# A PTSD model of muscle dysfunction: a neurobehavioral approach to muscle and joint dysfunction and pain offers new treatment options.

Robert Weissfeld D.C., C.N.T.1[§*] , Peter Litchfield Ph.D.2

1 Private practice in Denver CO, area 80210

2 Unafilliated,

[§]Corresponding author

Email addresses:

RW neurontogenics@gmail.com

## Abstract:

## Background

The treatment and assumed causes of routine musculoskeletal dysfunction (MSD) and pain not related to organic disease or recent injury are generally based on anecdotal mythologies of the subspecialty of the treating practitioner. Though each may clinically utilize manual muscle testing, the explanation of weak muscles will vary depending on whether the practitioner is orthopedically, neurologically or (applied) kinesiologically

oriented. We suggest a universal mechanism of muscle weakness, which may lead to more effective treatments for MSD.

## Methods

Clinical observation of bodywide changes in muscle function from certain treatments, a small pilot study which observed improvement in muscle function from eye movements as used in Eye Movement Desensitization and Reprocessing (EMDR) and a case study along with literature review and behavioral analysis are offered to understand and substantiate a neurobehavioral hypothesis of musculoskeletal dysfunction.

## Results and Discussion

The pathogenesis of MSD may be characterized by a condition we refer to as *musculoskeletal posttraumatic stress disorder* (mPTSD). Muscle imbalance and resulting joint dysfunction, postural distortions and pain, we hypothesize, are often the result of learned strategies of adaptation to trauma or stress, and subsequent compensations to those strategies. Based on this hypothesis and the above method, principles by which optimal muscle function may be immediately restored without retraining or therapeutic application to the involved areas are introduced and explored. Relevant neurophysiological and behavioral mechanisms involved in creation of adaptive strategy and learning, memory and conditioning are discussed. The benefits of various treatments, including acupuncture and spinal manipulation may, in some cases at least, be the consequence of unwitting manipulation of the neurobiology of memory. This may occur by disruption of reconsolidation of conditioned somatic memories, which have been demonstrated to become

labile and subject to disruption following activation. We explore the relationship of norepinephrine (NE) to this process and explore the effect of various therapies on NE.

## Conclusions

N*eurontopathic medicine,* as we introduce it, is a novel treatment approach with a putative mechanism and an evolving set of 'laws' that govern its application. We contrast it with allopathic medicine approaches and discuss its implications for clinical practice.

# BACKGROUND

About one-third of all primary care visits are for musculoskeletal pain,[1] and such pain and the accompanying musculoskeletal dysfunction (MSD) is an equal opportunity employer across many provider specialties. Most who treat MSD non-surgically think of their work as providing either mechanical, energetic or neurological treatment or neuromuscular retraining. Mechanical treatment includes aligning joints, breaking adhesions, stretching tight ligaments and muscles and tightening loose ligaments (i.e. prolotherapy) or muscles (strength training). Energetic /neurological treatment includes acupuncture, electrical stimulation, TENS, low level laser or spinal manipulation to address nerve interference. Retraining includes neuromuscular reeducation or muscle retraining, retraining balance via balance board or ball exercises, resistance exercises, or EMG biofeedback.[2] Each of these treatments, while potentially worthwhile, are based on assumptions which may not be

universally accepted, and may or may not have clear scientific models or efficacy studies behind them.

So with this wide variety of treatments, how well do we really understand MSD? We understand, to some extent least, *what* happens in joint failure, we know about inflammatory process, adhesions, joint degeneration, ligamentous laxity; we have names and generally accepted, but not generally evidence based treatments for tennis elbow, patellofemoral syndrome and shin splints, and we generally attribute them to overuse, wrong shoes, or wrong sports technique, though attribution in any patient is usually an educated guess or based on the understandings of the practitioner's specialty. We also know the nerve pathways, and understand where entrapments may occur, and we can chart out sclerotogenous, dermotogenous, and trigger point pain patterns, but the observation in clinical practice is that often the symptoms don't fit these patterns.

We understand the human body to have 17 separate segments that are linked together by soft tissues.[3] The motion of any one body segment can influence any or all of the other segments. Movement never involves just a single muscle and coordinated motion of multi-joint systems involves neural connections which are distributed among muscles based on their common function.[4] We further understand general and specific details regarding central nervous system integration of sequential and combined activations of muscles into specific behavior, (muscle synergies) and how intentionality and planning of muscular strategies occurs in the brain, (central motor programming) appropriately planning, 'rehearsing', coordinating and timing the muscle synergies for subsequent behaviors.

Depending on whether a condition is seen by an orthopedist, occupational medicine specialist, physiatrist, acupuncturist, chiropractor, physical therapist, athletic trainer or myofascial therapist, however, MSD treatment can be vastly different, as each specialty and even each practitioner brings different assumptions to the treatment table. Much of what we think we, as clinicians, know about muscle function is based on anecdotal mythology of our specialty. Even if these treatments are beneficial for a patient, the mechanisms are often a matter of conjecture based on "logical argument" and there may be little evidence that the treatment modality actually improves treatment outcomes.[5] Tendonitis, (tendon inflammation), for instance, is frequently diagnosed and treated, yet it is generally a misnomer, as inflammation is rarely found on tendon biopsy. [6,7]

All specialties, however, are likely to agree that a change in the function of muscles supporting the involved joints is a feature of all MSD. Palpatory examination even on asymptomatic joints will reveal some muscles to be relatively flaccid, while others will be in relative spasm, and both varieties may be weak if tested.

Most non-surgical physical specialties utilize some manner of treatments referred to as retraining, offering repetitive treatment or prescribing repetitively done exercise for patients. This may take the form of repetition of a particular exercise for purposes described as strengthening or stretching and may include repeated joint or myofascial manipulation. Such retraining taken to its logical end contains the inference that that the body has somehow lost or forgotten its normal function and needs to be re-educated, or lost its internal structural model and needs to be remodeled or otherwise pushed and cajoled back to correct posture. Chiropractic alone historically

incorporates the concept, at least, of restoring an "innate intelligence" of the body, an understanding that would assume that correct function has not been lost. Such restoration of innate function is the opposite of retraining, yet an individual chiropractor may espouse both, repeating adjustments to retrain the spine *and* restore the function of innate intelligence. Our hypothesis, which suggests neurological mechanisms for the loss and ultimate recovery of innate function, will offer new perspectives to this discussion.

This paper proposes a new hypothesis that asks for a radical shift in the way we view the etiology, maintenance and treatment of MSD and MSD related pain based on the understanding of conditioned learning, which is part of all MSD and, we will assert, not necessarily eliminated by most existing physical medicine procedures.

To understand and support such a paradigm-shifting hypothesis, and because the hypothesis brings together several diverse systems of understanding, we will present, in this order (following introductory statements) 1) a listing of clinical observations and a classical behavioral analysis of those observations, 2) a pilot study that objectifies those observations, 3) a case history from that study, 4) a review and synthesis of relevant neurophysiological data regarding the establishment, learning and repetition of (mal)adaptive and compensatory conditioned motor function, 5) a presentation of factors that may lead to altered function, 6) a neurophysiological analysis of relevant aspects of memory which leads to 7) a presentation of a secondary neurological hypothesis that provides a mechanism by which such results may be achieved and 8) a literature review that suggests how chiropractic, acupuncture and other treatment modalities may each affect memory to achieve their therapeutic effect.

The restoration of a pre-conditioning (before the onset of conditioning), more fundamental (innate) behavioral state requires application of therapies in a manner that directly and purposefully addresses what we propose is a primary factor in the etiology and *the* primary factor in the maintenance of MSD and MSD-related pain. This inquiry will synthesize research and understandings from the fields of classic behavioral psychology, traumatology, neurobehavioral physiology, energy psychology, applied kinesiology, chiropractic, physical therapy and acupuncture. Taken to its logical conclusions, the hypothesis necessarily challenges assumptions of some disciplines. But where it challenges, it also may suggest to the creative and perceptive practitioner new ways of using their own particular toolset to interact more effectively with the body and improve their results with patients.

When clinical findings by the author, and experimental data and a case history are viewed with existing understandings about neurobehavioral aspects of post traumatic stress disorder (PTSD), a new view of the pathogenesis of MSD come to light. **An answer to the question of the cause of routine muscle weakness found in MSD may lie in specific interactions of pain and trauma with systems that create, learn and repeat motor conditioning.** Understanding the cause of muscle weakness, we will show herein, leads directly to effective new treatment modalities, which may be easily demonstrated in a clinical setting.

### Post-traumatic adaptation

Learning and memory play a major role in PTSD. According to van der Kolk,[8] PTSD includes "psychological effects of trauma [that] are stored in somatic memory

and expressed as changes in the biological stress response." "Intense emotions at the time of trauma initiate the *long-term conditional responses* to reminders of the event, which manifest as amnesias and *hyperamnesias*." [Italics added.] Unassimilated somatic responses evoked in trauma involving both arousal and defensive responses are shown to contribute to many PTSD symptoms. [9] We hypothesize that routine MSD may share these neurobehavioral mechanisms, and also include other mechanisms related to creation, learning and memory (conditioning) of individual, specific adaptations to pain, stress and trauma .

Adaptation as a concept was popularized by Hans Selye, who hypothesized that systemic stress elicits a generalized (neurohormonal) adaptive reaction, one that is common in many organisms. Selye mostly spoke of non-specific reactions to stress.[10] We, however, will examine mechanisms that may create and maintain *specific* adaptations, particularly those related to MSD, postural adaptations observable in humans as individualized patterns of altered muscle facilitation (weakness and strength). We will examine how these specific, individualized adaptations are established and persist following injury due to the ability of the body to establish new patterns of muscle activation for new conditions and remember past outcomes in order to predict future outcomes.[11]

### Muscle function and testing

In the early 1980's the author (Weissfeld) studied with Alan Beardall DC, who introduced concepts and methods he called Clinical Kinesiology (CK) which purported to address such specific, individualized adaptation. CK evolved from the

field of Applied Kinesiology, (AK) [12] an alternative medicine discipline that provides feedback on the functional status of the body using the strength and weakness of muscles. AK and CK and other systems of *applied kinesiology* as they are generically known, diverge from the academic field of kinesiology - the study of human movement and its applications - as taught in many universities. ('AK', from here on will generically refer to all such systems.) AK systems draw many inferences from the muscle test beyond the simple observation of muscle weakness or strength. The question of the validity of inferences drawn from AK muscle testing is not one that we will address. Though Weissfeld's observations of muscle function came in the context of AK, **this paper will view muscle strength or weakness from its basic *behavioral* face value: a weak muscle is an observable *behavior* which, (in the absence of organic pathology) necessarily follows behavioral principles**. Our hypothesis is based on behavioral and ultimately neurobehavioral analysis, and results we speak of are generally achievable *without* the application of AK procedures.

Likewise, we put aside other physiological assumptions based on neurological explanations for weakness such as central or peripheral nerve pathology, organic muscle disease or torn muscles, which are fairly rare in clinical practice. Stripped of inference, weakness is simply the inability to perform the particular behavior of resistance to force. **Because muscle activation, (weakness or strength), <u>is</u> behavior, it is always affected or determined by factors that determine behavior, including motivation, perception, cognition, emotion, learning and memory**.

The pilot study we present, and most of the conclusions we draw, are free of the assumptions of AK and in fact offer an alternative view of some of the AK's precepts.

The central hypotheses we will present will, by and large, be supportable by that study. Weissfeld's clinical observations over the course of over 23 years of private clinical practice are noteworthy however, as it was the strength and consistency of those observations, and the development of methodologies that made such results predictable and repeatable, which led to the pilot study. The pilot study then led directly to the development of a set of hypotheses based on its results, and further developed by literature review.

Before we present the observations, we offer several definitions important for understanding those observations. The study we will present and the clinical observations are based on manual muscle testing (MMT), which is a well-accepted method of evaluating functional, non-pathological, non-radicular, and non-organic injuries, and is used in most neurological and orthopedic disciplines. [13, 14] Isometric testing was used in the course of the observations and the study. We define a weak muscle as one that is *unable to lock* in a static position to moderate manual pressure. The isometric test yields a binary result, as opposed to the 5-point scale used to grade neuromuscular disease, [15] not generally not the cause of routine MSD. The 5-point scale is of limited use in routine MSD, because most weakness found therein is only a 4 of 5 on that scale.

The isometric test, properly instructed and performed, has the subject hold the limb in place, resisting whatever pressure is applied, not applying counter pressure against the hand of the tester. The subject responds with the same pressure as the tester is applying. Performed in this manner, isometric testing removes some of the gray area in testing, because the test is not based on judgment of maximal contraction. The

attempted application of maximal force brings up a question of the subject's focus and willingness to apply maximal force. Attempted maximal force also increases the chances of the subtle changes in position by the subject to recruit accessory muscles. This may be missed by even the most experienced testers. These factors are magnified in cases where the test causes pain. Likewise, the tester has to make a qualitative judgment regarding the amount of force that is being applied and the amount of resistance that is coming back, unless pressure gauges are used, and they are not practical for a busy clinical practice that may test and retest 10-20 muscles in a single visit.

While a strong muscle will certainly lock to greater force, the discovery of unlocking requires far less than maximum force in most individuals. We will refer to the unlocked condition as *muscle inhibition* or the more generally used term 'weakness', though inhibition is more technically accurate. *Altered muscle facilitation* includes either muscles that are inhibited, or muscles that do not appropriately exhibit the reverse myotatic reflex [16] – temporary weakening in response to manual stretching of the belly or tendon. Such a muscle is locked in strength and unable to appropriately weaken. (The reverse myotatic reflex is a protective spinal reflex, which diminishes the contractile force of a muscle when over-stretch is perceived via the muscle spindle cell or golgi tendon organ stretch receptors in the belly or tendon of a muscle, respectively.) We will revisit the significance of this reflex later.

