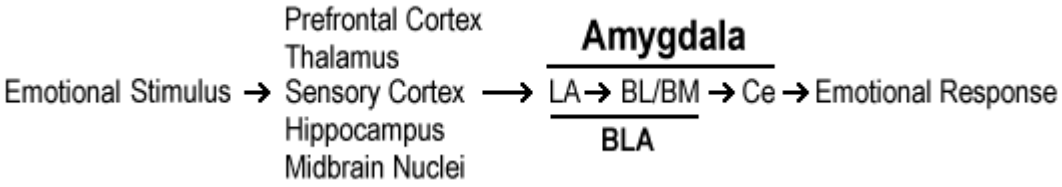

Fig.1

The Ce projects neurons to the nucleus accumbens, locus coeruleus, paraventricular nucleus, the hypothalamus, the prefrontal cortex and other structures. (Fig 2)

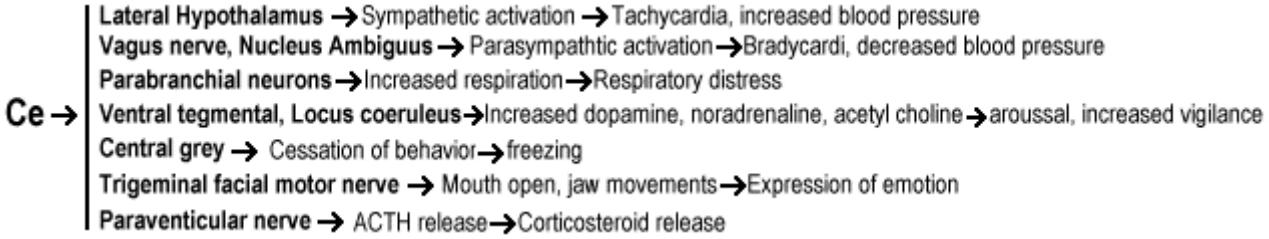

Fig. 2

Of all the emotional states we experience, none is more primitive or powerful than fear. If we understand how a fear response is disrupted, we may be able to understand how tapping works. For a model of fear we chose phobias.

ENCODING FEAR

In humans, facial expressions of fear are characteristic, easily recognized and involuntary. Evolution has crafted this form of communication to promote survival in the face of present and future threats. While fear has obviously been useful for survival, an inappropriate fear response can cause physiological changes that produce distress. Phobias are such fear responses and as such they provide no evolutionary advantage. Phobias are characterized by a persistent, irrational and excessive fear of objects or situations. Since there is no imminent danger associated with these objects or situations, they can be considered conditioning stimuli (CS). Phobias can be directed to anything: bugs, colors, numbers, light, dark, bridges, tunnels, elevators and planes. Not everyone develops a phobia. It has been suggested that a special genetic and environmentally modulated neurobiological landscape is necessary to encode a phobia.
(Gapenstand&Annas&Ekbolm&Oreland&Fredrikson. 2001). This unique moment would be almost impossible to reproduce. Treatment that disrupts the encoded phobic response may therefore extinguish it forever.

Phobias are learned and as such are fundamentally different than responses to innate fears. A fear response, (FR) is generated by sensing an innate fear, which are also called Unconditioned Fear Stimuli (UFS). Such stimuli, which are reflective of the fear of being killed, are hard wired in the brain and include: fear of the unknown (novel situations), heights (falling), closed spaces (being trapped), open spaces (no place to hide), creepy crawly things (land based predators) and something coming out of our visual fields (air based predators).

