Asia-Pacific Chiropractic Journal

HYPOTHESIS

Mind, trauma & muscle inhibition Part I:

Experiment and case history yield novel theory of muscular PTSD

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Abstract: *Background:* Musculoskeletal problems are by far the most costly and prevalent group of maladies we face in the U.S. and weakness of individual muscles — clinically revealed through manual muscle testing — can probably be found in every condition. Muscle weakness is listed as a factor in most named problems — e.g. plantar fascitiis, whiplash, tennis elbow. It is also routinely found in 'non-specific' or unexplained problems, most notably low back pain. Weak muscles can also be found in non-symptomatic areas. Diagnostically known as muscle inhibition, it is nonetheless missing from the 70,000 codes of the International Classification of Diseases. While several mechanisms that may describe the onset of inhibition are well accepted, research fails to present a mechanism for the ongoing persistence of the condition, which is known to outlast even 'successful' healing by decades. Resistant to even aggressive physical therapy and targeted exercise, muscle inhibition is essentially a permanent condition.
Hypothesis: Muscle inhibition is sustained by maladaptive learning. Specifically, previous incidents have inculcated (unconscious) beliefs that use of particular muscles will lead to bad outcomes, creating automatic avoidance of use of those muscles.
Method: We experimentally attempt to treat muscle inhibition by applying a psychotherapeutic technique theorised to erase maladaptive learning by blocking 'memory reconsolidation' -the re-storage of memories rendered unstable following recall. Saccadic eye movement (as used in Eye Movement Desensitisation and Reprocessing (EMDR), an accepted therapy for post traumatic stress) is performed by subjects immediately after testing each weak muscle. Experienced muscle testers applied the method on 136 randomly selected weak muscles in 8 subjects.
Results: Eye movements immediately strengthened 91% of muscles, with 84% remaining strong 15 days later. Just over half those muscles 'spontaneously' recovered, assumedly a vicarious effect from treatment of preceding muscles. In a control group of 42 weak muscles, 88% remained weak over similar time period.
Case history: A case history applying this experimental method as the primary therapy adds practical understanding.
Conclusion: This experiment suggests a novel model of muscular PTSD (mPTSD), in which memories of stress and trauma become associated with the use of certain muscles (or specific vectors of movement) leading to chronic avoidance of use of those muscles. Erasing the information from the brain that led to that avoidance is the putative mechanism of cure. A simple treatment for inhibition (mPTSD), if broadly accepted, will be a disruptive innovation in musculoskeletal care, which has been ignoring muscle inhibition in both its studies and treatments for over a century.

Indexing Terms: Muscle inhibition, PTSD, chiropractic, theory, hypothesis.

Background

> *'The pace at which fundamental discoveries of basic science are being uncovered is accelerating, as is the speed at which medical practice is being transformed', states a 2015 Forbes article. 'Metamorphic changes are sweeping a wider breadth of clinical areas more regularly than at any time in the history of science.'* (Gottlieb, 2015)

... this paper addresses the ubiquitous problem that might be commonly called 'chronic muscle weakness' and proposes a novel theory which may better guide the clinical response'

For neuromusculoskeletal disorders, however, while stem cells and platelet rich plasma may offer new hope for some by fortifying degenerated joints and ligaments, and imaging and surgical methods are always improving to find and repair damage, they do nothing to enlarge our basic understanding of

musculoskeletal dysfunction. Fundamentally, they don't help to prevent or treat acute injuries or stop degeneration from occurring in the first place.

Progress in the treatment of 'routine' musculoskeletal problems is stagnant. For over 40 years, despite lack of actual evidence supporting its use, (van den Bekerom et al., 2012) R.I.C.E. – rest, ice, compression and elevation, has been the standard of care in acute injuries. The originator of the acronym now saying that ice and rest may actually delay recovery. (Mirkin, 2014)

'*The World Health Organization*', a 2012 paper on the neurophysiology of arthritis pain reports, '*ranks musculoskeletal disorders as the most frequent cause of disability in the modern world, affecting one in three adults*' worldwide. '*Even more alarming*', it states, '*is that the prevalence of these diseases is rising while our knowledge of their underlying causes is fairly rudimentary. Patients yearn for their chronic pain to disappear; however, currently-prescribed analgesics are largely ineffective and are accompanied by a wide range of unwanted side effects [including addiction*' (Denisco et al., 2008) and death. (Science Daily, 2005) As such, '*millions of people worldwide are suffering from the debilitating effects of joint pain, for which there is no satisfactory treatment.*' (McDougall & Linton, 2012)

According to the *Bone and Joint Initiative*, an ongoing organisation that emerged from the '*bone and joint decade*' (2002-2011), 126.6 million Americans (about one in two adults) suffer from a musculoskeletal condition. Including both treatment and indirect costs like lost wages, The Initiative states, musculoskeletal problems cost the U.S. an astounding $950 billion annually, (years 2004-2006) or 7.4% of the gross domestic product. '*Beyond these statistics*', The Initiative Laments, '*the human toll in terms of the diminished quality of life is immeasurable*' and '*unlikely to improve in the foreseeable future*'. (usbji.org, 2016)

The Initiative hopes its data collection will help to '*delineate the underlying mechanisms of these diseases and their response to treatment. Through such research, novel preventive and therapeutic approaches with potential to mitigate the societal and personal impact of musculoskeletal disease will emerge,*' (Jacobs et al., 2011) but the ongoing data has only highlighted the problem. Despite the new methods offered for chronic musculoskeletal degeneration, no explanations of underlying mechanisms or new treatments have emerged in the nearly 20 years of its existence that might help us avoid these chronic problems, let alone treat them successfully while they are acute.

This paper is the first of a four-part series that we hope will answer the call of The Initiative. It is a novel investigation into a ubiquitous problem that might be commonly called '*chronic muscle weakness.*' In this, the first part of the series, we define the problem and present a new hypothesis which may reveal a core piece of the underlying mechanism of of chronic muscle weakness. To support the hypothesis, we utilise a currently accepted treatment method in a new way to immediately resolve muscle weakness.

The prevalence of R.I.C.E. as the go-to treatment for mild-to-moderate musculoskeletal injuries is based on the assumption that tissue damage is the driver of pain and inflammation in injuries. The hypothesis and experimental treatment results we present here will challenges this basic assumption.

