Dr. Joaquin Andrade
NEUROHUMORAL MECHANISM OF TAPPING.

INTRODUCTION
Many clinicians have observed that different sensory stimulations yields some results on some persons and some disorders.
Sensory stimulation used are mostly kinesthetic, either external or internal (tapping, massaging or holding certain loci on the skin, adopting certain positions of the body, arms and legs, breathing, etc.), visual, external and internal, different protocols that include eye movements, blinking, etc.) and auditory-verbal, also external or internal, (counting, chanting, doing affirmations, talking about something, etc.). We have also explored the use of two additional senses, taste and smell as extra ways of inducing sensory overload.
Absolute requisite for results, is that a very specific representation of the problem (the core issue, as memory, sensation, emotion, imagination, etc.) is clearly present at the conscience at the same time that stimulations are made.
As most emotional disorders are vulnerable in a short time to these techniques, we have proposed the conceptual name of "Brief Multi Sensory Emotional Interventions".

DIFFERENT METAPHORS TO
EXPLAIN "WHY".
The obvious clinical results of tapping have been explained by many theories and metaphors, most of them never verified.
The existence of so called "meridians" and "points", the flow of an h "energy" along them, the "chakras", the existence of "disbalances" in the network, or "perturbations", etc.
So what do we really have, and what can we really verify?

The 14th century british philosopher, theologician and Franciscan monk, William of Occam (1285-1349), frequently used, in his arguments against papal power, the so called Principle of Parsimony or Simplicity: "Plurality should not be assumed without necessity"). The principle is used as a logical tool to eliminate pseudo-explanatory excesses and decide among explanations:one should always choose the explanation of a phenomenon that requires the fewest assumptions or leaps of logic. In any given explanation, "Occam's razor" helps us to "shave off" the concepts, variables or constructs that are not really needed to explain the phenomenon.
Being reasonably updated with the tremendous amount of information that the neurosciences offered us in the last ten years, and applying "Occam's razor", along with the Principle of Uncertainty Maximization, widely used in the field of mathematical modelling of systems, which states that in inductive reasoning, "use all, but no more than the available information", the mechanism of tapping can be explained with enough scientific rigor in about 80% of the cases.

AFFERENT SENSORY STIMULATIONS.
These techniques use the somato-sensory system, known for fifty years. Distributed all over the extension of the skin, but with different concentrations in different areas, there are sensory receptors specialized in receiving, transducing and sendind to the CNS all kinds of mechanical stimuli: the mechano receptors (Pacini, Meissner, Ruffini, Merkel's discs, free nerve endigs, etc.).
Mechanical stimuli on those areas (tap, touch, hold, rub, etc.) is transduced into digital signals mediated by the Ca ion (Guo, Miao, Shang,etc.) that travel by the afferent somato sensory pathways to the brain.

Mediation of Ca ion in signal transduction can be demonstrated by the supression of signal registration after the local injection of a chelating agent (Andrade et al,preliminary studies).
One thing we can be sure about is that every time we tap or do any other kind of mechanical stimuli to any area of the skin, we are involving the mechano receptors.
In other type of interventions, such as collar bone breathing, excess of energy correction and Wayne Cook posture, there are also involvement of muscle Golgi-Mazzoni proprioceptors and joints uncapsulated receptors.

THE PATHWAY.
Travelling by regional nerve trunks, A Beta myelinated axons, that end in dorsal roots and ganglia (I neuron), the signal reaches the medulla. Due to upper and lower, intra and inter dermatomic bifurcations, stimuli initiated in different anatomical localizations can end on the same pathway, which could partially explain why different protocols get similar results...The signals travel upward through the medulla, contralaterally following the median lemniscus (II neuron) and ipsilaterally along the dorsal medulla and reach the thalamus, where they synapse in the anteroposterior lateral nuclei and finally ascend by the cortico-thalamic neurons to the somato sensory cortex at the parietal lobe, the four Brodman areas: 3b, 3a, 1 and 2.
The thalamus modulates the afferent sensory inputs, widening or narrowing the focus to increase the transmission in relevant areas and to inhibit the non relevant signals under those particular circumstances. From the thalamus, particularly from its auditive area (LeDoux, Woodson) neurons are sent to the amygdala, that synapse with GABAergic inhibitory interneurons of the lateral nuclei of the amygdala.

AT THE CNS.
From there, signals travel to cortical areas of higher hierarchy, including the prefrontal cortex, and to deeper limbic structures that have high significance in emotional modulation.
Several studies of functional brain imaging (Hui et al, Andrade et al, preliminary report) have repeatedly demonstrated that when different types of mechanical stimuli are applied to the skin, fluctuation of signals can be registered at least at two different cortical areas distinct from the somatosensory cortex:INCREASE OF ACTIVITY in the orbito frontal regions, at the basis of the prefrontal cortex and at the posterior thalamus; and DECREASE OF ACTIVITY in at least ELEVEN deep structures, some of them strongly involved with emotional processing: Hyppocampus, parahyppocampus, hypothalamus, amygdala, putamen, caudate nucleus, anterior insula, cyngulated anterior gyrus, ventral tegmental area, nucleus acumbens and temporal pole.
The amygdala acts as a paralell processor, receiving, in one of its subsystems, the basolateral nuclei, (where the stimuli that condition the fear response are stored –Maren-), monosynaptic projections from the dorsal hippocampus, the thalamus and the cortex. The other important subsystem of the amygdala is the central nucleus, efferent, that sends signals to different brain areas involved in the autonomic and behavioral responses to fear.
Ruden notes that as the central nucleus of the amygdala projects fibers to different CNS structures related with stress, trauma, mood disorders and addictive behavior, functional actions of tapping interventions on them can be predicted, strictly based on the existence of those neural paths.