### Clinical observations

Weissfeld, over many years, has made a number of clinical observations about muscles and the changes in muscle facilitation that occur from treatment using methods largely derived from AK and CK. Because of the methodological specifics of these methods, such observation may not be widely shared by kinesiological practitioners. While many of these observations were initially baffling to the author and continue to seem strange on the surface, they become understandable when biological principles of learning and memory are applied. The observations are as follows:

1 **Muscle re-facilitation**. *A weak muscle will predictably strengthen greater than 95 percent of the time when appropriate treatment is applied immediately after testing.* This testing followed by immediate treatment is referred to by Weissfeld as *targeting*. The targeted muscle strengthening occurs from the application of diverse treatments indicated in the AK system as applied by Weissfeld. The immediacy of treatment following testing appears to be a key factor for the success of the targeting protocol.

2 **Postural generalization.** *Targeting, treating and strengthening a single weak muscle, when that muscle is selected and treated with appropriate protocols, while immediately changing the function of that muscle, may also alter the function of several muscles, or it may alter body-wide patterns of muscle facilitation to a state significantly different from initial patterns.*

3 **Treatment generalization.** *The changes in local or body-wide patterns of muscle facilitation often arise from what would might be considered to a*

*trivial treatment, an application apparently unrelated and distant from the targeted muscle or area of complaint.* Examples of the treatments are: a 5 -10 second microcurrent or laser stimulation of one or two acupuncture points, one or two spinal or extremity adjustments, or 20-30 seconds of myofascial treatment administered to a muscle distant from the targeted muscle. In addition, a diverse variety of treatments may all have similar effects on muscle facilitation. We hypothesize that such perplexing outcomes can be accounted for if muscle strength and weakness is understood to be centrally organized and orchestrated.

4 **Intelligently patterned organization.** *Occasionally, initial or subsequent (post treatment) patterns of muscle facilitation may have a discernible and unusual organization; i.e. all flexors weak and extensors strong in an entire limb, all muscles in one limb inhibited, classic reciprocal gait facilitation patterns in a supine patient, or weakness of left knee muscles when the right knee is symptomatic.* Such patterns likely represent an organized behavioral response, again, most likely centrally orchestrated.

5 **Pattern resolution.** *Following treatment of a targeted muscle, such organized patterns are most likely to change to random ones, and random patterns will display a decreased level of altered facilitation, though occasionally application of treatment results in more altered muscle facilitation than before the treatment.*

6 **Symptom resolution.** *The local and body-wide changes and decreases in altered muscle facilitation following treatment are often accompanied by a*

*change or reduction in the patient's symptoms either immediately or incrementally over a number of such treatments that address each subsequent facilitation pattern that emerges.* The decrease in symptoms often occurs even if there was no direct treatment to the area of complaint. This is true for both acute and chronic conditions.

7 **Muscular-emotional equivalence.** *If patient focus on a stressful memory or painful emotion is substituted for the muscle challenge, and similar treatment protocols are applied, the level of psychological pain as measured by visual analogue scale (VAS) or subjective units of discomfort scale (SUDS – non-visual 0-10 rating of intensity) is often immediately and significantly reduced.*

Reflection on these observations brings forth a number of questions.

What neurophysiological mechanism could explain rapid body-wide muscle facilitation changes?

How does relatively minimal treatment distant from the area of complaint change function and symptoms in the area of complaint?

What can account for the immediacy of the changes in both function, and often symptomatology, again from minimal treatment distant from the area of complaint, a response that occurs even in acute cases where tissue damage is often assumed?

By what mechanism can the same treatment protocol apply to both emotional and physical pain?

### Behavioral analysis

The seven observations, listed above, at first glance defy explanation. Therefore, before we delve into the central hypothesis of this paper, we will look at these observations based on the well-established precepts of behavioral analysis. This behavioral analysis, while ultimately not necessary to understand of the mechanisms of muscular activation, is included in the interests of rounding out and understanding the larger context of behavioral study, and demystifying the observations under the light of a well-accepted discipline. In the final analysis, however, the ***neuro***behavioral hypothesis of muscle dysfunction that we will present is an evolutionary departure from classical behavioral psychology, and thus a portion of this analysis will be contradicted as look at the neurological underpinnings. As analogized to a car, behavioral psychology refers to observation of the behavior of the car when the gas pedal or brake pedal is pushed and how the position of the gear shift may change that behavior. Understanding the activity of the fuel injectors, transmission, engine and gas pedal linkage are not relevant to observing that behavior.

"Behavioral physiology" understands behavior as fundamentally psychological in nature. Behavioral physiology studies principles that govern how physiology acquires and processes information, both about "itself" and about its environment, and how it makes use of this information for self-regulation and biological homeostasis. In behavioral terms, this processing constitutes what is known as "learning," and includes the principles of attention, motivation, emotion, memory, sensation, perception, and reinforcement. Learning is a fundamental life process of physiological reconfiguration. In fact, all living things, from single cells to complex

physiological systems, learn. Classically, behavioral physiology is studied as separate from the neurophysiology that may underlie it, an important distinction for this paper because as we will see later, an observed behavioral change may look quite different when viewed through the underlying neurological processes.

The principles of biological learning can be easily, perhaps even elegantly, applied to patient responses to CK assessment and treatment techniques and can explain what might otherwise be considered "mysterious" outcomes, outcomes that might otherwise also require equally mysterious explanations. "Applied behavioral analysis," as it is known in clinical circles, can explain all of the apparently odd outcomes reported by Weissfeld and likely those of other applied kinesiology techniques. An introduction to terminology and a behavioral analysis, based on the principles of classical conditioning and operant conditioning and their interaction, is presented below. Since the preponderance of physical medicine training and practice does not include it, we include a review of basic behavioral terms. These behavioral principles are in play in *every* dysfunction and clinical interaction, and yet are often overlooked by researchers and clinicians in both conventional and alternative medicine.

**Classical conditioning** (also known as Pavlovian conditioning) is understood as part of basic biology. When STIMULI predict "meaningful" events, the result may be classical conditioning. An event, the **unconditioned stimulus (UCS)**, **elicits** an unconditioned response or reflex (UCR). A version of this response attaches to the predicting stimulus. When this happens, the stimulus is said to be a **conditioned stimulus (CS)**, and the response to this stimulus, becomes a **conditioned response or**

**reflex (CR)**. An example is **aversion conditioning**, where alcohol is associated with a drug (the UCS), that results in vomiting (the UCR). The taste of this alcohol (CS) now produces nausea (CR). Presentation of the alcohol (CS), however, without the drug (UCS), ultimately results in **extinction** of nausea (CR). Extinction, as a behavioral term, means the diminishment of a conditioned response, specifically the reduction of a behavior, over time, that does not receive reinforcement. Extinction of the behaviors of postural alteration from a sprained ankle occurs after the pain subsides.

Other key classical conditioning principles relevant to the present analysis include conditioned inhibition, external inhibition, and disinhibition. **Conditioned inhibition** is the classical conditioning of inhibitory responses, e.g., heart rate suppression, rather than an excitatory response, e.g., heart rate acceleration. **External inhibition** is the disruption of an excitatory response through the introduction of a new stimulus event into the perceptual field; the CR simply fails to appear. **Disinhibition**, like external inhibition, is the disruption of a classically conditioned inhibitory response through the introduction of a new stimulus event; the conditioned inhibitory response fails to appear.

Examples of classical conditioned physiology include blood sugar regulation, drug tolerance, withdrawal responses associated with addiction to drugs, immune system suppression, organ rejection, single cell learning, among many others. Emotional classical conditioning, negative and positive, includes conditioned fear, anxiety, panic, sexual arousal, hunger, sleep, wakefulness (alertness), and likes and dislikes of all kinds (e.g., places, people, and activities). Muscle responses, like salivation in

Pavlov's dog studies, can be easily and quickly classically conditioned to stimuli, such as specific emotional, social, and physical experiences, (specifically including *pain*).

**Operant conditioning** is basic biology (also known as instrumental conditioning). When RESPONSES predict events, operant conditioning may be the result. Learning takes place if the event predicted has "meaning." If the event ***increased the likelihood*** of the response, it is said to be **"reinforcement**," negative or positive. A **positive reinforcement** is the occurrence of an event (e.g., receiving money), and **negative reinforcement** (not to be confused with punishment) is the termination of an event (e.g., diminution of fear). The learned behavior is known as an **operant response**. **Punishment** is an event that ***decreases the likelihood*** of an operant response. When reinforcement is withdrawn for an operant response (i.e. not getting paid for work), **extinction** is usually the result.

An example of operant learning relevant to our discussion, is contraction of a muscle resulting in relief, safety, or a sense of control following an injury. The reinforcement is a negative reinforcement; the removal of pain, or fear, ***increases the likelihood*** of contraction. (This is *specific adaptation*, mentioned earlier.) This is an example of **active avoidance learning**. Unfortunately, however, although the original basis for the pain is long since gone (recovery from an injury), the learned operant (e.g., contraction) remains intact, and continues to be **emitted** based on reduction of fear. Central to our hypothesis is the understanding that, without a behavioral intervention, the behavior (learning) may remain intact regardless of any mechanical intervention that may be introduced. The altered postures conditioned

following trauma may remain intact. Learning to "not contract" for relief, pain reduction, fear reduction, or safety is **passive avoidance learning**.

Another important element of operant conditioning is the **discriminative stimulus** (written, $\mathbf{S^D}$), which sets the occasion (when/where) for an operant behavior, as follows: $\boldsymbol{S^D \rightarrow behavior \rightarrow reinforcement}$. An unusual example is operant learning by chicken embryos, just days old, that learned to control their environment by changing heart rate. Increasing heart rate (the operant) turned on a warming light (positive reinforcement), and decreasing heart rate turned it off (negative reinforcement). In the presence of a vibration ($S^D$), the same embryos learned to reverse the behaviors, where increasing heart rate (operant) turned the warming light off (negative reinforcement), and decreasing heart rate turned it back on (positive reinforcement). Posture and muscle activation patterns may be similarly conditioned. A standing posture, following a sprained ankle, becomes the $S^D$ for postural adaptation originally learned by the reduction of pain (negative reinforcement) that occurred when putting more weight on the healthy ankle, along with all attendant body-wide changes in muscular activation.

Behaviors, i.e., physiological activities, are regulated by very specific circumstances. **Learning is stimulus specific.** Conditioned responses are emitted (or triggered) by very specific stimuli, conditioned (CS) or discriminative ($S^D$), but as a result of **stimulus generalization** both classical and operant behaviors will also be elicited/emitted by similar but different stimuli. Stimulus generalization may not only occur due to external - perceived - stimuli, it may also become embedded in

seemingly unrelated complex patterns of coping behavior, such as maladaptive muscle patterns.

**Learning is also state specific.** Context changes the meaning of stimuli that regulate behavior, both conditioned stimuli (CS) as a result of classical conditioning, and discriminative stimuli ($S^D$) as a result of operant conditioning. As context changes CS and $S^D$ events yield diminished responses, different responses, or no responses at all. Changes in response strength and probability are a function of stimulus generalization. These contextual changes ***may be internal***, not just external. ***The internal contextual changes are referred to as "state" changes.*** What is learned in one state may not transfer to another state; stimulus generalization may not occur. When this happens we say that the learning is **state-dependent**. An extreme example is the drug addiction, e.g., alcoholism, where constellations of complex personal and social behaviors are biologically tied to drug and non-drug states, behavioral patterns that are elicited and emitted accordingly. Patterns of muscle activation as well may be state dependent.

**Vicious circle avoidance learning** involves the interaction of classical conditioning, operant conditioning, and cognitive learning. For example, fear may be classically conditioned to the thought, the intention, and initiation (CS) of using a constellation of muscle contractions in a "usual manner." The patient learns an alternative way of muscle responding, a new set of operants, which is reinforced by fear reduction (and avoidance of pain). The same stimuli that serve as a CS, e.g., intention, also now serve as an $S^D$ for the new operant muscle patterns; they trigger the motivation for the operant and provide for its reinforcement. When the injury is

healed, the newly reinforced muscle pattern may unwittingly remain, unidentified by practitioner and patient; the behavior has not been extinguished (unlearned) because it continues to be reinforced through fear reduction. Without changing what has been learned, the behavior is chronic and self-sustaining, potentially leading to compensatory responses in other muscle groups, potentially causing a new kind of pain and discomfort, a mystery perhaps to both practitioner and patient who may then mistakenly formulate misleading mechanical explanations.

The analyses to be presented are only several of many that could be formulated to account for the observed effects of CK interventions. They serve as an example. More information would be required to make a determination of the most likely factors involved. A behavioral analysis of **muscle weakness**, for example, might be as follows:

The instructions given to patients predispose them to "resisting," contracting the "relevant" muscles. Although in most cases, there is resistance, in some cases there is, relatively less resistance. In other words, in some cases there is a response (a contraction) to pressure (a stimulus) and in other cases there less (decreased response, decreased contraction). The failure of full response (contraction) may be the consequence of two potentially competing discriminative stimuli involved: (1) the implicit (or explicit) instructional set given to patients is an $S^D$ for "resisting," (i.e., contracting the "relevant" muscles), and (2) the applied pressure is an $S^D$ for muscle inhibition, a case of passive avoidance learning where "not responding" had been historically reinforced though pain or fear reduction. A second alternative is that the second $S^D$ (pressure) is emitting a more highly reinforced muscle response pattern

elsewhere in the body, one historically associated with successful avoidance of pain or discomfort, a case of active avoidance learning; thus, you would expect to see other muscles resisting, ones perhaps not under direct observation. And, a third alternative possibility may be that the first stimulus, the instructional $S^D$, is competing with a CR, a conditioned inhibitory muscle reflex response elicited by the CS (pressure applied), learned as a result of injury or trauma. In each case the target muscle shows up as "weak."