A second challenge to the supremacy of tissue damage comes from observation of treatment of top athletes. In Part II of this series, we will delve more deeply into the potential benefits of treating muscle inhibition by looking retrospectively at the outcomes of chiropractor Craig Buhler's 20 plus year tenure as part of the medical staff of an NBA team. During that time, he discovered that if acute injuries are treated immediately using methods we will be discussing, mild to moderate sprain-strain injuries can regularly recover in minutes, not weeks, without the swelling that is currently treated with R.I.C.E.. (Buhler, n.d.; Jarvis, Kelly B, 1998) If borne out in future studies, this hypothesis and treatments for musculoskeletal problems that arise out of it

will prove to be a disruptive technology for musculoskeletal medicine. A solution to muscle inhibition, if universally applied, will necessitate a re-evaluation of much of what we have believed about the course and treatment of musculoskeletal problems.

The hypothesis we are putting forth is that chronic weakness of individually tested muscles, more technically known as '*muscle inhibition*', is sustained by maladaptive memories, making it similar to some descriptions of post traumatic stress disorder (PTSD). Therefore, we will refer to the mPTSD model or hypothesis 'm' being muscle) when referring to it.

Muscle weakness, an egregiously ignored issue

While there is a paucity of studies looking directly at the relationship between muscle weakness and myofascial pain, (Mense, 2008, p. 24) weakness is a stated factor in diverse 'named' conditions like whiplash, (Jull, 2000; Mense, 2008, p. 258; Prushansky et al., 2005) tennis elbow, (Coombes et al., 2012) runner's knee, (Ireland et al., 2003; Thomee et al., 1999) and plantar fasciitis. (Allen & Gross, 2003) to name a few. Weakness is also mentioned in conjunction with 'non-specific' or unexplained pain syndromes in which mechanisms are unclear. These represent the bulk of musculoskeletal conditions, most prominently low back pain. (Cuthbert et al., 2018; Russo et al., 2018) Weakness is also a cause loss of athletic performance. (Freeman et al., 2013; Mehran et al., 2016)

By definition, the finding of weakness of one or more muscles crossing a joint means that there is muscle imbalance. Other muscles must be recruited to compensate for those that are not fully engaged. (Hodges & Tucker, 2011; Mense, 2008, p. 258; Russo et al., 2018) This causes altered patterns of movement and suggests that the affected joint will be less stable. These factors then predispose those with muscle weakness to future injuries and osteoarthritis. (Michael V. Hurley, 1999; O'Reilly et al., 1997; Rice & McNair, 2010; Slemenda et al., 1997)

'Weakness' is an ambiguous term that can have several meanings. 'Muscle inhibition' is a more specific term referencing the persistent weakness of individual skeletal muscles. Universally, inhibition is clinically revealed with manual muscle testing (MMT). Where weakness can refer to atrophy of one or more muscles (lack of muscle mass) or an individual's subjective experience, 'inhibition' states a neuronal involvement, in which something is interfering with the neuronal signals that are reaching an otherwise healthy and often non-atrophied muscle. (Mense, 2008) A muscle with disuse atrophy because of being in a cast for instance, may or may not be inhibited. Inhibition refers to a mechanism for weakness, but also for the weakness itself. While inhibited muscles are always weak, weak muscles are not always inhibited.

While several mechanisms that may describe the onset of inhibition are well accepted, research fails to present a mechanism for the ongoing persistence of the condition, which is known to outlast even 'successful' healing by decades. (Gabler, 2016; Rice & McNair, 2010)

One accepted initiator of inhibition is known as the '*arthrogenic inhibitory reflex*', in which joint trauma or swelling causes a reflex shutdown of alpha motorneurons (αMNs) of certain muscles. The arthrogenic inhibitory reflex is thought to be a protective measure to prevent the spindle stretch reflex from operating during an injury, potentially causing damage to a muscle that is being rapidly stretched. In an ankle inversion sprain for instance, the *peroneus* muscles of the lateral ankle are rapidly stretched and could tear if they contracted forcefully due to the stretch reflex. *Peroneus* muscles are typically inhibited following an inversion injury. (Palmieri et al., 2004; Palmieri-Smith et al., 2009) Similarly, receiving the lion's share of the research into what is known as '*arthrogenic muscle inhibition*' (AMI), quadriceps are routinely inhibited following anterior cruciate ligament injuries. (Gabler, 2016; Hart et al., 2014; M. V. Hurley et al., 1994; Lewek et al., 2002; Rice & McNair, 2010)

Following the injury, inhibition has also been suggested to be protective, preventing certain movements in the injured joint. Evidence that comes from the immediate treatment of inhibition in sprain-strain injuries however, as mentioned above regarding treatment of NBA players, suggests that the opposite may be true: muscle inhibition could be the cause of many of the symptoms of injury, rather than a protector from the injury.

Neurologists have also described a similar phenomena of '*functional weakness*', '*weakness which is both internally inconsistent and incongruent with any recognizable neurological disease.*' Found through muscle testing as well, (Stone et al., 2010; Van der Ploeg & Oosterhuis, 1991) it is unclear if there is overlap between AMI and functional weakness, since the two literature streams do not refer to each other. Though authors point out that it may arise in conjunction with a physical injury, functional weakness is considered to be part of 'conversion syndromes' in which emotional upset is converted into physical symptoms. (Stone et al., 2009) Awareness of the phenomenon of muscle inhibition, by whatever name is hardly new though; it has been a topic of discussion since the late 19th century. (Dehne, 1955)

By reviewing different literature streams we find that all manner of pain and stress will at least transiently alter muscles. In addition to the arthrogenic reflex, pain, (Falla et al., 2004, 2014; Hodges & Tucker, 2011; Le Pera et al., 2001; Mense, 2008; Muceli et al., 2014) psychological stress, (Hudes, 2011; Stone et al., 2010; Van der Ploeg & Oosterhuis, 1991) and biochemical or organ stress, (Gangemi, 2006; Hoffman & Mendel, 1977; Schmitt & Yanuck, 1999; Walther, 2000, p. 14) have all been said to promote at least acute weakness of individual muscles. Muscle weakness without atrophy is also thought by some to result from the presence of trigger points in muscles. (Mense, 2008, p. 18) Regardless of the initial cause however, inhibition can become chronic, affecting any voluntary muscle in the body.