These survival stimuli do not reach conscious because details are unimportant, only the emotion of fear is experienced. Avoidance is mandated. Accordingly, the thalamus, which is the first sensory connection in the brain, has direct projections to the amygdala *(Doron NN & LeDoux JE. 1999)*. *(Fig. 3)*

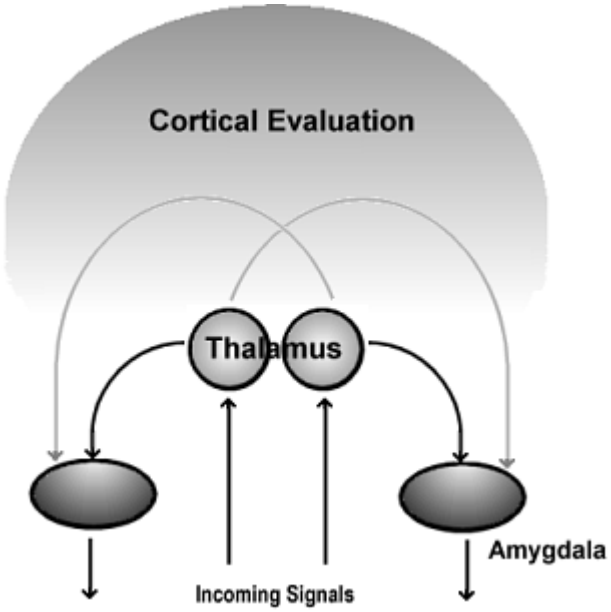

Fig. 3

An innate (unconditioned) fear stimulus leading to a FR in the presence of another object or situation sets the stage for the generation of the phobia. For example, traveling over a bridge (CS), you look down and see the height (UFS). It is the height that causes you to become fearful. This occurs at the subconscious level, you are not immediately aware why you are frightened, however, since you are consciously aware that you are on a bridge, if the neural landscape is primed, the bridge then becomes associated with the fear response. Thus, when you bring an image of a bridge to consciousness, you become fearful. (*Fig. 4*) It is important to note that not all CS that produce a fear response reach consciousness. Thus, in Panic disorder and PTSD much of the conditioning stimuli remain in the subconscious. These subconscious CS can still produce a fear response through the final common pathway, the amygdala, and fear makes us remember.