When the same **weak muscle is retested** a short time later, immediately after the introduction of an intervention, the same stimulus, $S^D$ or CS (pressure), now in fact does trigger the intended response, a contraction, i.e., resistance. What makes the difference? The avoidance responses, passive or active, rely on classically conditioned fear (the CR) for their motivation. The CK interventions may trigger external inhibition of the CR (fear) required for triggering the avoidance behaviors, fear that was classically conditioned to the same stimulus (pressure serving as both a CS and as an $S^D$) at a time of injury and trauma. Without fear the avoidance responses, "not contracting" (passive) or contracting other muscles elsewhere (active), are weakened, or disappear altogether, thus allowing the competing $S^D$ to emit its operant, contraction of the target muscle. In the case of classically conditioned muscle inhibition, the introduction of the intervention may result in disinhibition, i.e., disruption of the classically conditioned inhibition, as in the case of external inhibition which disrupts excitatory classical conditioning.

These behavioral analyses are supported by at least three considerations. (1) The types of interventions that will have this effect, while not irrelevant, are highly

variable; external inhibition and disinhibition require only an element of novelty, or distraction, or shift in attention. (2) The target muscle responses (contractions, resistance) occur immediately following the intervention, a condition prerequisite to external inhibition and disinhibition. And, (3) over time the CK treatments become more successful. This would be expected as a result of the extinction of classically conditioned responses, including emotional and inhibitory CRs, as a result of exposure to the CS without negative consequences. Without the CRs, passive and active avoidance responses, the operants, are extinguished as a result of the disappearance of both motivation (fear) and reinforcement (fear reduction).

Besides accounting for muscle weakness and the outcomes of implementing CK techniques for targeting, treating, and strengthening single weak muscles, the following CK outcomes can also be easily accounted for:

- *Muscles besides the target muscle behave differently as a result of targeting a single muscle.* Learned behavioral patterns constitute the organization and configuration of simpler responses, operantly reinforced behavioral patterns. The $S^D$ in question is likely to exert stimulus control over specific patterns of activity, e.g., a learned avoidance (passive or active) defensive pattern in response to fear, pain, and trauma that involved the recruitment and organization of specific muscles responses.
- *Significant changes in general body-wide muscle activity patterns may result from targeting single muscles.* Learned muscle behavioral patterns may comprise an overall physical posture, an observable posture that it tied together into a yet greater posture that includes other physiological systems,

e.g., emotion and the autonomic nervous system, as well as psychological experience by the patient. The CK intervention that targets a single muscle, and momentarily dismantles the passive avoidance habit, may disengage the defensive physical posture which in turn may have a significant impact on seemingly unrelated or distantly related symptoms, e.g., anxiety and memories.

- *Changes in muscle activity may be remote from both the target muscle site and the site of treatment, and appear unrelated.* In the absence of a systems perspective of physiology, and without an understanding of how learning is fundamental to physiological functioning, this outcome may seem mysterious. Based on the above considerations, this finding is self-evident.
- *Target muscle activity may be part of a larger behavioral pattern*, e.g., left knee shows weakness, while right knee is injured. Left knee and right knee muscle behaviors involve the same muscles on opposite sides. **Response generalization** is basic to biological learning. A reinforced response is a "category" of similar responses. Obviously the original response can never be identically reproduced. Other considerations have already been previously described.
- *Organized patterns of muscle behavior may disappear altogether and/or become organized differently.* This outcome is entirely consistent with the above analyses, e.g., the defensive pattern may "stop on dime," simply as a function of which discriminative stimulus is dominant, the one controlling the

contraction or the one regulating passive avoidance. Stimulus control is specific, powerful, and immediate.

- *Symptoms may disappear immediately or incrementally as more muscles are targeted across the same or subsequent treatment sessions.* Symptoms which disappear immediately may do so as a function of which discriminative stimulus is in play. The behaviors emitted by these stimuli may mediate different physiological outcomes, i.e., what the patient experiences. Symptoms may disappear incrementally overall in the patient's life as a function of extinguishing the classically emotional and inhibitory responses that motivate and reinforce the previously learned defensive responses that comprise a fundamental physical and emotional posture.
- *Positive outcomes are often reported even if the treatment is in an area apparently unrelated to the area of complaint.* Behavioral changes in muscles in one location are likely to lead to associated changes in unsuspected places in other locations as the overall postural pattern is altered.
- *Recalling stressful memories and painful emotions yield positive outcomes from the same CK techniques as do muscle dysfunction.* This finding is indeed consistent with the present behavioral analysis. Extinction of emotional responses and memory triggering, as a result of desensitization during exposure to conditioned (CS), results in extinction of passive and active avoidance learning behaviors, as well as classically conditioned inhibition. The originally learned adaptive behaviors, pre-trauma behaviors,

may then be reinstated and reinforced upon the extinction of these alternatively less adaptive behaviors.

# METHOD

Respecting the seventh and final observation, above, namely the ability to apply the same treatment to both psychological and muscular conditions, an investigation of possible connection between the methods and results of AK/CK based treatment and those of a number of treatments collectively known as Energy Psychology (EP) seemed to be in order. EP methods use non-informational sensory or motor stimulation to achieve psychological benefits. It was thus reasonable to hypothesize that similar mechanisms were at play in both contexts.

To examine this possibility, Weissfeld undertook an informal, uncontrolled study on 7 subjects. The study purpose was to determine if side-to-side eye movements, as used in Eye Movement Desensitization and Reprocessing (EMDR), the most well known of the EP therapies, would change the function of weak muscles in a similar manner to which to that of the AK/CK protocol treatment.

EMDR desensitization is the application side-to-side eye movements, in practice generally done simultaneously with focus on an emotionally charged stressor, for 20-50 seconds. The eye movements are the central therapeutic piece of the larger EMDR protocol that enjoys the widest professional acceptance and has the largest research base of any of a group of *physiological* stimulation methods used to treat *psychological* disorders. (Others generally falling under the umbrella title of Energy Psychology (EP) – not an exhaustive list here - EFT, Emotional Freedom Technique

and TFT, Thought Field Therapy both of which use tapping of acupuncture points, and the chiropractic-based NET, Neuro-Emotional Technique. Of these, EMDR enjoys the largest and most reputable research base, though other EP methods do have research17.) Rated as "probably efficacious in the treatment of civilian post-traumatic stress disorder" by the American Psychological Association, EMDR often has the effect of immediately reducing the intensity level of psychological pain. [18,19]

There were several similarities between using AK/CK protocol for psychological distress, and the various EP methods. The first is the need to activate the stressful feeling or memory (or weak muscle) with simultaneous or closely following application of sensory stimulation (treatment). The second is the finding of immediate change in symptoms following therapeutic intervention. I do not generally use side-to-side eye movement with my patients, nor do I regularly use any of the other named EP therapies, though I consider what I do to use similar principles. If side-to-side eye movements strengthened weak muscles, it would suggest that similar principles applied to both emotional and motor conditioning.

Weak muscles are defined for this study as muscles not able to lock isometrically, generally the equivalent of 3.5 or 4 on the 1-5 on muscle grading scale. The simple study was non-blinded, using random volunteers who had not self-selected as patients in the practice. Individual muscles around presently or occasionally symptomatic joints were tested to find those that were displaying isometric unlocking (inability to hold a stationary position against pressure) to a manual test.

After first determining the weak muscles around the area of complaint, weak muscles were tested in random order and followed up immediately with side-to-side

eye movement for about 20 seconds. Targeted muscles in several subjects were alternatively treated with acupuncture point tapping as done in the EP procedure Emotional Freedom Technique (EFT) [20, 21].

## RESULTS

In every case, without exception, 20 seconds of side-to-side eye movement or EFT point tapping had the effect of strengthening the inhibited muscle that had just been tested.

One of the subjects, SM, a 40-year-old female school counselor in otherwise good health, reported that she had successful right medial collateral and ACL repair 20 years earlier, after a skiing accident. She reported being released from physical therapy when the knee was at 90% compared to the opposite knee. Some pain remained in the knee in certain movements but not enough to significantly interfere with daily living. SM had 5 muscles in the ipsilateral knee and ankle (usually tested and corrected as well when working with knee problems) that were inhibited, and eye movements immediately restored locking to all.

A 1-month follow-up on SM showed that all muscles remained intact to isometric tests, and pain was not present except when she did a particular twisting movement that she only did when dancing. She did report however, unprompted, that she still couldn't shake the image of her knee twisted at an impossible angle as it was after the accident (a common clinical finding when muscles are routinely tested). When she thought of that image, several of the previously tested muscles that had strengthened were again inhibited. After doing the eye movements while she held the image in

mind, the visualization of her mangled knee was difficult to bring to mind at all (again, a common clinical finding, also common to EMDR). All the muscles that had been weakened by the visualization were now locking appropriately, and re-imaging the visualization did not change their facilitation. The pain in the twisting movement was immediately gone as well.

## DISCUSSION

Putting aside the obvious limitations of design and size, the study, the results, save for the unanimity of the responses were not surprising, from Weissfeld's experience over thousands of muscles refacilitated via other methods.

The various EP therapies have all found that mild, non-specific therapeutic inputs to the nervous system have the ability to create immediate specific changes when the inputs are performed immediately subsequent or simultaneously with activation of a pathological response that is being targeted. Eye movement (EMDR) or tapping points (EFT, TFT) or spinal stimulation or manipulation (NET) don't have the desired effect unless their therapeutic stimulation comes simultaneous or immediately subsequent to conscious focus on the emotion, thought or memory that they are attempting to address. Research (addressed later) has strongly indicated that memories are subject to erasure when memory formation is blocked following their activation.

Given these findings, what can account for the changes in muscle facilitation following eye movements? We conclude that change in muscle behavior could only be accounted for by a change in learning, either by creating new learning or altering

what is already in memory. In the brain there are at least two different types of learning that may be differentiated, each with different sites of operation. The effect could be located in any, all or several of the following areas:

Is the effect on procedural memory, the memory of how muscles organize to create patterns of movement? Are we altering memories of motor patterning? This would put the effect in the basal ganglia, cerebellum and motor cortex, brainstem or extrapyramidal areas that control primitive postural reflexes.

Given the similar results on emotion with eye movements, is the change in the emotional learning – how the muscle is perceived via the limbic system or other area? This would likely put the effect in the amygdala or limbic system.

### Muscle and posture strategies and prediction

Procedural memory, sometimes known as instrumental memory, is the name for memory of muscular activation routines that are the templates of behavior, the complex patterns of movement or manipulation of objects that may be called upon to respond to needs of the environment, and adapted as needed in real-time, as in an experienced driver swerving around a pedestrian who stepped out in front of his car. Procedural memory is implicit or unconscious. Compared to emotional conditioning, the exact mechanisms of procedural memory are poorly understood, [22] though it is known that they are enacted mostly through interactions between the basal ganglia, cerebellum and motor cortex, brainstem, and extrapyramidal areas that control primitive postural reflexes.[23] The cerebellum also is critically involved in motor learning. [24]

Two concepts need to be elucidated to delve into the findings: *muscle synergies* and *movement prediction*, both of which rely on implicit memory for their success.

Muscle synergies are pre-determined routines of coordinated muscle activity that are building blocks of movement. They are task-dependent groupings of muscle activations that are combined and reused to generate a variety of behaviors. [25] There are a limited number of possible motor synergies, [26] much fewer than if each muscle had to be separately controlled through each movement. [27,28] The repertoire of movements also includes alternative synergies, allowing adaptation or muscle substitution. [29,30,31,32]

Muscle synergies require a coordinator, or organizer. [33] The "schema theory" [34] holds that schemas are not sensory or motor but rather *memorized* relationships – links between several sensory or motor components of action. Posited as existing in the parietal cortex, which adds the relationship of the body to maps of the outside world, the schema may associate different representations of the body in various areas.[35] It includes a repertoire of all possible actions built on past actions. A gymnast, for instance, would have a much larger repertoire of how to get around an obstacle than the average pedestrian. Out of this repertoire, a motor strategy is automatically defined that groups together various synergies to plan a motor event. Such planning, according to principles of behavioral physiology, will be inextricably connected with the context in which it was originally learned. [36]

In order to have success in movement, the brain must be able to predict the outcome of the movement. [37] Prediction is based on learning from past consequences, including sensory consequences and outcome consequences. [38] In

fact, before any movement takes place, the brain runs a feedforward simulation of the movement, with all the attendant neurological activation (including both facilitation and inhibition) of motor and sensory pathways that remain inhibited to actual movement. [39] Posture too is predictive, with alterations of posture undertaken before the anticipated movement. Contrary to expectation, posture is not maintained by error detection and correction. [40] Posture is subject to learning, as well. [41,42]

Prediction is an extremely important concept, because it translates past experience – learning - into present muscle facilitation - behavior. We will use a term *predictive equation* to refer to the central integration of many the factors which altering execution of motor patterns. Some of them are:

- feedforward of somatosensory information regarding the current internal state – visual, proprioceptive, vestibular, cutaneous and nocioceptive;[43]
- feedforward sensory information about the immediate environment to be interacted with, [44]
- learned expectations of the qualities of the environment (is the ball you are about to lift foam or lead?), [45]
- learned expectations from previous results with similar movements with regard to postural and muscular synergies applied successfully or not, and emotional characteristics, [46] or the context of that movement (Did previous use of movement cause pain, or happen when pain or pleasure happened to be present? Where did it happen and when in your life?), and

- supraspinal calibration of stretch and other somatosensory receptors and reflexes, presynaptically modulating spinal reflexes, which may be based on some or all of the above factors of the predictive equation, and in turn altering the equation. [47]

These factors all interact to yield the choice of a particular movement strategy over others, [48,49] and, the slower the movements, the more they are subject to realtime alteration by sensory feedback at central or spinal reflex levels.

### The role of the cerebellum and basal ganglia

The cerebellum, entirely subconscious, is a storage area for conditioned motor learning.[50] It participates in planning and modification of motor activities though comparison of the intended movement with the outcome movement, pre-movement postural planning, timing and regulation of muscle tone. [51, 52] It plays an important coordinating or organizing roll with its homunculi organized functionally rather than by body part. In the cat, for instance, the neurons controlling whiskers, mouth and paws, all used for cleaning, are grouped together. A particular area may thus be represented in several functional organizations within the cerebellum. [53] In addition to its role in motor activity, the cerebellum is also a part of an integrated network regulating emotional behavior [54] and participates in fear learning. [55] It shows increased blood flow in individuals with PTSD.[56]

The basal ganglia, second among suspects for conditioned learning that may be central to muscle dysfunction, are important in procedural memory and control of movement, and are well connected to the amygdala.[57] Because of those basal

ganglia-amygdala connections, the amygdala may be a key trigger of post-traumatic aspects of muscular function.