Clinical experience shows that weak muscles are invariably found around areas of current pain and injury, areas of past, recovered injury, but also, at times, in areas in which no injury is recalled and no current symptoms are present. Thus, it is probably not an exaggeration to say that muscle inhibition is ubiquitous, like other usual findings of musculoskeletal examinations such as palpatory pain or reduced range of motion.

Officially however, muscle inhibition, which can now define as '*neurologically induced weakness of healthy individual muscles found on muscle tests*', does not exist. '*Muscle inhibition*', '*functional weakness*' (or other '*functional neurologic disorders*' (Stone et al., 2014)) are not present among the 70,000-plus codes in the International Classification of Disease. '*M62.81, Muscle weakness (generalized)*', a vague designation that could refer to a patient's experience or to an examination finding, is the closest we can come to giving a diagnosis of muscle inhibition. This means that it is difficult to epidemiologically study muscle inhibition, link it to other findings, retrospectively determine treatments that might be effective, or know whether a treatment of inhibition leads to better outcomes.

An irreversible condition?

The general belief in the literature is that muscle inhibition is irreversible, as obliquely evidenced by decades of AMI research failing to put forth a single treatment that permanently reverses the condition. (Hart et al., 2014) The literature states that AMI is '*rehabilitation resistant*', (Hart et al., 2010) unresponsive even to '*intensive*' (M. V. Hurley et al., 1994) or '*aggressive*' (McVey et al., 2005) physical therapy and resistance training or exercise. (Hart et al., 2014; Park et al., 2012; Rice & McNair, 2010)

Treatments that are performed with the presence of muscle inhibition - virtually all treatments, that is - are at best helping patients compensate for the missing muscles or deal with symptoms created by inhibition. As stated in the 2010 book *Muscle pain: diagnosis and treatment*:

'*Strengthening the inhibited muscle with an exercise program often reinforces the abnormal motor behavior, and makes it more difficult for the patient to recover normal function.*' Even when outward function is restored, it may come at the cost of locking patients into a lifetime of dysfunctional movement. (Mense & Gerwin, 2010, p. 7) Nonetheless, this practice is the norm in rehabilitation practice.

As such, finding a successful treatment for muscle inhibition should be on the top of the list of goals for musculoskeletal medicine, but research into condition is almost completely off the radar.

Contrarily, however, muscle inhibition has been successfully and lastingly reversed by practitioners of the chiropractic sub-specialty of Applied Kinesiology (AK) since the 1960s. The initial basis of AK was a protocol for reversing inhibition, developed by chiropractor George Goodheart. The protocol involves treating a set of specific reflex points located around the body that are unique to each muscle. Those reflexes, many of which came from systems developed by others, seem to relate to the resources each muscle requires to operate. Points are treated that relate to the flow of blood (neurovascular reflexes), lymph (neurolymphatic reflexes) and (acupuncture) meridian energy to a muscle, and points of interconnection between the muscle and a specific organ (or organs, in a later version of the protocol). The treatment includes neurological activation of the muscle, addressed by adjustment of specific vertebrae, and massaging of the origin, insertion, and spindles of the muscle. Following the treatment, the targeted weak muscle is immediately restored to normal functionality; where before it tested '*weak*' it now tests '*strong*', that is. (Frost, 2013; Gin & Green, 1997; Goodheart, 2005; Walther, 2000)

The efficacy of this canonical protocol was taken for granted perhaps, as AK grew to include other, more controversial procedures. Perhaps because these other procedures have consumed most of the attention in AK, in addition to a lack of research funding, the protocol has never been given the efficacy studies it deserves. (My own training included this protocol, but the experimental treatment we will describe bares no external resemblance to it.)

Beyond muscle inhibition, a second AK treatment paradigm

The mPTSD hypothesis being presented grew out of observations that ensued from my investigation of a new way to treat inhibited muscles that began in about 1993, an early attempt would develop over several decades. The mechanisms of this method are unknown however, meaning that it is not evidence that can be used to substantiate the hypothesis, so we will only offer a superficial description here. Nonetheless, the results I observed from working in this way show what is possible in the treatment of muscle inhibition.

Going beyond its canonical muscle correction protocol, it was discovered by Goodheart that inhibited muscles would temporarily strengthen when touching any of the reflex points of the protocol. For instance, having the patient touch a neurovascular reflex while testing a weak muscle, that muscle will temporarily test strong if that reflex is involved with the weak test.

It was further discovered that touching an aberrant reflex point, or in fact any area of the body that was might be injured or stressed, would weaken a strong muscle, known then as an '*indicator muscle*', as long as the contact was held. In other words, touching that same neurovascular while testing a strong muscle will cause that muscle to weaken. The same is true, in most cases (we will not talk about exceptions here) when bringing the attention of the nervous system to any kind of stress whatsoever.

Upon finding an area that was stressed and caused an indicator muscle to weaken, say an injured area of the body, it becomes possible to prospect for other contacts that then strengthen the indicator muscle. When one is found, it suggests that there is at least a relationship between the initial stressor and the contact that creates the change in muscle strength. If the second

contact was an area of the body that could be directly treated, like an acupuncture point or spinal vertebra, the suggestion is that treating that area will relieve a portion of the stress in the first area.

If the second area was an organ reflex it would indicate that stress in the organ was related to the initial stress, but it doesn't say what that stress is, or what to do about it. ('Organ stress' is rarely an indication of frank disease process, only that the organ is over- or under-activated in the context of the particular pattern of activity that is the current focus.)

Still holding that organ point which strengthened the muscle, further prospecting can be done to discover a treatment that might help the organ. In this way, taking an 'open-ended' approach, it becomes possible to find and address longer causative chains. These might reveal relationships which are known to be theoretically possible. Studies of viscerosomatic and somatovisceral relationships, (Walters, n.d.) psychoneuroendocrinology and psychoneuroimmunology, psychophysiology and behavioural physiology (Ader & Cohen, 1993; Gregurek & Gregurek, 2015; Litchfield, 2006; Rizzi et al., 2020) and so on, show that in effect, anything can cause anything

Where practitioners now generally acknowledge these relationships, and suggest general remedies like massage, yoga, meditation, psychotherapy, or psychotropic drugs, muscle testing arguably allows clinicians to more directly address specific interrelationships between various systems.