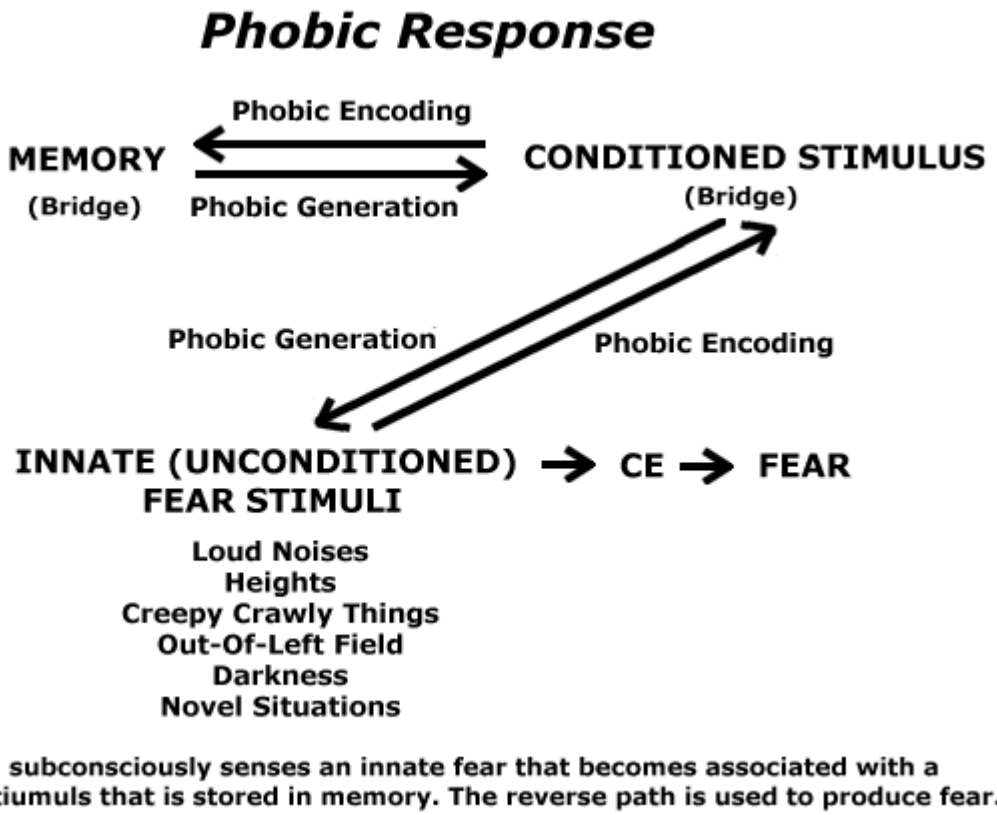

Fig. 4

NEUROPHYSIOLOGY

One laboratory model for the study of disorders of fear and its treatment is Pavlovian fear conditioning (*Maren S 2001)*. Fear conditioning occurs when a conditioning stimulus (CS), generally a tone or a light, is followed by and unconditioned fear stimulus (UFS), generally a mild foot shock. Conditioned fear requires learning and produces a stereotypical freezing behavior that can be measured and used for research purposes. After several pairings of the CS with UFS, the animal comes to react with fear to the CS. It is the anticipation of the shock (the tone or light) that produces the fear, not the shock itself. Thus, unlike phobias, conditioned fear is an appropriate response designed to increase survival. This association is felt to be stored in the BLA. Research data suggests that glutamate agonists enhance learning and glutamate antagonists inhibit the learning of the fear response in mice (*Myers KM, Davis M 2002).* Glutamate, an excitatory amino acid, is involved in activating genes that are necessary for memory storage and retrieval *(Reidel &Platt &Micheau 2003).* These genes alter the wiring and firing of neurons. This implies that glutamate is released locally where learning takes place. GABA, an inhibitory amino acid, inhibits glutamate and, as such,

GABA agonists inhibit fear conditioning and GABA antagonists accelerate it *(Myers & Davis 2002).*

While a phobia and conditioned fear are encoded differently, the association between the CS and the UFS in the amygdala is the same, leading to activation of the Ce and a fear response. Thus, information about the neurochemistry of fear extinction may be of help in understanding tapping.

EXTINCTION TRAINING

In the animal model, eliminating a conditioned fear response uses a technique called extinction training. Here, exposure to the CS is done in a non-threatening environment. During this training, learning takes place. These new pathways lead to a decrement in the fear responses from the CS. Extinction does not appear to be simple forgetting (where no, non-reinforced CSs are presented). In animals, if extinction training is carried out so that the CS no longer produces the FR, spontaneous recovery (recovery of response over time), renewal (recovery of response is CS is presented in a novel environment), or reinstatement (recovery of response after presentation of US in conditions where the US/CS link was forged) can occur over time.

In the animal model of fear conditioning, chemical approaches to extinguishing this response have been carried out. Thus, two animals were given a shock after a tone and this process was repeated until they froze in response to the tone. Then they both received infusions of anisomycin, a protein synthesis inhibitor *(Nader & Schafe & LeDoux 2000).* One animal received the infusion after the tone (where the animal froze) the other without the tone (no freezing). The animal that received the anisomycin after the tone no longer froze when exposed to the tone, permanently. The animal that received the anisomycin without exposure to the tone, still froze when the animal heard the tone. This experiment was repeated with a GABA agonist muscimol *(Muller & Corodimas &Feidel & Ledoux 1997).* As long as the muscimol was in the animals system, the animal that received the muscimol immediately after the tone did not react to the tone. The animal that received the muscimol without hearing the tone, froze with fear. The conclusions were that a fear response could only be disrupted shortly after being activated, that protein synthesis was involved and that a GABA agonist could temporarily disrupt the fear response.