Coordinating activity among cortical areas, and possibly responsible for 'chunking' representations of motor and cognitive action sequences so that they can be implemented as performance units, [58] the basal ganglia respond to signals from the limbic system related to successful completion of movement by promoting memory storage of the successful combination. [59] The basal ganglia may, along with the motor cortex, [60] be the implementer of action sequences initiated by the amygdala and/or the cerebellum. Disruption of established memories in any of those areas could disrupt resulting behavior, but evidence points to combined action of the cerebellum (motor conditioning) and amygdala (emotional conditioning) in evocation of motor behavior. [61,62] As we will see, memory traces in both areas are similarly affected by the same neurochemical factors.

### PTSD conditioning, musculoskeletal dysfunction and the amygdala

The amygdala (particularly the basolateral nucleus of the amygdala) is a major memory center for conditioned behaviors, and it is particularly conditioned by pain and trauma. It is overactive in PTSD, while the activity of the hippocampus, and the activity of the conscious, rational prefrontal cortex are diminished. [63,64,65] The hippocampus holds non-emotional, declarative and spatial memory, additionally storing memory of the context of pain and trauma, ($S^D$, in behavioral terms). Activation of the amygdala increases memory consolidation in other areas of the

brain, [66,67] meaning that what occurs under stress or arousal will be more likely to be learned.

Central to our thesis, we propose that a more limited version of PTSD, a *musculoskeletal PTSD*, (mPTSD) which is a subset of PTSD but may also be present distinct from the psychological state of PTSD, may exist. It may be triggered or held in the cerebellum and/or triggered by amygda activation of defensive patterns in the basal ganglia, motor cortex or cerebellum. In mPTSD, we hypothesize, adaptive motor reactions to various stressful or traumatic events are learned and then repeated, either consistently or in response to particular circumstances. This smaller-scale variant of PTSD proceeds from traumas of less impact, like a sprain/strain injury, [68] but would also be a feature of classical PTSD. The *definition of mPTSD* then, is a learned adaptive condition of altered muscle facilitation that follows stress or trauma. It is comprised of *s*pecific muscle and postural strategies composed of altered muscle facilitation based on the specific adaptations to specific stress or trauma.

Psychological, vestibular and autonomic aspects of the nervous system will be involved in mPTSD, [69] as activity of each is necessarily a part of muscle activation and posture. Once the stressful or traumatic incident is completed, or tissues have healed, the raison d'etre for the adaptation is no longer present, yet its results remain in memory, thus becoming maladaptive, creating PTSD or mPTSD.

SM, the study participant with the repaired ACL in fact reported the persistent image of her knee in its injured state, a symptom of PTSD, and the resolution of that was part of normalizing the function of the knee. So in that case, what appeared to be just a functional problem revealed hidden psychological aspects, not an unusual

finding in clinical experience, but also not necessarily crucial to the resolution of MSD by the methods we are proposing.

Psychological, vestibular and autonomic aspects of the nervous system will be involved in mPTSD, as activity of each is necessarily a part of muscle activation and posture. [70] (The immune system too may be affected.) [71] (This may be one explanation of the clinical improvements in other systems sometimes noted when we are treating musculoskeletal complaints. Generalization of benefits is often noted or from spinal manipulation, and vestibular training can improve musculoskeletal complaints.) [72]

### Conditioning of musculoskeletal PTSD

As stated earlier, several storage areas for conditioning in the brain are thought to the amygdala and the cerebellum. PTSD is a conditioned reaction whose triggers are largely located in the amygdala, [73] which, when aroused, triggers reactions throughout the neuraxis including motor reactions. mPTSD, to the extent that it exists, may be built on the same circuits. The amygdala may be the focal point for maintenance of the conditioned reactions of mPTSD, and likely one area therapeutically impacted by EP approaches such as EMDR. The integrative functions of the basal ganglia might respond to cues from the amygdala and incorporate them into the predictive equation. The cerebellum, on the other hand may be the more important area for storage for the conditioned motor reactions, or it may yield its contribution in response to or in combination with the amygdala.

Phenomenologically, at least five factors may relate to the onset, conditioning and maintenance of mPTSD which, interacting among each other, may be instrumental in the development of learning of new motor strategies or set up triggers (CS) for activation of existing alternate motor strategies (CR). Each will add to the predictive equation, with all the attendant activations of motor, proprioceptive and sensory pathways. The potential conditioning experiences and maintenance mechanisms for mPTSD are:

1 Uncompleted trauma-reaction engrams [74,75,76,77]

1.1 *Somatic experiencing* is a theory and treatment approach introduced and elaborated by Peter Levine Ph.D., which views trauma memories as uncompleted trauma reactions. The theory states that the bodies of traumatized individuals portray "snapshots" of their unsuccessful and uncompleted attempts to defend themselves in the face of threat and injury. Trauma response that is coded in the brain as unsuccessful engenders an incomplete survival response to the threat. This incomplete response unconsciously activates or moves towards threshold muscle activation behaviors that are triggered continually or periodically to complete their defensive or offensive imperative.

2 Post-traumatic repetition-learned muscle synergy engrams [78, 79,80,81,82,83,84,85,86]

2.1 Following an injury, tissue damage and resulting pain create the need for altered muscle function to protect the injured tissues and avoid pain. The antalgic postures require either creation of new muscle synergies or muscle

and postural strategies, or re-organization of existing synergies or strategies. This may sacrifice efficiency and cause asymmetry of forces acting on joints, affecting movement and posture. These ongoing imbalances can adversely affect joint function, possibly encouraging arthrosis of involved joints, leading to further alteration of posture. These synergies are then repeated - essentially practiced – and perfected, and learning not to perform such behaviors is notoriously difficult, particularly when the patterns are outside of conscious awareness after the pain is gone.

3 The tendency to over-react to trauma [87,88,89,90]

3.1 When we treat a strain/sprain injury or an 'overused' body part with elevation, ice and compression, we are giving a nod to the concept that the body has reacted to stress or trauma by initiating an overwrought inflammatory reaction. Increases in norepinephrine and cortisol that are initiated by pain and stress cause overconsolidation – formation of stronger memory engrams. Anecdotally, 'walking off' a minor lower extremity injury seems to be effective. Does this remind the brain that the body is still functioning acceptably, possibly short-circuiting the tendency towards over-reaction?

4 Psychological posture engrams [91,92,93,94,95]

4.1 Psychological states and traits affect posture, and thus affect muscle tension and balance, and presumably add to the predictive equation.

5 Compensations to all of the above [96,97,98]

5.1 Compensation represents the ongoing attempts to maximize function given the conditions that are in place. 'Successful' compensation allows the body to function well enough – though it will necessarily be less efficient and more stressful than the pre-adaptation/compensation state. Successful in this case means that given all conditions, the body is doing the best that it can, even if that includes operating in pain.

5.2 Muscular compensation is well known to practitioners of various forms of applied kinesiology. Because of the importance of these phenomena, we offer several examples.

5.2.a It is not uncommon for patients to present with weak muscles on the opposite side from their complaints. This is known as *switching* in Applied Kinesiology. Briefly rubbing the 'kidney 27' acupuncture points bilaterally will predictably correct the situation, often with the interesting result of the exact same pattern of muscles weakness now transferred to the side of the complaint, where it 'belongs'.

5.2.b The reverse myotatic reflex (stretch reflex), is a spinal reflex that inhibits the activity of a muscle that is overstretched. It is possible to elicit this reflex manually, by stretching the golgi tendon organs or muscle spindles. If a previously intact muscle is tested immediately after such manual stretch, it should be inhibited (weak). The absence of this reflex is a compensation response, wherein the muscle has been essentially locked into strength, by definition a proprioceptive dysfunction. [99] Correction of this compensation will frequently

cause the involved muscle to test as weak, revealing its truer functional compromise. (Correction of this compensation could be a preventative factor in muscle tears or strains, both traumatic and repetitive, because the muscle continues to contract despite the forces that should evoke the self protective relaxation and lengthening.)

5.2.c Correction of compensation runs the risk of temporarily increasing symptoms that had been masked by the compensation.

5.2.d It is possible that compensation may be part of the mechanism that locks adaptation into a more permanent state by stabilizing the dysfunction.

**Memory erasure following activation**

Damasio (1999) coined the term 'body loops', [100] to describe states of somatic activation induced by central emotional activation that feedback to somatosensory receptors. This essentially means that the awareness of an emotion comes as a result of awareness of sensations in the body.

Scaer, (2001) postulated a related process that includes memory: "Myofascial pain probably represents procedural memory for the specific defensive motor stretch reflex and its proprioceptive template precipitated by the movement of the body in the MVA [motor vehicle accident], thereafter elicited by stress or any movement pattern reminiscent of the accident, in the form of bracing and muscle spasm." [101] PTSD and mPTSD are both conditioned memory problems wherein the memories themselves, not the mechanics of memory are the problem. They are problems not of

hardware, rather of the data that creates the functional software which formats behavior.

Until recently, memory, including conditioned memory, was thought to be indelible. (This supposition forms the basis of classical behavioral analysis, which does not look at the underlying neuromechanics of behavior.) The neurological understanding was that the expression of behavior could either be covered up by new learning or separated from the trigger that elicited the behavioral response to that learning, but that the learning it self still remained. First elucidated by Pavlov, [102] the term *extinction* in behavioral science means the reduction of a behavior that does not receive reinforcement. Extinction occurs when the expected result does not follow the conditioned stimulus it was tied to. As an example, mice are conditioned to associate a footshock with a tone. When the tone appears without the following footshock, fear behavior eventually disappears.

The understanding of extinction changes when it is applied to the behind-the-scenes neurophysiology. There, extinction is seen to be not an end to the conditioned memory engram at all, rather it is the formation of a new memory that covers up, but does not abolish, the old memory. [103, 104] Extinction is may be caused in part by new learning in the prefrontal cortex [105]. It is context specific (a discriminative stimulus, $S^D$, in behavioral terms) with the learned contextual reference stored in the hippocampus, so changing the setting may change the learning. [106] The *neurophysiology* of extinction then, is an *additive* process requiring new learning.

If we look at neurophysiological mechanism of extinction, the addition of new learning, it is unlikely that eye movements, which are information neutral and do not

contain meaning relevant to a specific muscle, would be capable of adding new information that would cause extinction. But what other mechanism could account for this?

Around the year 2000 a new theory about the permanence of memory was beginning to seep into the literature, shaking established assumptions. The preponderance of evidence showed that once activated, memories, (fear inducing memories in the amygdala were among the first studied), became unfixed from storage and subject to being lost if they were not *reconsolidated*. Where we previously thought of memories like files on our computers where the saved version of them remain even if our computer crashed, memory now could be seen more like a file from a filing cabinet, which, if blown away by the wind, was gone for good. This was even found to affect well-established memories. [107]

If a conditioned memory is activated (CS), as in the conditioned mouse expecting a foot shock when hearing a tone, it becomes labile, and needs to be stored again - reconsolidated - or it ceases to exist. If a substance that prevents the formation of new memories is injected into the amygdala and the conditioned tone, is played, the mouse will show fear behavior as expected but in subsequent trials it will not show fear on hearing the tone.

Could methods that rely on sensory input (EMDR and the others) to treat affect disorders have their effect by preventing reconsolidation? Inhibition of reconsolidation is already considered a potential treatment for PTSD via pharmacological approaches. [108] This would likely put their action in the amygdala, the home of fear conditioning, though it is worth noting that EP procedures including

EMDR claim to work as well across a spectrum of painful affective states [109,110] and even for physical pain [111]. EMDR treatment of grief [112] for instance, which seems to have a very different spectrum of brain activity than fear, [113] indicates that if inhibition of reconsolidation is a factor in its effectiveness, the amygdala is only one of the possible places of effect.

It has been shown with fear memory in the amygdala at least, that activation (retrieval of a specific memory) produces a synaptic potentiation in the lateral amygdala that is selective to the reactivated memory and that disruption of reconsolidation is correlated with a reduction of that synaptic potentiation. Thus, both retrieval and reconsolidation alter memories via synaptic plasticity at selectively targeted synapses, [114] paralleling the way a specific muscle can be targeted for disruption of its maladaptive function.

Areas of conditioned motor learning may be the areas of storage for mPTSD conditioning. Motor conditioning involves the ipsilateral cerebellar nuclei, bilateral cerebellar cortex, anterior vermis, contralateral pontine tegmentum, ipsilateral hippocampal formation, and bilateral striatum (basal ganglia). [115,116] Of these, the cerebellum is the best suspect for mPTSD conditioning, as it strongly adds to the predictive equation (see above) and is also involved in fear learning and PTSD. [117,118] It could well work jointly with the amygdala in formation of PTSD and mPTSD. Cerebellar motor conditioning has shown to be subject to both inhibition of reconsolidation upon activation, and to extinction. [119]

A model of non-reconsolidation of conditioned motor (or for that matter, emotional) learning upon successful therapy may not mean that the learned movement

(or defensive emotional activation) strategies themselves are erased. There are two possible explanations for our observations.

1. The learning that mapped the somatic behavior may have been erased by the blockage of reconsolidation, meaning that the memory of *how* to adapt in that specific way is eliminated.

2. The memory of *how* to accomplish the action may still be present, but the *trigger* (CR) which evokes the behavior may have been disrupted.