One article, presenting two case histories of apparent resolution of hypothyroid conditions using an AK method, is particularly useful for understanding of maladaptive function suggested in the mPTSD hypothesis. Bablis and Pollard (2009) provide a description of the application of a method of AK known as Neuro-Emotional Technique (NET). In these cases, muscle tests are used to to pinpoint particular emotional states that may be associated with patients' hypothyroid, and the past incidents which fostered self-beliefs that engendered these states. In NET, each emotion has been found to be associated with specific vertebral levels, which are treated by adjusting those levels while the patient focuses attention on memories, emotions and beliefs that have been revealed. In these two case histories, a series of such treatments are done, each discovering and addressing different past incidents and their related emotional pattern. Lab tests before and after the series of treatments show normalisation of thyroid function in both patients, along with a reduction in associated symptoms. (Bablis & Pollard, 2009) Dozens of peer-reviewed articles relating to these and other uses of MMT are available in a compendium established by Scott Cuthbert, one of the primary AK researchers. (Cuthbert, 2017)

NET is one of a series of '*energy psychology*' therapies that appear to work on similar principles. In our proof-of-concept experiment, we apply one of these, the better-known Eye Movement Desensitisation and Reprocessing (EMDR), to treat inhibited muscles.

This treatment of a hypothyroid may coincide with principles of allostasis, or predictive regulation of physiology. Past history creates expectations that then are drive physiological activity. When expectations do not comport with reality, they can lead to maladaptive responses. Patients with PTSD, for instance, have a greater incidence of type 2 diabetes. This may occur because PTSD predicts that one might need to fight or flee, both of which require additional glucose to feed muscular activity. Expectation of stress or enhanced physical activity is known to raise blood glucose to meet the expected demand. PTSD patients also show high amounts of inflammatory markers, as if in expectation of needing to repair tissue damage. (Kleckner et al., 2017; Roberts et al., 2015; Sterling, 2004, 2011)

The mPTSD model will suggest that similarly, past experiences in which the use of particular muscles has been associated with negative outcomes later causes the motor control system to predictively avoid relying on those muscles. The difference is that raising blood sugar is proactive preparation for an expected future, muscle inhibition is counteractive or avoidant preparation.

A half generation behind Goodheart, chiropractor Alan Beardall was another muscle testing innovator. In the late 1970s and early 1980s, his Clinical Kinesiology (CK) first expanded the number of muscle tests from the 70 or so specific muscle tests listed in other texts to over 300 bilaterally tested muscles, complete with the reflex treatments for each. (Beardall, 1980) Then, Beardall went on elucidate many of the principles of the aforementioned open-ended testing that allowed for the finding of otherwise hidden relationships between the various systems of the body. (Shane, 2005) He described this as treatment of adaptation, though we are terming it maladaptation here, because any adaptation that outlives its usefulness becomes maladaptive. While Beardall met an untimely demise in a 1987 traffic accident, his work continued to develop through the continued clinical research done by those of us who had the good fortune to study under him. It was a version of this (mal)adaptation-addressing method that I began to apply to muscles in 1993.

Treating muscle weakness as (mal)adaptation

In the context of this investigation, adaptation can be defined as a set of behavioural strategies developed during or following stress or trauma that serve one of two purposes. The first purpose is to avoid pain or other bad outcomes, and the second is to compensate for functional deficits. We will see in subsequent parts of this series that muscle inhibition may be a two step process, containing both kinds of adaptation, but for now we will just look at the results of treating muscle inhibition, to see what is possible.

The treatments 'suggested' by CK-variant I use are often brief and simple, like a single spinal or extremity adjustment, 15 seconds rubbing an acupuncture point or massaging a muscle. Interestingly, it is a rare exception when the treatment works directly on the muscle that we are attempting to target. The treatments are often physiologically and anatomically unrelated, in any obvious way at least, to the muscles that are being addressed. Yet, the changes can be so immediate and sweeping as to be almost unbelievable.

These treatments routinely reverse weakness in many muscles at once, '*bulk erasure of inhibition*', we might say. There seems to be little relationship between the severity of symptoms, the number of muscles that are weak, the chronicity of the problem, and the treatments that are required. The muscles in a chronic case with severe symptoms may require a similar, seemingly trivial or random treatment as an acute, symptomatically mild condition. At other times though, the treatment ends up winding through many layers of adaptation, perhaps touching on habitual emotional postures or patterns of organ and endocrine stress, the nature of which is generally obscure. This treatment alone can result in the immediate and lasting relief or even resolution of both acute and chronic musculoskeletal symptoms, though results must sometimes be rounded out by other treatments based on more common notions. Depending on the history, diagnostic imaging becomes the next option if muscles are restored but symptoms continue.

Though obviously a breakthrough, I had no idea at first how this treatment was working. Given the unproven nature of its methods, the variability of the treatments, and frankly its apparent weirdness, I sought a way to understand and present these findings.

It seemed that the only entity that could change in this way was memory, information stored in the brain that caused muscles to fail to respond to intentions to contract. Thus the hypothesis was formed: muscle inhibition is the result of maladaptive memories, but nothing in the literature indicated that such memory change was even possible. These were motor memories, procedural memories akin to riding a bicycle or swimming which were generally reputed to be indelible. It took science catching up to the observations to begin to formulate the mechanism of what was occurring.