Extinguishing a fear response has also been accomplished electrically. Wistar rats had electrodes placed in the dorsal raphe nucleus, the source of serotonergic projections to the brain. After being trained in a step down avoidance procedure, fear memories could be permanently disrupted by stimulation during post training

testing. This implied modification of associative processing. In another experiment, depletion of serotonergic neurons prevented the loss of fear. These results imply that serotonin plays a role in extinction *(Fiberger HC, Lepiane FG, Phillips AG, 1978)*

For humans, extinguishing of a phobia has been studied with a technique called Systematic Desensitization. This approach produces an extinction of the fear response (*Davis, M, Myers KM, 2002)* and uses the methods describe for extinction training in animals.

Research has documented a potential mechanism for these observations. A group of inhibitory neurons intercalated between the BLA and the Ce have been described *(Pare D, Royer S, Smith Y, Lang EJ 2003).* (*Fig 5).*

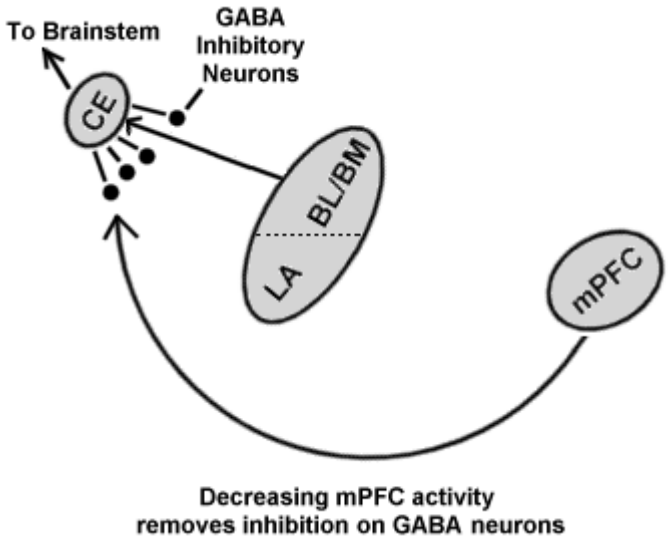

Fig. 5

Here, if danger is present, as evaluated by the prefrontal cortex, then an inhibitory signal is NOT sent to the inhibitory GABA neurons in the amygdala. If danger is considered minimal or absent, such as during desensitization, then the prefrontal cortex becomes unavailable to send a signal to these GABA neurons, allowing for activation of these inhibitory neurons and blocking the Ce→brainstem transmission *(Sotres-Bayon, F, Bush DEA, LeDoux JE. 2004).* This process makes sense in that it allows for conscious evaluation of danger. Desensitization does not affect the encoding of the response as it leaves the CS to UFS pathway intact thus allowing for reinstatement, renewal and spontaneous recovery to occur. Here as well, glutamate enhances and GABA diminishes the effectiveness of extinction training *(Davis M, Myers KM. 2002).*

WHY TAPPING WORKS

Using this information, we would like to speculate about a potential mechanism for tapping of the fear response. Tapping begins with imaginal re-activation (affect activation) of the feared object *(modified from Callahan 2001*) *(Fig. 6).*

TAPPING PROTOCOL

After affect activation have the patient take an SUD (Subjective Unit of Distress) and write it down. Then tap gently but firmly under the right eye 5x, over the right eyebrow 5x, under the right armpit 5x and just below the suprasternal notch 5x. The Gamut procedure follows by tapping on the back of the right hand, just behind the knuckle of the small finger while the patient does the following:

1. Closes eyes
2. Opens eyes
3. Looks down to the left
4. Looks down to the right
5. Rotates eyes in a big circle
6. Rotates eyes in the opposite direction
7. Hums Happy Birthday
8. Counts to five aloud slowly
9. Hums Happy Birthday

This sequence can be repeated. The patient then looks up, takes a deep breath, hold for a count of three and then rolls eyes to floor. An SUD is taken.
This process is repeated until the SUD no longer drops or goes to 1 or 0.