Behavioral observation cannot differentiate between the two, but both are different from extinction. Treatments to the body that add sensory input, particularly those that include sensory or motor input to the local area of muscle dysfunction - massage, exercise, practice of certain movements, stretching, etc – may add location-specific information that can alter the old pattern through extinction. This new information, if effective in changing facilitation patterns, may do so because of specific meaning to the system, encouraging specific new, hopefully more successful compensation on top of preexisting adaptation (extinction). The old pattern is altered either by encouraging novel compensations that may involve the whole postural system, or by encouraging intelligent alteration of the current prevailing facilitation patterns based on unknown, and, in the vagaries of clinical practice, unknowable specific effects of such inputs. It may also be said that nothing occurs without meaning. Eye movements or any input will have some unavoidable amount of meaning, based on the individual's learning from past experiences using side-to-side eye movements or even the subject's reaction to being instructed to do a strange thing.

So, by what mechanisms might reconsolidation be inhibited by various therapies?

**The locus ceruleus – fearmonger?**

Norepinephrine (NE), produced by the locus ceruleus (LC) may turn out to be the major enforcer reconsolidation acting throughout the brain. Activation of the LC enhances consolidation in the amygdala, [120,121] and it turns out, the cerebellum as well. [122,123] The LC receives pain, proprioceptive, visual, auditory and vestibular input, both filtered and unfiltered by the thalamus.[124] Physical or emotional trauma both stimulate the LC, increasing its output of NE which in turn can actually cause memory *overconsolidation*. [125] Activation of the amygdala in turn activates the LC, creating a positive feedback loop. [126] Activation of the LC elicits postural asymmetry and movement changes. [127] The LC affects posture via projections to the spinal chord and is itself affected by changes in the spinal chord from postural changes. The cervical joint proprioceptors, activated in velocity changes and perturbation of balance, have connections into the LC. [128] The resulting increase in NE increases memory consolidation and reconsolidation and its elevation has been associated with PTSD. [129,130] Beta-blockers like propranolol (which block noradrenergic receptors for NE) are considered a potential treatment for PTSD.[131]

In fact NE seems to play a part in consolidation and maintenance of conditioned learning in general, in many areas of the brain, [132] so anything that inhibits the LC will tend to reduce consolidation and reconsolidation of conditioned memory. This may be used to clinical beneficial feature when dealing with physically and affectively painful circumstances, which by their nature increase NE. A partial list of endogenous LC/NE inhibitors include opioids [133,134,135] and GABA, [136,137]

both of which may also act to affect extinction and reconsolidation directly in the amygdala.[138,139,140]

The cerebellum, the likely area for mPTSD conditioning, if not emotional conditioning, is similarly affected by NE [141] and GABA, [142] directly and thus would also be modulated by anything that modulates the LC. Thus, we see a mechanism by which the same therapeutic modalities can treat both affective and motor dysfunction.

The LC has a close relationship to spinal proprioceptors, which, by way of the thalamus, cortical sensorimotor integration areas,[143] and cerebellar vestibular afferents, trigger LC production of NE to increase general arousal in response to the orienting reflex of head, neck and ocular tracking system scanning for biologically imperitive threat, feeding or reproductive opportunities. [144] The spinal proprioceptors themselves are affected by reflex arcs that summate somatosensory afferents in the dorsal horn of the spinal column, [145,146] and also affect the LC via peripheral sensation and pain [147,148] via various pathways. These effects may serve to alter posture and spinal muscle tone and alignment, [149] and, in a positive feedback loop may further increase LC activity.

### Energy psychology and norepinephrine

Human experience of the seamless nature of memory belies fragility built into the system. That a brief and seemingly trivial sensory input can disrupt and rapidly alter long held memory patterns, immediately changing physical or emotional pain into mild discomfort defies the logic of day-to-day experience. In the absence of

appropriate therapy or corrective experience, the elicitation of the memory of trauma will increase NE, [150] and reconsolidation seems to reinstate a similar enough version of the conditioned reaction that change if it occurs, is only noticed after the passage of a good deal of time.

The various EP therapies may each contribute differently to a transitory or ongoing downregulation or blockade of NE. Each works with the same general procedure which includes: 1) determination of present state of patient, and use of clinical judgment as to what the patient's immediate priority is 2) elicitation and focus of attention on a specific feeling, emotion, image, thought or memory, 3) application of therapeutic modality, simultaneously or shortly following elicitation, and 4) recalibration of the affective state to determine what change has occurred. The effectiveness of EP therapies is likely more dependant on the clinical judgment and skill of the practitioner in choosing and eliciting the most central and important focus than on the modality of treatment, although it has been found that, in chemical blockade of protein synthesis required for reconsolidation at least, stronger blockade results in more robust disruption of conditioning. [151] The same may be true in establishing the somatic focus when treating MSD. Following is a discussion of how each therapy may effect downregulation of NE. (Other theoretical explanations for EP therapies do exist, but will not be entertained here.)

**EMDR -**

- Rapid eye movement sleep-like eye movements may cause a suppression of NE in the LC as occurs in sleep. [152] Horizontal eye movement is linked to nucleus

prepositus hypoglosis which has inhibitory GABA efferents to the LC, normally activated by the eye movements of sleep. [153]

- EMDR has been shown to improve galvanic skin response [154] which is associated with a decrease in norepinephrine [155]
- PTSD has been found to create right and left hemispheric differences in brain function, with hemispheric imbalance. [156] Additionally the interhemispheric integrator, the corpus callosum, is smaller in PTSD. [157] Interhemispheric communication supports the cortical integration of traumatic memories into general semantic networks. This integration can then lead to a reduction in the strength of hippocampally mediated episodic memories of the traumatic event as well as the memories' associated, amygdala-dependent, negative affect. [158] The side-to-side eye movements thus may activate a whole brain processing of the focused-on piece of trauma that had not been possible before. Speculatively, such successful processing of traumatic activation may decrease arousal, thus reducing NE facilitated reconsolidation.

Because the same results are seen with alternating tapping or audio tones, [159] alternating right and left hemisphere activation may be a more likely mechanism than one that depends on the particular neurological aspects of eye movement.

**Acupuncture and acupuncture point tapping.**

Thought Field Therapy (TFT), originated by Roger Callahan Ph.D. in the late 1980's manually taps condition-specific patterns of acupuncture points simultaneously with activation of the stressful target to get its results. An offshoot of TFT, Emotional

Freedom Technique (EFT) which employs group of points that overlaps those used in TFT, is not condition specific with regards to point selection and requires points to be tapped in no particular order. There is some debate over the need for the more complex algorithms of TFT, [160] but all acupuncture point tapping is done for a much shorter time than would be the case with needles, and is similar from patient to patient. One study, however, actually showed better efficacy for tapping over acupuncture. [161] For the purposes of this exploration, due to the lack of neurophysiological research on point tapping, we are necessarily limited to using the much larger literature regarding neurophysiological effects of acupuncture, understanding the speculative nature of applying them to TFT and EFT.

A PubMed search on acupuncture and norepinephrine interestingly yielded conflicting evidence about the effect of acupuncture on NE, with some reports of increased NE, some decreased and some unchanged. Analysis suggests, however, that this is far from surprising. Acupuncture chooses from an assortment of hundreds of points, may include auricular (ear) points (not technically considered acupuncture points) and practitioners may treat the same symptom with different points based on a multifactoral analysis of the individual patient. Acupuncture points are considered to be either tonifying or sedating, depending on point selection and technique. If that maps to brain arousal, which is connected to NE levels, various points and techniques may modulate NE differently. But NE increase has other effects, including inhibition of pain and sensation in the dorsal horn of the spinal chord. [162, 163, 164] So points used for analgesia may actually be increasing NE, while points chosen for emotion, or stress as in TFT/EFT may be decreasing it.

Stimulation of opioid production or reception, both of which can downregulate amygdala or LC is often considered a major component of acupuncture. [165,166]

Thus it would seem that inhibition of NE could be achieved with the proper acupuncture technique, also allowing the possibility of achieving the goal of alteration of conditioned motor or emotional reaction.

### Spinal Manipulation

The orientating reflexes provide a model for the effect of head and neck movement on arousal. Elicitation of the orienting reflexes, in which head and eyes fixate and track on a threat or reward, stimulate the LC, [167] likely via both direct spinal afferents [168] or though vestibular [169] or amygdalar [170] connections. The LC in turn increases arousal via NE projections throughout the brain. The vestibular system, activated by neck and head movement, is also closely connected to the LC. The high level of comorbidity of anxiety and balance problems is based in this connection. [171]

The connection between the spine, particularly cervical proprioceptors and LC, both directly and via the vestibular nuclei, provides a compelling line of investigation for the beneficial effects of spinal manipulation (SM). Subluxated or fixated joints anywhere in the spine necessarily have altered muscle facilitation which will affect somatosensory afferents, muscle strategies. Because of the connection to the vestibular system, we would thus predict that SM would reduce anxiety. Several studies have in fact borne this out. [172,173]

SM (particularly of upper cervical segments) that restores alignment may normalize proprioceptive afferents reducing input to the LC and decreasing NE. When this is applied to specific aspects of pathological conditioning, as is done in the chiropractic-derived EP method of emotional treatment, Neuro-Emotional Technique (NET), the associated downregulation of NE would then prevent reconsolidation of the conditioned emotional reaction. The ongoing relief of spinal-mediated LC activation following successful reduction of spinal joint tension could also have longer lasting effect on general arousal. SM has also been found to increase serum beta-endorphin levels, [174] presenting yet another potential mechanism of action.

### The neurontogenic effect

To quote the poetic neurobehavioral researcher Alain Berthoz, "there is no perception of space or movement, no vertigo or loss of balance, no caress given or received, no sound heard or unheard, no gesture of capture or grasping that is not accompanied by emotion or induced by it." [175] Put into clinical terms, elimination of the affective component of the conditioned reaction seems to result in the disturbance of reproduction of its motor component (behavior). [176] The blockade of reconsolidation is a mechanism for the elimination of at least the emotional component of post traumatic compensatory or adaptive muscle activation patterns.

*When post-trauma conditioned adaptative motor learning is eliminated, what remains?*

The answer to that question seems to be that latent correct motor function, [177] minus its adaptive component is reestablished. This latent correct function necessarily

includes more than just the area that was experienced as malfunctioning. It has been observed by Weissfeld to create a trend of improvement of altered muscle activation throughout the body. The post treatment correction generally includes symptom reduction as well.

A term is needed to describe the process of restoration of latent pre-trauma function. We call it the *neurontogenic effect, or neurontogenesis*, derived from a combination of the terms *neuro*- relating to the nervous system, and ontogenesis - relating to the fundamental nature of the thing. A therapy is said to have a neurontogenic effect when it putatively restores the pre-trauma function of the nervous system to preeminence by disruption of reconsolidation. Forms of treatment that utilize this principle may be known as *neurontopathic medicine* or *neurontopathic treatment.* Such treatment need not include physical treatment at all, because, we hypothesize, it is addressing centrally held memory engrams.

Pertaining to the musculoskeletal system, restoration is observed by noting the normalization of muscle activation on a local or body-wide level, and concurrent with symptom resolution. Neurontogenesis, then, is a descriptor of the removal of conditioning by prevention of reconsolidation that rejuvenates function (behavior) towards its latent, pre-conditioning, state.

# SUMMARY AND CONCLUSIONS

Stress or trauma causes specific adaptations which become maladaptive when learned and repeated beyond the time of their need. The lability of such conditioned learning following activation makes them subject to erasure if reconsolidation is

blocked. This has been fairly well documented in the amygdala, but also observed in the cerebellum, putative home of conditioned somatic learning. Once consolidated, the elicitation of a memory will necessarily call forth the memory complex engaged at the time the memory was made. This memory complex may include data from sensory, motor, or psychological modalities. Elicitation of a cognitive, somatic or emotional memory may trigger, consciously or not, other modalities of the memory, bringing specific post-traumatic conditioning a labile state.

Norepinephrine (NE), produced by the locus ceruleus (LC) in response to stress or activated memory of stress, increases consolidation and reconsolidation of conditioned memories. When the memory of trauma (or one of its corollary modalities) is elicited and NE is inhibited via therapeutic input or agents, the reconsolidation of the elicited modality is prevented. This may occur because of suppression of NE production, which would normally seamlessly reconsolidate the traumatic memory and its modalities.

Musculoskeletal-PTSD (mPTSD) is stress/trauma or post-stress/trauma somatic adaptive learning, specifically altering muscle patterns, which has become maladaptive. The cerebellum may be the main locus of conditioned motor memories of the trauma and post-trauma adaptation. The conditioning that may cause mPTSD and be imprinted to memory arises from:

1 body maps of whatever uncompleted state the musculoskeletal system was eliciting in trying to respond to trauma,

2 post-traumatic repetition-learned adaptational muscle and posture strategies to deal with pain or other dysfunction,

3 any historical trauma-encoded patterns on-board at the time of stress or trauma,

4 psychological postures and the memory complexes that accompany them

5 further compensations to all of the above

These conditioned motor patterns utilize existing and possibly new muscle synergies, developed into muscle and posture strategies that take into account the sum total of all of the above to create compensation strategies. These are necessarily both contextual – associated with specific behaviors, thoughts, emotions, postures; and layered- clinically seen as patterns of muscle facilitation and inhibition that may change radically when addressed appropriately.

Clinical treatment of mPTSD which utilizes the neurontogenic effect of restoration of latent, more correct function, necessarily addresses function from the top layer (the observable set of muscle behaviors) inwards, by the same 4 steps that apply to psychological treatment in energy psychology modalities: 1) determination of present state of patient, in this case by testing of muscles both in the areas of complaint and elsewhere, and use of clinical judgment to determine the present priority 2) elicitation and focus of attention on the specific muscle determined to be a priority, by testing, stretching or otherwise using that muscle or assuming a posture that places stress on that muscle, 3) application of therapeutic modality, simultaneously or shortly following elicitation, and 4) recalibration of the muscle pattern.

According to this model, treatment that achieves the neurontogenic effect may be anything that decreases NE (arousal) when performed during or immediately following the elicitation of a pathological behavior. Successful neurontogenesis is

often immediately discernable by noting a change in function or symptoms, generally, though not necessarily, towards what would be considered a clinical improvement. An apparent worsening of the condition is also possible, caused by the elimination of a compensatory response. This aggravation will be transitory, and often eliminated by elicitation treatment of the next clinical priority.