A new understanding of memory allows for unlearning

In 2000, a barely noted theory about the nature of memory that had been around since the 1960s (Misanin et al., 1968) was revitalised and validated. The theory was considered so important that Joe Le Doux, head of the NYU neurological research team and popular author on emotion and the brain, did a series of interviews to give it the recognition it deserved. In old models of learning, memories were considered (relatively) permanent and consolidated only once, at their inception. Le Doux announced, and his team held forth in a new study that instead, when activated (recalled), memories may become destabilised (labile) and therefore subject to change or even effective erasure. (LeDoux, 2007; Nader, 2015) To endure, activated memories must undergo a '*gene-expression-and protein-synthesis-dependent restabilization process*' called reconsolidation. (Radiske et al., 2017) Once recalled and destabilised, memories become subject to being updated or even unlearned, given the proper conditions. (Nader et al., 2000; Przybyslawski & Sara, 1997)

Reconsolidation theory states that activated memories may be updated with new information if categorically similar information is presented within a six-hour window. The recalled memory might be reinforced or strengthened by reconsolidation as well. (Forcato et al., 2013)

Several conditions however, may lead to what amounts to a blockage of reconsolidation, in which the destabilised memory is not stored again; memory erasure, essentially. In early studies, reconsolidation was chemically disturbed by using agents that block the consolidation of new memories as well. *Propanolol*, for instance, which blocks the binding of norepinephrine required for learning, would cause amnesia for an activated memory. The earliest experiments were done on fear-conditioned animals. (Nader et al., 2000; Przybyslawski & Sara, 1997)

For destabilisation of a memory to fully take place, a 'prediction error' must also occur; new information - cognitive, sensory, or sensorimotor - must be presented following the activation, information that is different from that which is predicted by the memory. (Sevenster et al., 2013, 2014) If that new information is similar, like adding new words to an already existing list of words, or, pursuant to motor learning, (Wymbs et al., 2016) perhaps like a new foot placement added to a known dance, the memory can be updated, adding in the new information. If, on the other hand, the new information sufficiently contradicts the old, the existing memory may fail to be reconsolidated; it ceases to exist. (Alberini & LeDoux, 2013; Ecker, 2012; Przybyslawski & Sara, 1997; Sara, 2010; Schiller et al., 2010; Sevenster et al., 2014) Reconsolidation has been demonstrated to occur in practically every form of memory that has been studied, including motor memories. (Nader & Hardt, 2009; Wymbs et al., 2016)

Reconsolidation and the erasure of (maladaptive) memories

If muscle inhibition were a learned phenomenon, a logical speculation would be that testing of an inhibited muscle would activate the memory that is sustaining that inhibition. That memory, according to the reconsolidation model, would then be destabilised. With the memory destabilised, the application of some agent that could block reconsolidation would cause the memory to be lost. Normal strength would then be restored.

The treatments that I was doing, with their variability and unknown effects, were not suited to gaining more understanding about memory's effects on muscles. Another therapy seemed to fit the bill, albeit coming from an odd source for a musculoskeletal treatment - psychotherapy. Several psychological therapies, part of a group of therapies known as 'energy psychology' have since been postulated to operate on the same principle, the blockage of reconsolidation. (Feinstein, 2012; Welling, 2012)

The best known and studied of these is EMDR. The core therapy of EMDR is saccadic (side-to-side) eye movements performed simultaneously with thinking about an emotionally charged

stressor or memory. It enjoys the widest acceptance and has the largest research base of any of the energy therapies, group of therapies that apply physiological activations like this. Rated as 'probably efficacious in the treatment of civilian post-traumatic stress disorder' almost two decades ago by the American Psychological Association, EMDR often has the effect of immediately reducing the intensity level of psychological pain associated with (maladaptive) memories. (Carlson et al., 1998; Chambless et al., 1998; Feinstein, 2012).

The central therapeutic application of EMDR first has the patient focus on a memory or current discordant emotion, thought, or feeling state. With that memory or current state in focus, the patient is instructed (in classical use of EMDR) to follow the therapist's fingers right and left at about 1 cycle per second. This procedure will often create an immediate and significant alterations in the emotional state associated with the memory, generally reducing or eliminating the emotional charge. (Korn, 2009; Shapiro, 1995; Shapiro & Forrest, 2016)

The use of side-to-side eye movements as a therapeutic device has a history that goes back several hundred years. An interesting account presented by Hartmann (2006) describes how a version of the method was used by Sigmund Freud in his early explorations until associations with mind control stemming from a popular novel about the evil hypnotist Svengali forced him to abandon it. (Hartmann, 2006) But it goes back even further, with many parallels to Mesmerism, a therapy that spread rapidly throughout Europe and America in the 18th century. (McNally, 1999) The method was 'rediscovered' in 1987 by psychologist Francine Shapiro who incorporated it into the more extensive psychotherapeutic procedure she named EMDR. (M. Grant, 2000) It was only after the research on reconsolidation became known that some began to speculate that the eye movements might be blocking reconsolidation, though this still has not been conclusively established. (Solomon & Shapiro, 2008)

Various forms of bilateral stimulation, visual, auditory or tactile, have been found to be effective at facilitating this erasure. (Chiamulera et al., 2014) All use the same general method, evoking a memory while providing some kind of sensory stimulation; eye movements are the exception with added motor activity. Similar findings have also come from therapies that tap specific acupuncture points (Church et al., 2009; Feinstein, 2008, 2010; Wells et al., 2003) and NET, which treats a particular set of spinal segments that relate to each organ-emotion, combination, associations derived from traditional Chinese medicine. (Bablis & Pollard, 2009; Rossi & Caretto, 2007) These methods all were developed for and primarily used on emotional issues, but evidence shows that they are effective at addressing pain, (Bablis et al., 2008; M. Grant, 2000; Mark Grant & Threlfo, 2002; Mazzola et al., 2009; Schneider et al., 2007) and even physiological disorders, as the earlier case histories of NET showed. (Bablis et al., 2006; Bablis & Pollard, 2009; Callahan & Callahan, 2011)

If EMDR could have a similar effect to the treatments I was doing, it would support the hypothesis that muscle inhibition is due to maladaptive memories. This is the first experiment that I know of that has attempted to document direct functional muscular changes from these kinds of treatments.

Proof-of-concept experiment

For this experiment, we extracted just the eye movements from EMDR's multi-step psychotherapeutic process. The experiment was performed by four experienced practitioners (including myself) who used MMT extensively in their practices. Subjects were in good general health and not suffering from acute musculoskeletal problems that may have led them to seek care, though some had low grade ongoing issues of the kind that many take for granted. The weak muscles treated were not necessarily in these symptomatic areas, and symptom changes were not closely tracked. Subjects were neurologically sound males and females, ranging in age from their

late 20s to late 60s. Except for one subject, all study participants signed informed consent documents. The number of muscles per subject ranged from 8 to 29.