Fig. 6

We believe that 'affect activation' is the critical aspect for success of this method. During affect activation, we propose that glutamate is locally released in areas corresponding to the neural circuit that initially encoded the conditioned fear. Without local release of glutamate, no amount of tapping or sensory stimulation will be effective. Tapping or other sensory stimulation (Massage, eye movement, etc.) then causes a generalized release of serotonin via ascending pathways. This release is non-specific and global, that is, it is not related to the content or context of the feared object. *(Fig 7).* We speculate that serotonin release by multi-sensory stimuli is different than that seen in non-reinforced CS used in extinction training.

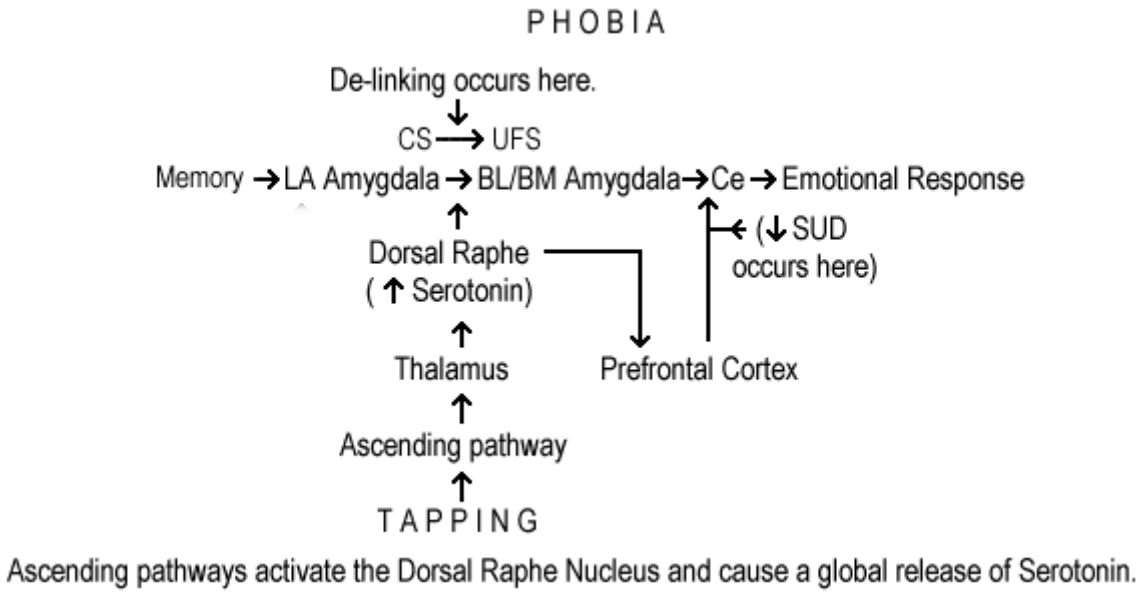

Fig. 7

During sensory stimulation, two events can occur. We speculate that serotonin decreases the inhibitory signal from the prefrontal cortex to the intercalated neurons and allows for GABA release, thus inhibiting Ce outflow and the patient experiences a decrease in distress (decreased SUD during treatment) *(Fig. 8).* It is again important to note that both the memory, as stored in the cortex and the connection between the CS and the UFS remain intact. This, retained memory CS →UFS pathway allows for renewal, reinstatement and spontaneous recovery.