### Clinical implications of a neurontogenic approach

The prevailing and generally unquestioned musculoskeletal treatment model is an additive model, the aim of which is to restore function by retraining the body to exhibit correct function, encouraging more successful compensation to maladaptive function. This parallels allopathic medicine, defined by Merriam-Webster's Online Dictionary, (2007) as "relating to or being a system of medicine that aims to combat disease by using remedies (as drugs or surgery) which produce effects that are different from or incompatible with those of the disease being treated." [178] In terms of conditioning, the allopathic model is an extinction model, wherein the pathogenic conditioning is not eliminated, but altered by the addition of new learning. A neurontopathic model is based on elimination of specific maladaptive conditioning behaviors by disrupting their reconsolidation.

A neurontopathic medicine model posits that functional healing (resolution of pathological function) may occur from the reinstatement of latent correct function. The subtraction or erasure of this adaptive learning can immediately restore function without repetitive retraining. Retraining is the physical medicine version of a drug administered to alter function.

While virtually unaddressed in musculoskeletal therapies, conditioning is well understood, if not directly addressed in psychotherapies. With the exception of energy psychotherapies, the new understanding of disruption of reconsolidation has not yet impacted mainstream practice, and most treatment, however sophisticated, is still based on an allopathic model. Cognitive-behavioral therapy for instance, based on the idea that thoughts cause feelings and behaviors, [179] *adds* new thoughts or learning which will essentially create new, better adapted conditioning. Even psychodynamic therapy, also known as insight-oriented therapy, while seeking deeper roots of an issue, has as a goal increasing understanding (an additive process) of the influence of the past on present behavior that may fail to disrupt the behavioral conditioning itself.[180]

Organs and endocrine function are also subject to conditioning (recall Pavlov's dogs, trained to salivate at the sound of a bell) [181] and thus likely subject to disruption of reconsolidation and resultant normalization of function. We know of no reliable way to monitor such activation for research purposes. Given the role conditioning likely plays in internal function, this area is ripe for research.

Maladaptive conditioning is therefore a factor in most conditions and to the extent that it is present, complete restoration of healthy function is impossible without addressing it. Because of the wide range of therapeutic inputs and conditions that that may utilize these mechanisms, we foresee the possibility of evolving multidisciplinary explorations of neurontopathic methods. The scope, laws of application and interpretation of results will be an evolving study.

While neurontogenic treatment of mPTSD is of demonstrated benefit in the reduction of symptomatic MSD, it is worth noting that it may offer a worthwhile

preventative benefit as well. Anterior cruciate ligament injuries, for instance, are believed to have altered muscle activation as part of their cause. [182,183] Removing maladaptive muscle strategies from previous injuries could be preventative of many injuries in athletes who place demands on their bodies that will magnify the effects of muscle imbalance. Additionally, loss of the reverse myotatic reflex (which causes overstretched muscles to reflexively stop contracting and lengthen), commonly found in compensatory or adaptive patterns, could be a risk factor for injury. Any past injury site is likely to have alteration of muscle function on examination, whether the area is symptomatic or not.

It is worth considering the implications of the putative existence of mPTSD on how clinicians interact with the body. We can now venture a general mechanism that answers the question posited earlier: 'why is a muscle weak?' Muscle weakness, the mPTSD hypothesis posits, is not a random event, it is, rather, the result of an intelligent decision made at some point in the recent or distant past, which has become habituated and, in the absence of ongoing need for the adaptation, has become maladaptive.

Behavioral and neurobehavioral analysis suggest that a muscle test, whether the result is strength or weakness, is always in context. This means that even in a supine, relaxed subject, an isolated muscle test is a single piece of a larger strategic pattern in which the organism happens to be engaged. A muscle test may be best understood as one point of observation of a larger pattern, a pattern which can shift and change as its different facets are treated or challenged via sensory input, purposeful assumption of

a posture, or intentional or unintentional focus on particular memory, emotion or thought that unconsciously activates an associated motor pattern.

The context is necessarily part of the test – the practitioner and practitioner's instructions become the $S^D$, in ways that may be better planned if the practitioner is aware of the possibility. This understanding provides a mechanism for some 'applied' aspects of applied kinesiology systems such as *therapy localization*, wherein manual contact of an area of the body causes a previously strong muscle to become weak or visa-versa. At the same time, if muscle weakness is understood as first and foremost a learned adaptive or maladaptive behavior, the connectivistic aspects of applied kinesiology (i.e. specific neurolymphatic or neurovascular points related to specific muscles) may be challenged.

In context, strong is not always good and weak not always bad, and a previously strong muscle that weakens after therapy or usage may be progress. Recall the 'switching' compensation, where the patient presents with inhibition of left knee muscles when the right knee is symptomatic. In such cases it is difficult to assess and therapeutically impact the symptomatic knee until its muscles are displaying their true(r) state. Given this understanding, in the absence of frank nerve pathology, we can no longer think of the muscle test as an absolute or long-term condition. Being contextual and changeable, shifting displays of facilitation patterns provide a window into the immediate state of the patient. If treatment applied to the aberrant patterns changes the display and symptoms, the idea of a fixed diagnosis may lose relevance.

If, on the other hand, the same weakness recurs after neurontogenic treatment with no ongoing improvement either in frequency or intensity of symptoms, it may be an

indication of actual (not behavioral) pathology at some level that requires adaptation on the part of the body. When confronted with a patient in pain or illness, particularly when their condition cannot be adequately explained by pathological, pathological, radicular or organic disease, neurobehavioral maladaptation is likely to be present as a central cause, not just a peripheral reaction.

When treating a patient, these differentiations allow us to begin to more precisely utilize methods that will best support the patient. We can now ask if our desired effect is (re)training - encouraging more successful adaptation, or de-training - elimination of outdated (mal)adaptation.

Using the 4 step procedure, (1- determine the present state and priority, 2- evoke a specific aspect of the negative state, 3- apply a treatment and 4- re-evaluate) it is possible that many therapies will successfully achieve the neurontogenic effect of restoring function without (re)training. Several chiropractically evolved techniques that we are familiar with *may* be achieving *some* of their results by systematically working with maladaptive memory elimination.

We suggest that many conditions are primarily the result of maladaptation, and all MSD and pain will have, as either a cause or an unavoidable result, adaptive conditioning that will then contribute to the symptomology and maintenance of the condition. The actual contribution of adaptive conditioning to any single patient will be variable, empirically determined on a patient to patient basis. This determination may be made by screening for organic pathology before undertaking neurontogenic treatment (particularly if signs, symptoms and history indicate probability) and/or as a conclusion after a short series of treatments without satisfactory progress.

We hope that this paper provides a framework for beginning to investigate the scope of its effects on musculoskeletal, psychological, neurological and even biochemical dysfunctions, which, according to behavioral precepts, will be affected by learning.

This paper, introducing as it does, a new paradigm for thinking about health and disease based on clinical observation and a small pilot study, is written in the hope that it spurs further interest and research in new ways of dealing with musculoskeletal dysfunction and other conditions that may also have maladaptive conditioning at their core. We welcome any and all comment, and invite inquiries and collaborations on future research to elucidate its hypothesis.

# Abbreviations

MMT – manual muscle test(ing)

NE- norepinephrine

AK – Applied Kinesiology (the organized specialty)

CK- Clinical Kinesiology

EMDR – Eye Movement Desensitization and Reprocessing

EP- energy psychology

MSD - musculoskeletal dysfunction

PTSD- post traumatic stress disorder

CR- conditioned reflex

CS- conditioned stimulus

UCS - unconditioned stimulus

UCR – unconditioned reflex

$S^D$ - discriminative stimulus

LC - locus ceruleus

SM - spinal manipulation

## Competing interests

RW employs the principles elucidated herein in his private practice in the evaluation and treatment of patients.

## Authors' contributions

RW was primary author of this paper, from conception of the hypothesis and study to final editing and approval of final manuscript, including all observations and studies.

PL provided additional depth, context, focus and editorial assistance for the Background section, in particular adding most of the content regarding behavioral physiology and principles, and elucidating implications of behavioral principles to musculoskeletal function.

## References

1. Marcus, DA: *Chronic pain: a primary care guide to practical management.* Humana Press: 2005

2. Esty, ML, Perlman, E: **Neurotherapy treatment of fibromyalgia using EEG-based stimulation.** Fibromyalgia Frontiers 2003, **Volume 11**, Number 4

3. Zhang, D, Jain, AK: *Advances in biometrics: International Conference*. ICB 2006, Hong Kong, China, Springer, 2006

4. Enoka, RM: *Neuromechanics of human movement*. Human Kinetics 2002

5. Denegar, C: *Therapeutic modalities for athletic injuries.* Human Kinetics 2000; 3

6. Astrom M, Rausing A: **Chronic achilles tendinopathy, a survey of surgical and histopathologic findings.** Clin Orthop. 1995, **316**:151-164.

7. Jozsa L, Reffy A, Kannus P, Demel S, Elek E: **Pathological alterations in human tendons.** Arch Orthop Trauma Surg. 1990, **110**:15-21.

8. van der Kolk, BA: **The body keeps score: memory and the evolving psychobiology of posttraumatic stress.** Harvard Rev Psychiatry 1994, **1**:253-65

9. Ogden, P, Minton, K: **Sensorimotor Psychotherapy: One Method for Processing Traumatic Memory.** Traumatology, 2000, VI (3):Article 3 Published online. [http://www.fsu.edu/~trauma/v6i3/v6i3a3.html]

10. Selye, H: *The Stress of life*. New York: McGraw-Hill, 1956

11. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press 2000

12. Walther DS: *Applied Kinesiology. Volume 1: basic procedures and muscle testing*. Volume 1. 2nd edition. Pueblo, CO, Systems DC; 1981.

13. Cuthbert, SC and Goodheart, GJ Jr: **On the reliability and validity of manual muscle testing: a literature review** Chiropr Osteopat. 2007; **15**: 4.

14. *Guides to the Evaluation of Permanent Impairment, 5th Edition***.** American Medical Association, 2001:510.

15. Kendall FP, McCreary E, Provance PG: *Muscles: Testing and Function***.** Baltimore: Williams & Wilkins, 1993.

16. Gantseva, ZA: **A method of study of the human myotatic reflex** Journal Bulletin of Experimental Biology and Medicine, 1962 **52,(3)**

17. Feinstein, D: **Energy Psychology: A Review of the Preliminary Evidence Draft version 2.3** 9/10/06 [http://www.innersource.net/energy_psych/epi_research.htm] (used with permission of author)

18 Chambless, D.L. et al: **Update of empirically validated therapies II**. The Clinical Psychologist, 1998, **51***,* 3-16.

19. Carlson, J., Chemtob, C.M., Rusnak, K., Hedlund, N.L, & Muraoka, M.Y: **Eye movement desensitization and reprocessing (EMDR): Treatment for combat-related post-traumatic stress disorder.** Journal of Traumatic Stress, 1998, **11***,* 3-24

20. http://www.emofree.com

21. Wells S, Polglase K, Andrews HB, Carrington P, Baker AH: **Evaluation of a meridian-based intervention, Emotional Freedom Techniques (EFT), for reducing specific phobias of small animals**. J Clin Psychol. 2003, **59**(9):943-66.

22. Pittenger,C, Fasano, S, Mazzocchi-Jones, D, Dunnett, SB, Kandel, ER, and Brambilla, R: **Impaired Bidirectional Synaptic Plasticity and Procedural Memory Formation in Striatum-Specific cAMP Response Element-Binding Protein-Deficient Mice.** The Journal of Neuroscience, 2006, **26**(10):2808-2813

23. Scaer, R: *The Body Bears the Burden – Trauma, Dissociation, and Disease*. Haworth Medical Press, NY, 2001

24. Kronenbuerger, M, Gerwig, M, Brol, B, Block, F, Timmann, D: **Eyeblink conditioning is impaired in subjects with essential tremor.** Brain 2007, **130(6)**:1538-1551

25. Kargo WJ, Nitz DA: **Early skill learning is expressed through selection and tuning of cortically represented muscle synergies**. J Neurosci. 2003, **23(35)**:11255-69.

26. Assaiante C, Mallau S: **Development of postural control in healthy children: a functional approach.** Neural Plast. 2005, **12**(2-3):109-18

27. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press 2000

28. Weiss, E.J. & Flanders, M: **Muscular and postural synergies of the human hand**. *J Neurophysiol,* 2004, **92**, 523-535

29. d'Avella, A, Bizzi, E: **Shared and specific muscle synergies in natural motor behaviors.** PNAS, 2005**, vol 102**, **no. 8**, 3076-3081

30. Kao, JT, Pink, M, Jobe, F, Perry, J: **Electromyographic Analysis of the Scapular Muscles During a Golf Swing**. The American Journal of Sports Medicine, **Vol 23**, No 1

31. Westgaard, R.H. and de Luca, C. J: **Motor Unit Substitution in Long-Duration Contractions of the Human Trapezius Muscle.** The Journal of Neurophysiology, 1999, **Vol. 82 No. 1,** 501-504

32 Grasso R, Ivanenko YP, Zago, M, Molinari, M, Scivoletto, G, Castellano, V, Macellari, V, Lacquaniti, F: **Distributed plasticity of locomotor pattern generators in spinal cord injured patients**. Brain. 2004, **127(Pt 5)**:1019-34.

33. Reinkensmeyer DJ, Emken JL, Cramer SC: **Robotics, motor learning, and neurologic recovery**. Annu Rev Biomed Eng. 2004, **6**:497-525.

34. Schmidt, RA – **A Schema theory of Discrete Motor Skill Learning**. Psychological Review 1975, **82** 225-260

35. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press 2000

36. Damasio, A: *The Feeling of What Happens*. Harcourt, Brace and Co. 1999:147-8

37. Stinear CM, Byblow WD, Steyvers, M, Levin, O, Swinnen, SP: **Kinesthetic, but not visual, motor imagery modulates corticomotor excitability**. Exp Brain Res. 2006, **168(1-2)**:157-64

38 Byl NN: **What can we learn from animal models of focal hand dystonia?** Rev Neurol (Paris). 2003, **159(10 Pt 1)**:857-73.