No specific instructions were given for doing the muscle testing; examiners used their own usual method for determining the status of muscles. However, all were AK practitioners who assess muscles using sub-maximal break (SMB) testing. In an SMB test, after instructing and demonstrating the general mechanics and purpose of the muscle test, the subject is placed in the proper position to isolate a particular muscle and instructed to hold the limb in place. Stating something like 'resist' or '*hold*', the examiner ramps up force against the limb in the appropriate vector to require contraction of the muscle being targeted. With that position and vector, the muscle is (relatively) isolated from synergists (agonists), though of course stabilising muscles must also contract.

Particularly in the shoulder, where the whole complex must often be stabilised by other muscles to test a single muscle, the weakness of a muscle test could reflect the weakness of a stabiliser. Thus, the failure to correct a muscle could be the result of other stabilisers not resolving. The force is ramped up by the examiner until stable, robust resistance is perceived (making it sub-maximal) or the muscle gives way. The test is isometric, with the force of the examiner and subject equally matched such that in a normally facilitated or '*strong*' test, the limb remains stationary throughout. Performed on neurologically healthy individuals, the SMB test has been demonstrated to produce a binary result; either the muscle is found to lock in place from the outset of the test or it does not, beginning to give way immediately upon testing. (Caruso & Leisman, 2000, 2001; Conable & Rosner, 2011) (We analyse the use of muscle testing to determine inhibition in Part III of this series.)

The practitioners treated 8 subjects with a total of 136 inhibited muscles. Aside from suggesting that patients be free from neurological or muscular diseases, no other criteria were established regarding selection of subjects.

The muscles were retested a minimum of 7 days, and a maximum of 42 days following the initial treatment (an average of 15.2 days). The duration was left to the discretion of the practitioner and subject, with the caveat being that the retest needed to occur at least 24 hours after the initial encounter.

The study directions instructed the practitioners to first identify and record, in the random order found, a list of weak (unlocked) muscles from one subject. There was no suggestion given about which muscles to test or which order to test them in. The number of muscles tested in each subject was left to the practitioner's discretion. Only muscles testing '*weak*' were recorded on the test form, not those testing 'strong'.

The test form consisted of nine columns to be filled in for each muscle:

		Pre-test	Post Test			Retest		
Date	Subject F. Name	Name of weak muscle	Strong	Weak	Spontaneous recovery	Date	Strong	Weak

Following the compilation of the list of weak muscles for each patient, beginning again from the top of the list, the same muscles were tested a second time, this time immediately followed by 15 repetitions of the side-to-side eye movements, instructed to be completed in about 15 seconds.

Immediately following the treatment, the (previously weak) muscle is tested for a third time, to see if its state of facilitation had changed. The appropriate result, '*strong*' or '*weak*', is circled on the form. The practitioner then moves down to the next muscle on the list and so on. After at least 24 hours the muscles on the list were tested a fourth time, to see if their state remained stable.

An additional column was added to the test form, to record '*spontaneous recovery*'. It was found that, after treating one or several muscles with the above procedure, some muscles strengthened without any (additional) treatment, suggesting that the treatment of muscles that came before had the effect of strengthening multiple muscles.

Results

The results (Table 1) are simple, binary outcomes of the test and retest. Either the '*weak*' muscles are reactivated immediately following treatment, or not. Reactivated muscles may then either remain '*strong*' or not after a delay of at least 24 hours. An additional designation was added, referring to muscles that strengthen 'spontaneously', without the need for direct treatment, that is.

The results showed that 91.18 (124 out of the 136) of weak muscles were strengthened by the eye-movements.. Of the 124 muscles that were reactivated, 70 or 51.47%, strengthened '*spontaneously*' without treatment. Without treatment here means that they were strong when they were reached on the list.

Retesting the previously weak muscles at least 7 days, but up to 42 days later, an average of 15 days, 20 of the strengthened muscles did not hold their correction, meaning 83.87% of muscles strengthened by the eye movement procedure achieved lasting correction. Thus, at the end of the study, 104 out of 136 muscles (76.47%) were reactivated and retained their strength over those time frames.

Table 1: Treated muscles

Treated muscles									
Practitioner/patient	Total # of weak muscles	Strong post-treatment	Weak post-treatment	Spontaneous recovery	% of muscles spontaneus recovery	Total % of muscles corrected	Days to retest	# muscles weak on retest	% of muscles sustaining correction
1/1	22	11	1	10	45.45%	95.45%	42	1	95.24%
2/1	24	10	3	11	45.83%	87.50%	7	1	95.24%
3/1	29	9	4	16	55.17%	86.21%	7	7	72.00%
2/2	16	6	2	8	50.00%	87.50%	7	2	85.71%
4/1	8	5	0	3	37.50%	100.00%	7	0	100.00%
2/3	12	6	0	6	50.00%	100.00%	21	1	91.67%
2/4	12	4	1	7	58.33%	91.67%	8	3	72.73%
2/5	13	3	1	9	69.23%	92.31%	22	5	58.33%
Cumulative	136	54	12	70	**51.47%**	**91.18%**	15.13	20	**83.87%**

A separate group of 42 muscles over 3 subjects was tested and not treated for similar time frames. Of those 42 muscles, 37, 88.1% remained strong an average of 15 days later. (Table 2).

Table 2: Untreated muscles

Untreated muscles	# of weak muscles	Untreated days	# of weak muscles post-wait	% of muscles staying weak
2/1	14	8	12	
2/2	14	20	12	
2/3	14	19	13	
TOTAL	42	15.67	37	88.10%

One practitioner, recruited online, stated zero changes from his attempts to use the eye-movement method. Given that his technique was not directly monitored, his results were hugely at odds with the rest of the practitioners, and no paperwork was received back, those results were not included in the tabulations. Another practitioner, an experienced muscle tester who used the AK muscle inhibition protocol routinely in her practice, was told about the eye-movement method in a personal conversation. When she was injured a short time later making it difficult for her to use the AK protocol, she used the eye-movements instead. While she didn't participate in the study, she reported that using the non-strenuous saccadic eye movements allowed her to continue to practice successfully throughout her recovery.

A case history of using eye-movements as the sole treatment

Before doing the above experiment, I treated a volunteer with the understanding that I would use only eye-movements to address her case. She had lingering effects following a skiing accident about 15 years prior in after which she had the anterior cruciate ligament, the medial collateral ligament, and medial meniscus all reconstructed. The surgery and post-surgery rehab was successful, but she stated that she felt some stiffness and occasional pain in the knee.