Serotonin →Prefrontal Cortex →Intercalated GABA Neurons ==>Ce → Brainstem

Fig. 8

Simultaneously, serotonin causes GABA release via serotonergic receptors in the BLA. We speculate that this combination, GABA and serotonin, inhibits glutamate from activating protein synthesis, preventing the re-storing and thus de-linking the CS to UFS pathway. This blockade prevents the ultimate re-activation of the Ce and the fear response *(Fig. 9).*

GABA
Serotonin
CS → Glutamate → **X** (UFS)

Fig. 9

To better understand de-linking, imagine your brain is like a beach filled with holes (CSs). As a specific thought activates an affective (fear) response, a certain hole in the BLA fills with glutamate, this then links with a UFS and sends a signal to the Ce. During tapping, when a serotonin wave flows in, GABA is released and the glutamate filled hole and only the glutamate filled hole solidifies (protein synthesis is inhibited and the link to the UFS is disrupted). Since the hole is now gone, the ability to re-activate the CS to UFS link by glutamate release is lost. This also explains the broad-based effectiveness of this therapy as serotonin release is not localized, it interacts in both the prefrontal cortex and in the amygdala wherever glutamate has been released. If the de-linking does not occur, the prefrontal inhibition decays and relapse occurs.

Thus, bringing a phobia to conscious thought activates a specific glutamate driven circuit that produces a fear response. Tapping raises serotonin and GABA is released in the areas where the CS/UFS association is encoded, and the prefrontal cortex. This decreases the distress by directly blocking Ce outflow and can de-link the CS/UFS connection. After successful tapping, the ability to generate a sharp picture of the CS is diminished because the efferent transmission from the Ce, which increases salience, does not occur. How tapping is transduced to a rise in serotonin and GABA remains uncertain, but a simple mechanical process involving sensory receptors has been proposed *(Andrade and Feinstein 2003*).

CONCLUSIONS AND OTHER THOUGHTS

This model suggests that activation of the affect followed by sensory stimulation provides a neurobiological basis for tapping therapy. This model provides an outline that explains the permanence, specificity, ability to generalize to other types of affective problems (via amygdala de-linking) and the temporal relationship between activation of the affect and a successful treatment. In addition, it explains the observed decrease in distress during treatment. Animal studies have confirmed experimentally the relationship between activation and the lability of a fear response. If we consider UFS→Ce the final common pathway then de-linking the CS→UFS allows us to understand the ready treatment of different phobias, PTSD, and other primary amygdala based emotional states.

For phobias, PTSD, panic disorder and other emotional states the amygdala is the final common pathway. For disorders such as OCD, addictive cravings, depression, generalized anxiety, the amygdala is one of many inputs to another part of the brain that affects these behaviors. Thus, OCD has an abnormally functioning caudate nucleus and addictive cravings have an abnormally functioning nucleus accumbens.

For example, tapping an individual for an addictive craving produces only a short-lived (hours to days) benefit. This procedure does not change the underlying dysfunctional system that produced the behavior, only that specific connection that produces a Ce efferent signal. The underlying dysfunctional systems are permissive stressors that continually activate the amygdala for re-learning and relapse. Treatments that seek to correct the dysfunction either by medications, psychosocial intervention, or removing amygdala based (such as PTSD) problems therefore becomes important.

Among the major controversies present in the field of Energy Psychology, of which TFT is representative, is the location and sequence of tapping. While the neurobiological model does not require a specific sequence of tapping, sensory receptor density (location where you tap) may affect the rate and intensity of serotonin release. It is possible that any stimulation that affects the serotonin system can be used. Thus, tapping, humming, mind-full meditation, cognitive tasks, eye movements, probably acute temperature change and other may be of useful.

It is interesting to speculate why serotonin reuptake inhibitors are useful in the treatment of primary amygdala based disorders (PTSD, phobias, panic diorder and other emotional states). It is possible that the SSRI's, by increasing serotonin, alter information processing (Spoont M, 1992). This may prevent glutamate release in the amygdala or allow for the prefrontal cortex to send a no-danger signal to the intercalated neurons. Return of these psychological problems after removal of the drug (unless the problem is dealt with in another way) is usual.

There is a general approach outlined here, that is, **Affect Activation** (locally release glutamate)**/Sensory Stimulation** (globally raise serotonin) (**AA/SS**). The goal for this therapy then becomes how best to activate the affect and find the appropriate sensory stimulation for the individual. Herein lies the skill of the therapist.

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