39. Gandevia, SC, Wilson, LR, Inglis, JT, Burke, D: **Mental rehearsal of motor tasks recruits α-motoneurones but fails to recruit human fusimotor neurones selectively**. The Journal of Physiology 1997, **505 (1)**, 259–266

40. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press 2000, 225

41. Tjernstrom F, Fransson PA, Hafstrom A, Magnusson M: **Adaptation of postural control to perturbations--a process that initiates long-term motor memory**. Gait Posture. 2002 Feb;**15(1)**:75-82.

42. Perrin P, Schneider D, Deviterne D, Perrota, C, Constantinescu, L: **Training improves the adaptation to changing visual conditions in maintaining human posture control in a test of sinusoidal oscillation of the support**. Neurosci Lett. 1998 **245(3)**:155-8

43. van Vliet PM, Heneghan NR. **Motor control and the management of musculoskeletal dysfunction**. Man Ther. 2006, **11(3)**:208-13

44. Ervilha UF, Farina D, Arendt-Nielsen, L, Graven-Nielsen, T: **Experimental muscle pain changes motor control strategies in dynamic contractions**. Exp Brain Res. 2005 **164(2)**:215-24

45. Fernández-Ruiz, J, Hall-Haro, C, Díaz, R, Mischner, J, Vergara, P, and Lopez-Garcia JC; **Learning Motor Synergies Makes Use of Information on Muscular Load**. Learning & Memory, 2000, **Vol. 7, No. 4**: 193-198

46. Damasio, A; *The feeling of what happens. Body and Emotion in the Making of Consciousness* Harcourt, 1999:147

47. Enoka, RM, *Neuromechanics of movement, 3rd ed*. Human Kinetics, Champaign, IL, 2002: 299,306

48. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press 2000

49. Riemann, BL Lephart, SM: **The Sensorimotor System, Part I: The Physiologic Basis of Functional Joint Stability**. J Athl Train. 2002, **37(1)**: 71–79.

50. Delgado-Garcia JM, Gruart A: **The role of interpositus nucleus in eyelid conditioned responses**. Cerebellum. 2002, **1(4)**:289-308.

51. Smith AM: **Babinski and movement synergism**. Rev Neurol (Paris). 1993, **149(12)**:764-70.

52. Enoka, RM: *Neuromechanics of movement, 3rd ed*. Human Kinetics, Champaign, IL, 2002: 341

53. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press, 2000

54. Damasio, A: *Looking for Spinoza* Harcourt, 2003:334

55. Sacchetti B, Scelfo B, Strata P: **The cerebellum: synaptic changes and fear conditioning**. Neuroscientist. 2005, **11(3)**:217-27.

56. Bremner JD: **Neuroimaging in posttraumatic stress disorder**. Psychiatr Annals 1998, **28**:445–450

57. Alheid GF: **Extended amygdala and basal forebrain**. Ann N Y Acad Sci. 2003, **985**:185-205.

58. Graybiel AM: **The basal ganglia and chunking of action repertoires**. Neurobiol Learn Mem. 1998, **70(1-2)**:119-36

59. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press, 2000

60. Chuma T, Faruque Reza M, Ikoma K, Mano Y.J: **Motor learning of hands with auditory cue in patients with Parkinson's disease**. Neural Transm. 2006, **113(2)**:175-85.

61. Sacchetti B, Sacco T, Strata P: **Reversible inactivation of amygdala and cerebellum but not perirhinal cortex impairs reactivated fear memories**. Eur J Neurosci. 2007, **25(9)**:2875-84.

62. Mintz M, Wang-Ninio Y: **Two-stage theory of conditioning: involvement of the cerebellum and the amygdala**. Brain Res. 2001, **897(1-2)**:150-6 .

63. Bremner JD: **Neuroimaging in posttraumatic stress disorder**. Psychiatr Annals 1998, **28**:445–450

64. Rauch, S.L., van der Kolk, B.A., Fisler, R.E., Alpert, N.M., Orr, S.P., Savage, C.R., Fischman, A.J., Jenike, M.A., Pitman, R.A: **A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script driven imagery**. Archives of General Psychiatry, 1996, **53**:380-387.

65. Shin, L.M., Kosslyn, S.M., McNally, R.J., Alpert, N.M., Thompson, W.L., Rauch, S.L., Macklin, M.L., & Pitman, R.K.: **Visual imagery and perception in posttraumatic stress disorder: A positron emission tomographic investigation**. Archives of General Psychiatry, 1997, **54:**237-233.

66. McGaugh JL: **Memory consolidation and the amygdala: a systems perspective**. Trends Neurosci. 2002, **25(9)**:456

67. Ferry B, McGaugh JL: **Role of amygdala norepinephrine in mediating stress hormone regulation of memory storage**. Acta Pharmacol Sin. 2000, **21(6)**:481-93

68. Hodges PW: **Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain**. Exp Brain Res. 2001, **141(2)**:261-6.

69. Scaer, R: *The Body Bears the Burden: Trauma, Dissociation, and Disease*. Haworth Medical Press, 2001

70. Scaer, R: *The Body Bears the Burden: Trauma, Dissociation, and Disease*. Haworth Medical Press, 2001

71. Altemus, M, Dhabhar, F, Yang, R: **Immune Function in PTSD**. Ann. N.Y. Acad. Sci., 2006, **1071**:167–183

72. Liebenson, C: **Sensory-Motor Training, Part I** Dynamic Chiropractic, 2001, **Volume 19, Issue 09**

73. Canli, T, Zhao, Z, Brewer, J, Gabrieli, JDE, Cahill, L: **Event-related activation in the human amygdala associates with later memory for individual emotional experience**. *J Neurosci*, 2000, **20**:RC99:1-5.

74. Levine, PA: *Waking the Tiger: Healing Trauma*. 1997, North Atlantic Books

75. http://www.theinstitute.org/trauma/levin_article.shtml

76. Scaer, R: **The neurophysiology of dissociation and chronic disease.** Applied Psychophysiology and Biofeedback, 2001, **26(1)**, 73-91

77. Bertolini A, Vergoni AV, Poggioli R, Gessa GL: **Morphine and beta-endorphin antagonize posture and locomotor disorders induced by the injection of ACTH 1-24 in the rat locus coeruleus**. Life Sci. 1986 **27**:38(4):373-7.

78. Antolic V, Strazar K, Pompe B, Pavlovcic V, Vengust R, Stanic U, Jeraj J: **Increased muscle stiffness after anterior cruciate ligament reconstruction--memory on injury?** Int Orthop. 1999, **23(5)**:268-70

79. Falla D, Bilenkij G, Jull G: **Patients with chronic neck pain demonstrate altered patterns of muscle activation during performance of a functional upper limb task**. Spine. 2004 **1**:29(13):1436-40.

80. Cools AM, Witvrouw EE, Declercq GA, Danneels LA, Cambier DC: **Scapular muscle recruitment patterns: trapezius muscle latency with and without impingement symptoms**. Am J Sports Med. 2003, **31(4)**:542-9.

81. Barnes, T.D.: Kubota, Y.: Hu, D.: Jin, D.Z. & Graybiel, A.M: **Activity of striatal neurons reflects dynamic encoding and recoding of procedural memories**. *Nature,* 2005, **437**:1158-1161

82. Chakravarthy SV, Ghosh J: **On Hebbian-like adaptation in heart muscle: a proposal for 'cardiac memory'**. Biol Cybern. 1997 **76(3)**:207-15.

83. Willingham, D.B.: Nissen, M.J. & Bullemer, P: **On the development of procedural knowledge**. J Exp Psychol Learn Mem Cogn, 1989, **15**:1047-1060

84. Panksepp, J. *Affective neuroscience: The foundations of human and animal emotions*. Oxford University Press 1998

85. Ranney, D: **Anatomy of Pain.** Ontario Inter-Urban Pain Conference, 1996, [http://www.ahs.uwaterloo.ca/~ranney/painanat.html]

86. St-Onge N, Duval, N, Yahia, LH, Feldman, A: **Interjoint coordination in lower limbs in patients with a rupture of the anterior cruciate ligament of the knee joint**. Knee Surg Sports Traumatol Arthrosc. 2004, **12(3)**:203-16.

87. Mannion AF, Adams MA, Dolan P: **Sudden and unexpected loading generates high forces on the lumbar spine**. Spine. 2000, **1**:25(7):842-52.

88. Schouten, WG, Wiegant, VM: **Individual responses to acute and chronic stress in pigs**. Acta Physiol Scand Suppl, 1997, **640**:88-91

89. Deeprose, C, Andrade, J, Varma, S, Edwards N: **Unconscious learning during surgery with propofol anaesthesia.** British Journal of Anaesthesia, 2004, **Vol. 92, No. 2:**171-177

90. Deebiec, J, Ledoux, J.E: **Noradrenergic Signaling in the Amygdala Contributes to the Reconsolidation of Fear Memory: Treatment Implications for PTSD**. *Ann N Y Acad Sci,* 2006, **1071:**521-524

91. Bloemsaat JG, Meulenbroek RG, Van Galen GP: **Differential effects of mental load on proximal and distal arm muscle activity**. Experimental Brain Research, 2005, **167(4)**:622-634

92. Knardahl S: **Psychological and social factors at work: contribution to musculoskeletal disorders and disabilities**. G Ital Med Lav Ergon. 2005 **27(1)**:65-73.

93. Ruggieri V, Giustini S: **Styles of modulation of emotional behavior: relationship with myographic tension and morphology of the shoulder**. Percept Mot Skills. 1991 **72(3 Pt 2)**:1167-71.

94. Weinberg RS, Hunt VV: **Effects of structural integration on state-trait anxiety**. J Clin Psychol. 1979, **35(2)**:319-22.

95. Cooper BC, Rabuzzi DD: **Myofacial pain dysfunction syndrome: a clinical study of asymptomatic subjects**. Laryngoscope. 1984 **94(1)**:68-75.

96. St-Onge N, Duval, N, Yahia, LH, Feldman, A: **Interjoint coordination in lower limbs in patients with a rupture of the anterior cruciate ligament of the knee joint**. Knee Surg Sports Traumatol Arthrosc. 2004, **12(3)**:203-16.

97. Riemann, BL Lephart, SM: **The Sensorimotor System, Part I: The Physiologic Basis of Functional Joint Stability** J Athl Train. 2002 **37(1)**: 71–79.

98. Ervilha UF, Farina D, Arendt-Nielsen, L, Graven-Nielsen, T: **Experimental muscle pain changes motor control strategies in dynamic contractions**. Exp Brain Res. 2005 **164(2)**:215-24

99. Schmitt, W Jr, Yanuck, SF: **Expanding the Neurological Examination Using Functional Neurologic Assessment: Part II** Intern. J. Neuroscience, 1999, **97**:77-108

100. Damasio, A: *The Feeling of What Happens*. Harcourt, Brace and Co 1999

101. Scaer, R: **The neurophysiology of dissociation and chronic disease.** Applied Psychophysiology and Biofeedback, 2001, **26(1)**, 73-91

102. Pavlov IP: *Conditioned reflexes: an investigation of the physiological activity of the cerebral cortex*. London: Oxford UP, 1927

103. Sangha, S, Scheibenstock, A: Morrow, R, Lukowiak, K: **'Extinction requires new RNA and protein synthesis and the soma of the cell right pedal dorsal in Lymnaea stagnalis.'**. J Neurosci 2003, **23(30):**9842-9851.

104. Suzuki A, Josselyn, SA, Frankland, PW, Masushige, S, Silva, AJ, Kida, S: **Memory reconsolidation and extinction have distinct temporal and biochemical signatures**. J Neurosci. 2004, **24(20)**:4787-95

105. Milad MR, Rauch SL, Pitman RK, Quirk GJ: **Fear extinction in rats: implications for human brain imaging and anxiety disorders**. *Biol Psychol*. 2006, **73**:61-71

106. Kalisch, Korenfeld, RE, Stephan, KE, Weiskopf, N, Seymour, B, Dolan, RJ: **Context-Dependent Human Extinction Memory is Mediated by a Ventromedial Prefrontal and Hippocampal Network** The Journal of Neuroscience, 2006, **26(37)**:9503-9511

107. Deebiec, J, Ledoux, JE: **Noradrenergic Signaling in the Amygdala Contributes to the Reconsolidation of Fear Memory: Treatment Implications for PTSD**. Ann N Y Acad Sci 2006, **1071:**521-524.

108. Debiec, J, Ledoux, .E: **Disruption of reconsolidation but not consolidation of auditory fear conditioning by noradrenergic blockade in the amygdala.** Neuroscience 2004, **129(2)**, 267—272.

109. Stapleton JA, Taylor S, Asmundson GJ: **Effects of three PTSD treatments on anger and guilt: exposure therapy, eye movement desensitization and reprocessing, and relaxation training**. J Trauma Stress. 2006 **19(1)**:19-28.

110. Fieinstein, D: **Energy Psychology: A Review of the Preliminary Evidence Draft version** 9/10/06 [http://www.innersource.net/energy_psych/epi_research.htm] (used with permission of author)

111. Threlfo C, Grant M: **EMDR in the treatment of chronic pain**. J Clin Psychol. 2002, **58(12)**:1505-20

112. Wade TC, Wade DK: **Integrative psychotherapy: combining ego-state therapy, clinical hypnosis, and eye movement desensitization and reprocessing (EMDR) in a psychosocial developmental context**. Am J Clin Hypn. 2001 **43(3-4)**:233-45.