I tested the muscles of the knee and ipsilateral ankle and had her follow my fingers with her eyes immediately after each weak muscle that I found. All strengthened immediately. A month later I re-examined her. She reported that her knee was improved, except for one twisting movement she did while dancing that still caused pain. She demonstrated the movement.

She also reported that over the month, she occasionally had flashbacks, recalling seeing her knee bent at an impossible angle, as it was after the injury. I retested all the muscles of the knee and ankle and found that the ones which were weak the previous month were now strong. Several muscles that had been strong the previous month were now weak, however. Instead of doing the eye-movements right after testing those muscles, I used the EMDR for its original purpose, clearing emotional memories. I had her recall the image of her mangled knee while doing the eye-movements.

I retested the weak muscles I had just found, and all were now strong. Further, she was immediately able to do the twisting movement without pain. I didn't retest her muscles again, but she reported that her knee was asymptomatic and it remained that way indefinitely.

Reconsolidation window

In informal experiments, waiting an increasing number of seconds after testing muscles before performing the eye movements, I found that there was a 7-10 second window in which the eye-movements would be effective. If I waited 10 seconds or more, before commencing the eye movements, the muscles would not be corrected. This conflicts with the research finding that activated memories remain unstable for 6 hours after being recalled.

It also conflicts with what I have also found, utilising the open-ended treatment described earlier. That is, the more muscles I test and weak muscles I discover, to a point, the the easier it seems to be to bulk erase all of the weakness, even if it goes well beyond the 10 second limit.

Discussion

A mechanism for muscle inhibition

Untreated, muscle inhibition, the weakness of individually tested muscles, is arguably a life-long deficit that plays a role in conditions that can be debilitating. Though this ubiquitous condition has been considered reversible by practitioners of Applied Kinesiology since the 1960s, the musculoskeletal community as a whole has no solution for it, where it is not completely ignored that is. The results of this experiment, with 90% of inhibited muscles being corrected immediately, and 80% remaining corrected over weeks, and over 50% being corrected without

being directly treated, all from from a 15 second treatment that did not touch the body, begs many questions. The fact that the treatment may erase memories by blocking reconsolidation is a clue as to the mechanism of correction, but also to the mechanism of inhibition itself.

To make its decisions, the nervous system takes information gained in the past - knowledge - and creatively applies it to achieve a purpose that it wants to manifest in the near or distant future. The problem with knowledge gained in the past is that, even if it was correct for that past time, it may not be correct now. This is particularly true of knowledge gained from pain, stress, or trauma. When the stress ends, the responses designed to adapt to its particular conditions may remain, creating maladaptation, often automatic behaviours that do not suit current needs.

The brain and nervous system are at the mercy of information, dedicated to serving its implicit and explicit edicts, in the context of deeper biological drives. Neuroplasticity is the structuralisation of learning, the accumulation of new information and development of the capacity to act on it. Thus, the brain is literally shaped by the information it holds.

In physical medicine, information is taken to be something that is added to. Repeated practice will inculcate new motor patterns that will eventually become the new normal in terms of behavioural expression, but no concept of the removal of information has existed. Here, we have essentially introduced a whole new concept for musculoskeletal practice: that outdated and therefore maladaptive information that distorts motor processes can be removed from the brain with some measure of precision. This is possible even if we have no idea what that information is, and probably can't know, because it exists in a code that probably can't be translated into cognitive understanding.

It is well accepted that in the motor control system (MCS), upcoming plans for movement are edited before being finally passed on to the motor cortex, which may or may not send 'commands' to the spine for possible expression. The cerebellum, which recalls the outcome of past motor experiences in order to predict the outcome of planned movements, is thought to be responsible for that editing. (Angel, 1976; Clark, 2015; Ito, 2004; Pynn & DeSouza, 2013)

A possible mechanism emerges in which the cerebellum, which holds multiple somatotopic maps of the body (Nitschke et al., 1996; Grodd et al., 2001) and is known to be an error predictor, (Popa et al., 2016; Schlerf et al., 2012) has learned from previous injuries that muscles, like the peroneus muscles in an ankle inversion injury, may fail or cause pain when used. Though the prediction may have been established from a single anomalous incident, it becomes the template for all future movements using those muscles.

In other words, the cerebellum may predict pain or failure when faced with motor plans that will employ muscles that have been associated with past failure. Those muscles, the mPTSD model posits, will then be edited out of motor or muscle synergies, combinations of co-activated muscles that habitually act together with their own varying order, force and timing. (Cheung et al., 2020; Latash & Anson, 2006; Muceli et al., 2014)

Synergies are thought to be hierarchically established early in life by trial-and-error in the cerebellum and motor cortex, an interaction which led one set of researchers to state in the title of their paper that '*The Motor Cortex Retains What the Cerebellum Learns*'. (Galea et al., 2011; Rispal-Padel, 1993; Thach et al., 1993) The motor cortex then '*tutors*' sub-cortical and spinal circuits to form a basic set of synergies. The hierarchy ends in spinal alpha motorneurons (αMNs), that are activated by spinal interneurons. These interneurons are themselves a computational system that takes the commands from the motor cortex and further integrates them into the current sensory schema of body's current position and relationship to its environment, though there is sensorimotor integration occurring at each level of behavioural formulation in the motor system. (Cheung et al., 2020; Graziano, 2009)

These basic synergies are tuned throughout life and recombined to learn new skills. Ballet dancers, for instance, tune synergies used for walking, transforming them into the skills needed for the difficult tasks they learn to accomplish. (Cheung et al., 2020) Muscle inhibition throws a monkey wrench into the complex mix of muscle synergies.

A given muscle may play a role in many synergies, acting as a prime mover through parts of some. With use of a prime mover eliminated, the entire synergy may need to be avoided, as the MCS silently rehearses its expression in advance of actual movement and predicts that it will result in pain or failure. The fact that muscles are often inhibited without atrophy (Mense & Gerwin, 2010, p. 18) supports a model in which those muscles are still used as secondary support. If they were completely unused they would rapidly atrophy.