EXPERIENCING EMOTIONS.

Actually experiencing an emotion,or recalling a past emotional memory as we do when tuning with the problem, INCREASES the activity at the amygdala and other limbic structures, as can be easily demonstrated with different imaging equipments.
Just recalling a traumatic memory (which includes a superposition of visual, kinesthetic, auditory, smell and taste perceptions) places that memory in a labile state which is sensitive to disruption, as have been suggested four years ago by a group of fear researchers at Prof. LeDoux lab at NYU (Muller, Izquierdo, Brioni,Nader, Schafe, Debiec and others). To retain the memory's consolidated state, protein synthesis at the LBA, or lateral basal nuclei of the amygdala is required. So, reconsolidation after reactivation and consequent lability to disruption, is a protein synthesis dependent state.The researchers injected the protein syntesis blocker anisomycin (aniso) to prevent reconsolidation, and the memory, in its labile
state, was disrupted.
Then, recalling a traumatic memory that is capable of producing anxiety symptoms, and of INCREASING the activity at the amygdala and other structures, and while holding
this "increased activity state" , I send to these same areas emotionally neutral sensory perceptions (like tapping on the skin) these neutral sensory signals that are capable of DECREASING the activity at the amygdala, act on the not so neutral sensory perceptions that are an important part of the traumatic memory and probably by synaptic inhibition (DECREASE of the activity) disorganize, interfere and add entrophy to the previous increased-activity emotional state, collapsing its symptoms-generating ability. Bandler´s empirical work on sub modalities suggests that when we change the sensory perceptions of a traumatic memory, its symptoms-generating power also weakens...sometimes, almost instantly. Flint also suggests that sensory stimuli and other interventions, like Process Healing, act by generating a learning process, which in part
is also neurologic, that changes the relation between traumatic memory and emotions.
The lability and vulnerability to distortion that memories exhibit when retrieved has probably two evolutionary purposes: first, the possibility of the progressive generalization of the response, as new associations can be made, which improves the probability of surviving, and two, the possibility to direct attention to relevant aspects of the environment, which also helps to avoid or neutralize dangers (Ekman, Frijda, Izard, Derryberry and Tucker)
It is not that memories (traumatic memories to our effects) are being erased, which, as suggested by recent research by Lattal, Abel et al at Penn's Dept. of Biology, rarely happens.
It's more that they remain stored but have lost the power to generate symptoms i.e. disruptions..

THE INTERVENTIONS.
From a neurohumoral perspective, all tapping systems have two distinct components: 1. Reactivation of the memory to make it vulnerable to distortion, and 2. Simultaneous sensory overload, that sends afferently, modulated signals also sensory in nature, but with zero emotional meaning. Those signals, probably disorganize, overload, interfere, and add entropy to the memory, which loses its power to generate symptoms, even when is never deleted.
Different eye movemets, auditive and verbal inputs, as well as olfactory and gustatory signals follow a similar model, travelling by shorter and less complicated neural paths.
Ruden suggests that the sensory interventions distort signal transmissions between the amygdala basolateral and central nuclei,

blocking the generation of fight and flight symptoms.
It is possible that the neurotransmitters GABA, serotonin and the intraneuronal second messenger nitrous oxide play critical roles in this mechanism, through complex feedback and feedforward functional interconexions. Maybe that's why lab tests in several anxiety disorders show elevated norepinephrine and low GABA and serotonin.
The GABA system in particular, plays a crucial role in anxiety control. It is known that benzodiazepines link to specific sites of the GABA receptors and enhance its mutual afinity, which results in an activation of the Cl ionic channels, and that the GABA agonist muscimol inhibit the fear response (Muller, Izquierdo, Brioni, Stork).
As most functional events in the CNS are at the same time or sequentially ionic or chemical, in which depolarizations are almost always accompanied by ionic movements of Cl, Na, K, Ca and elevations or decreases in neurotransmitter concentrations, this mechanism is called neurohumoral.

WHAT FOR?
The neurohumoral theory of tapping mechanism is no more than another metaphor. It doesn't exclude other explanations.
It also has six distint advantages:

1.- It's based on classical concepts and updated research on CNS.

2.- Speaks the language of science and is perfectly well accepted by MD's, Ph D's and other scientists.

3.- Follows Occam razor, tries to have logical coherence.

4.- Offers many lines to research.

5.- Gives logical explanation for relapses.

6.- Same with non respondents.