113. Najib, A, Lorberbaum, JP, Kose, S, Bohning, DE, George, MS: **Regional Brain Activity in Women Grieving a Romantic Relationship Breakup Am J Psychiatry** 2004, **161**:2245-2256

114. Doyere V, Debiec J, Monfils MH, Schafe GE, LeDoux JE: **Synapse-specific reconsolidation of distinct fear memories in the lateral amygdala.** Nat Neurosci. 2007, 10(4):414-6

115. Logan CG, Grafton, ST: **Functional anatomy of human eyeblink conditioning determined with regional cerebral glucose metabolism and positron-emission tomography.** Proc. Natl. Acad. Sci. 1995, **92:**7500-7504

116. Krakauer JW, Shadmehr R: **Consolidation of motor memory**. Trends Neurosci. 2006, **29(1)**:58-64.

117. Sacchetti B, Scelfo B, Strata P: **The cerebellum: synaptic changes and fear conditioning**. Neuroscientist. 2005, **11(3)**:217-27.

118 Bremner JD: **Neuroimaging in posttraumatic stress disorder**. Psychiatr Annals 1998, **28**:445–450

119. Inda, MC, Delgado-Garcia, JM, Carrión, AM: **Acquisition, Consolidation, Reconsolidation, and Extinction of Eyelid Conditioning Responses Require De Novo Protein Synthesis**. J Neurosci. 2005 **25(8)**:2070-80.

120. Kobayashi K: **Role of catecholamine signaling in brain and nervous system functions: new insights from mouse molecular genetic study**. J Investig Dermatol Symp Proc. 2001 **6(1)**:115-21.

121. McGaugh JL: **Memory consolidation and the amygdala: a systems perspective**. Trends Neurosci. 2002, **25(9)**:456

122. Cartford MC, Gould T, Bickford PC: **A central role for norepinephrine in the modulation of cerebellar learning tasks**. Behav Cogn Neurosci Rev. 2004, **3(2)**:131-8

123. Dietrichs E: **Divergent axon collaterals to cerebellum and amygdala from neurons in the parabrachial nucleus, the nucleus locus coeruleus and some adjacent nuclei. A fluorescent double labelling study using rhodamine labelled latex microspheres and fast blue as retrograde tracers**. Anat Embryol (Berl). 1985, **172(3)**:375-82.

124. Scaer, R: *The Body Bears the Burden: Trauma, Dissociation, and Disease*. Haworth Medical Press, 2001:10,11

125 Deebiec, J. & Ledoux, J.E: **Noradrenergic Signaling in the Amygdala Contributes to the Reconsolidation of Fear Memory: Treatment Implications for PTSD**. Ann N Y Acad Sci, 2006, **1071**:521-524

126. Cedarbaum JM, Aghajanian GK: **Afferent projections to the rat locus coeruleus as determined by a retrograde tracing technique**. J Comp Neurol. 1978 **178**:1-16.

127. Bertolini A, Vergoni AV, Poggioli R, Gessa GL: **Morphine and beta-endorphin antagonize posture and locomotor disorders induced by the injection of ACTH 1-24 in the rat locus coeruleus**. Life Sci. 1986, **38(4)**:373-7.

128. Craig AD:. **Distribution of brainstem projections from spinal lamina I neurons in the cat and the monkey**. J Comp Neurol. 1995, **361(2)**:225-48.

129 Scaer, R:*The Body Bears the Burden: Trauma, Dissociation, and Disease* Haworth Medical Press, 2001

130. Pitman, RK, Delahanty, DL: **Conceptually driven pharmacologic approaches to acute trauma**. CNS Spectr 2005 **10(2)**:99-106.

131. Debiec, J, Ledoux, JE: **Disruption of reconsolidation but not consolidation of auditory fear conditioning by noradrenergic blockade in the amygdala**. Neuroscience, 2004 **129(2):**267-272.

132. Kobayashi K: **Role of catecholamine signaling in brain and nervous system functions: new insights from mouse molecular genetic study**. J Investig Dermatol Symp Proc. 2001 **6(1)**:115-21.

133. Pepper CM, Henderson G. **Opiates and opioid peptides hyperpolarize locus coeruleus neurons in vitro.** Science. 1980 **209(4454)**:394-5.

134. Arnsten AF, Segal DS, Loughlin SE, Roberts DC: **Evidence for an interaction of opioid and noradrenergic locus coeruleus systems in the regulation of environmental stimulus-directed behavior**. Brain Res. 1981, **222(2)**:351-63.

135. Bertolini A, Vergoni AV, Poggioli R, Gessa GL: **Morphine and beta-endorphin antagonize posture and locomotor disorders induced by the injection of ACTH 1-24 in the rat locus coeruleus**. Life Sci. 1986, **38(4)**:373-7.

136. Bustos, SG, Maldonado, H,Molina, VA: **Midazolam disrupts fear memory reconsolidation**. *Neuroscience* 2006 **139(3)**, 831--842.

137. Taylor FB: **Tiagabine for posttraumatic stress disorder: a case series of 7 women**. J Clin Psychiatry. 2003, **64(12)**:1421-5.

138. Akirav I, Raizel H, Maroun M: **Enhancement of conditioned fear extinction by infusion of the GABA agonist muscimol into the rat prefrontal cortex and amygdala**. Eur J Neurosci. 2006, **23(3)**:758-64

139. Salinas JA, McGaugh JL: **The amygdala modulates memory for changes in reward magnitude: involvement of the amygdaloid GABAergic system**. Behav Brain Res. 1996 **80(1-2)**:87-98.

140. Pitman, RK, Delahanty, DL: **Conceptually driven pharmacologic approaches to acute trauma**. CNS Spectr 2005 **10(2)**:99-106.

141. Cartford MC, Gould T, Bickford PC: **A central role for norepinephrine in the modulation of cerebellar learning tasks**. Behav Cogn Neurosci Rev. 2004, **3(2)**:131-8

142. Attwell PJ, Cooke SF, Yeo CH: **Cerebellar function in consolidation of a motor memory**. Neuron. 2002, **34(6)**:1011-20.

143. Markowitsch HJ, Irle E, Emmans D: **Cortical and subcortical afferent connections of the squirrel monkey's (lateral) premotor cortex: evidence for visual cortical afferents**. Int J Neurosci. 1987, **7(3-4)**:127-48.

144. Scaer, R: **The Body Bears the Burden: Trauma, Dissociation, and Disease Haworth Medical Press.** 2001:53

145. Urasaki E, Wada S, Kadoya C, Yokata A, Matsuoka S: **Spinal intramedullary recording of human somatosensory evoked potentials**. Electroencephalogr Clin Neurophysiol. 1990 **77(3)**:233-6.

146. Lundberg A, Malmgren K, Schomburg ED: **Reflex pathways from group II muscle afferents. 2. Functional characteristics of reflex pathways to alpha-motoneurones**. Exp Brain Res. 1987, **65(2)**:282-93.

147. Kimura F, Nakamura S: **Locus coeruleus neurons in the neonatal rat: electrical activity and responses to sensory stimulation**. Brain Res.1985 **355(2)**:301-5.

148. Dunckley P, Wise RG, Fairhurst M, Hobden P, Aziz Q, Chang L, Tracey I: **A comparison of visceral and somatic pain processing in the human brainstem using functional magnetic resonance imaging**. J Neurosci. 2005, **25(32)**:7333-41

149. Kent, C: **Models of Vertebral Subluxation: A Review Journal of Vertebral Subluxation Research.** Journal of Vertebral Subluxation Research, 1996, **1(1)**:11-17

150. Southwick, SM, Bremner,JD, Ann Rasmusson, A, Morgan III, CA, Arnstena, A Charneya, DS: **Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder.** Biol Psychiatry. 1999, **46(9)**:1192-204

151. Solntseva SV, Nikitin VP, Kozyrev SA, Shevelkin AV, Lagutin AV, Sherstnev VV. **Effects of protein synthesis inhibitors during reactivation of associative memory in the common snail induces reversible and irreversible amnesia**. Neurosci Behav Physiol. 2007 **37(9)**:921-8.

152. Bergmann, U: **Speculations on the neurobiology of EMDR**. Traumatologye, 9998 **4(1)**:Article 2

153. Kaur S, Saxena RN, Mallick BN: **GABAergic neurons in prepositus hypoglossi regulate REM sleep by its action on locus coeruleus in freely moving rats.** Synapse, 2001, **42(3):**141 - 150

154. Wilson, D.L., Silver, S.M, Covi, W.G., & Foster, S: **Eye movement desensitization and reprocessing: Effectiveness and autonomic correlates**. Journal of Behavior Therapy & Experimental Psychiatry, 1996, **27(3):**219-229.

155. Süer C, Ozesmi C, Temoçin S, Doğan P, Ciliv G: **The effects of immobilization stress on electrodermal activity and brain catecholamine levels in rats**. Int J Neurosci. 1992, **65(1-4)**:91-101.

156. Wang S: **Traumatic stress and attachment**. Acta Physiol Scand Suppl. 1997, **640**:164-9

157. Villarreal G, Hamilton DA, Graham DP, Driscoll I, Qualls C, Petropoulos H, Brooks WM: **Reduced area of the corpus callosum in posttraumatic stress disorder**. Psychiatry Res. 2004, **131(3)**:227-35.

158. Stickgold R: **EMDR: a putative neurobiological mechanism of action**. J Clin Psychol. 2002, **58(1)**:61-75.

159. Bergmann, U: **Speculations on the neurobiology of EMDR** Traumatologye, 1998 **4(1)**:Article 2

160. Hooke, W: **A Review of Thought Field Therapy** Traumatology **3(2)**:Article 3

161 Feinstein, D: **Energy Psychology: A Review of the Preliminary Evidence Draft version** 9/10/06 [http://www.innersource.net/energy_psych/epi_research.htm] (used with permission of author)

162. Stahl S, Briley M. Understanding pain in depression. Hum Psychopharmacol. 2004 Oct:19 Suppl 1:S9-S13

163. Ma W, Eisenach JC: **Chronic constriction injury of sciatic nerve induces the up-regulation of descending inhibitory noradrenergic innervation to the lumbar dorsal horn of mice**. Brain Res. 2003, **970(1-2)**:110-8

164. Yu-Zhen Pan, De-Pei Li, Shao-Rui Chen, and Hui-Lin Pan: **Activation of δ-Opioid Receptors Excites Spinally Projecting Locus Coeruleus Neurons Through Inhibition of GABAergic Inputs.** J Neurophysiol 2002, **88**:2675-2683,

165. Wu GC, Zhu J, Cao X: **Involvement of opioid peptides of the preoptic area during electroacupuncture analgesia**. Acupunct Electrother Res. 1995, **20(1)**:1-6.

166. Bragin EO, Emel'ianenko IV, Popkova EV: **The ratio of the contents of beta-endorphin, adrenaline and noradrenaline in the hypothalamus of rats with an experimental stomach ulcer and during acupuncture**. Fiziol Zh SSSR Im I M Sechenova, 1989 **75(7)**:917-22.

167. Aston-Jones G, Chiang C, Alexinsky T: **Discharge of noradrenergic locus coeruleus neurons in behaving rats and monkeys suggests a role in vigilance**. Prog Brain Res. 1991, **88**:501-20.

168. Craig AD: **Distribution of brainstem projections from spinal lamina I neurons in the cat and the monkey**. J Comp Neurol. 1995,**361(2)**:225-48.

169. Neuhuber WL, Bankoul S: **Specifics of innervation of the cranio-cervical transition**. Orthopade. 1994, **23(4)**:256-61.

170. Usunoff KG, Itzev DE, Rolfs A, Schmitt O, Wree A: **Brain stem afferent connections of the amygdala in the rat with special references to a projection from the parabigeminal nucleus: a fluorescent retrograde tracing study**. Anat Embryol (Berl). 2006, **211(5)**:475-96

171. Balaban CD: **Neural substrates linking balance control and anxiety**. Physiol Behav. 2002 **77(4-5)**:469-75.

172. Holder, JM, Duncan, RC, Gissen, M, Miller, M, Blum, K: **Increasing retention rates among the chemically dependent in residential treatment: auriculotherapy and**

**subluxation-based chiropractic care.** Molecular psychiatry 2001, **Volume 6 Supplement 1**

173. Yates RG, Lamping DL, Abram NL, Wright C: **Effects of chiropractic treatment on blood pressure and anxiety: a randomized, controlled trial**. J Manipulative Physiol Ther 1988, **11(6)**:484–488

174. Vernon HT, Dhami MS, Howley TP, Annett R: **Spinal manipulation and beta-endorphin: a controlled study of the effect of a spinal manipulation on plasma beta-endorphin levels in normal males**. J Manipulative Physiol Ther. 1986, **9(2)**:115-23.

175. Berthoz, A: *Brain's Sense of Movement.* Harvard University Press, 2000:7

176. Vol'f NV, Tsvetovskii SB, Il'iuchenok RIu. **Effect of destruction of the amygdaloid complex on the cardiac and motor components of a conditioned defense reaction**. Zh Vyssh Nerv Deiat Im I P Pavlova. 1979, **29(4)**:751-6.

177. Fernández-Ruiz, J, Hall-Haro, C, Díaz, R, Mischner, J, Vergara, P, and Lopez-Garcia JC: **Learning otor Synergies Makes Use of Information on Muscular Load.**- Learning & Memory 2000 **7(4):**193-198

178. http://www.m-w.com/dictionary/allopathic

179. NACBT Online Headquarters, National Association of Cognitive-Behavioral Therapists [http://www.nacbt.org/whatiscbt.htm]

180. Haggerty, J: **Psychodynamic Therapy**. PsychCentral.com 2006 [http://psychcentral.com/lib/2006/psychodynamic-therapy/]

181. Pavlov IP: *Conditioned reflexes: an investigation of the physiological activity of the cerebral cortex*. London: Oxford UP, 1927

182. Griffin LY, Agel J, Albohm MJ, Arendt EA, Dick RW, Garrett WE, et al: **Noncontact Anterior Cruciate Ligament Injuries-Risk Factors and Prevention Strategies**. J Am Acad Orthop Surg, 2000, **8(3)**:141-150.

183. Hewett, T E, Zazulak, B T, Myer G D, Ford K R: **A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes**. Br. J. Sports Med. 2005, **39**:347-350