In the experience of practitioners who rely on muscle testing, most inhibited muscles test as a 4 (on the 0 through 5 scale). Though they break away immediately, measurable resistance is still occurring. (Caruso & Leisman, 2000; Conable & Rosner, 2011) Further though, the clinical experience in which patients unconsciously avoid positions that would rely on those muscles is more proof of prior knowledge of inhibition in the MCS. The muscle test then forces the patient to try to use that muscle as a prime mover.

Explaining the results of immediate treatment following injury

Whatever mechanisms initially led to the inhibition of a muscle - the arthrogenic reflex, pain, emotional or organ stress - the resulting avoidance is logically only the first stage muscle inhibition. In the second stage, new muscle synergies must be created which substitute other muscles for the ones that are now considered unreliable.

When an injury first occurs, those replacement synergies have not yet been developed. As a result we can speculate, the MCS (or other areas of the brain) interpret the avoided muscles and the synergies in which they participate as being injured, with all the attendant pain and inflammatory process that would accompany an actual physical injury to those muscles. Rizzi et al. (2020) posit that, since the cerebellum and hypothalamus are bi-directionally connected '*the hypothalamus represents the link through which the immune functions may influence the psychic functions and vice versa, the cerebellum, controlling several regions of the hypothalamus, could be considered as a primary player in the regulation of the multiple functional interactions postulated by psychoneuroendocrinoimmunology.*'

The perception of trauma may then be mediated by the cerebellum's 'conclusion' that certain muscles, and the synergies in which they operate, are dysfunctional because they are associated with trauma. It may send that information to the hypothalamus, which does the job it is designed to do: mobilise the immune system to take care injury, real or in this case inferred from circumstances. (Rizzi et al., 2020) According to Schmahmann (2004), '*the healthy cerebellum is involved in the generation of emotionally congruent autonomic reactions.*' (Schmahmann, 2004)

When muscles are not re-facilitated, a vicious cycle may be established, in which the prediction of pain and failure lead to actual pain and swelling, which, in turn reinforces the prediction of pain. When muscle inhibition is resolved right after an injury however, as it was by Buhler in his aforementioned treatment of NBA players, the brain's mistaken belief that the muscles are injured may be erased. Pain is immediately significantly reduced or eliminated and swelling doesn't take place.

AK protocol and trauma learning

If this is in fact the mechanism underlying muscle inhibition, the AK protocols, which also offer ongoing resolution of muscle inhibition, must, it seems, also be erasing the memories that have resulted in chronic muscle weakness. (This includes both the basic AK protocol and AMIT®, the

advanced version of the protocol perfected by Buhler working with NBA players.) There is no obvious answer as to how this might be occurring, but we can speculate several possibilities.

In our proof-of-concept experiment, we first tested the muscle and then did a procedure that assumedly altered neurological state of the body. We might analogise this to a process in which opening a file on a computer brings its information into RAM and shows it onscreen, while removing it from the ROM, the drive on which it had been permanently stored. If the computer is rebooted before the information is re-saved on the drive, the information will be permanently eliminated.

I and others have observed that, using the AMIT® protocol, if one reflex is insufficiently addressed, the correction may not take place. Several times I needed to go back to re-treat a reflex that was insufficiently treated. Buhler's experience, as he related in the AMIT® certification course I attended, is that unless all the reflexes are addressed, the muscles may not hold their correction. The reflex treatments, taking about 10 minutes to complete however, exceed the reconsolidation window that we found for applying the eye-movements, though it is well within the generally quoted window of 6 hours. The reconsolidation window is the time that a memory remains destabilised after it is activated.

It is logical that, when the brain has 'decided' that a muscle is going to be omitted as a prime mover, the resources reserved for that muscle, blood, lymph and energy, will be re-routed to other areas of the body. One interpretation of the success of the protocol is that the treatment is neurologically reconnecting those resources to the involved muscle.

But erasure, according to reconsolidation theory, requires re-activation of the memory. If that isn't accomplished through the muscle test because it is outside of the reconsolidation window, it may be achieved through work on spindles, origin and insertion of the muscle. This may activate stretch reflexes of the spindle and Golgi tendon organs, which could be expected to send a signal to the cerebellum (Pruszynski & Scott, 2012) that may activate and thereby destabilise the memory of stress or trauma. When the reflexes purportedly controlling resources to the muscle are also activated, the neurological state may be changed sufficiently to prevent reconsolidation of the memory.

Some AK practitioners, however, report successfully utilising an early method of Goodheart's alone to reactivate muscles. (Gin & Green, 1997) In the 'origin insertion treatment', the tendon ends of the muscle are firmly massaged, with the assumed outcome being correction of micro avulsion of the periosteal attachments of the tendons that occurred during the initial injury, (McDowall, 2004) or perhaps other micro tears in the muscle. (Clarkson & Hubal, 2002) Buhler however, working with the same athletes on the Utah Jazz day in and day out, believes that the entire protocol is required for the muscles to hold their corrections. More study is needed to understand these contradictions.

Conclusion

Larger implications of the mPTSD model

Muscle inhibition, the weakness of individual muscles clinically revealed through MMT, is a ubiquitous and ubiquitously ignored problem that is probably an unseen cause or aggravator of all manner of myofascial and joint problems. Where it is examined in most studies, it is considered to be irreversible, though it has been successfully treated by practitioners of Applied Kinesiology (AK) for over five decades.

This paper has introduced and supported with experimental and a case history evidence a hypothesis that muscle inhibition is sustained by maladaptive learning. This learning may be acquired from stress and trauma, including joint injury, pain, psychological, and biochemical or organ stress, thus earning the name muscular post traumatic stress disorder, or mPTSD. This is

reinforced by the fact that EMDR, from which we borrowed the eye-movement therapy, is an accepted treatment for PTSD.

Overall, the paradigm of recovery presented here could be called 'informational healing', the erasure of memories that engender maladaptive function. It expands on the understandings of EMDR, NET and other energy psychology treatments, which mostly focus on consciously available memories, beliefs and emotions as the information that is driving maladaptive function.

The informational healing paradigm might apply to any maladaptive physiological response, not just muscular maladaptations. Apparently, what is required is first, devising a method of getting the body to bring forth the memory which is invoking the maladaptive response, thereby destabilising it, and then second using one of the so-called energy psychology treatments to change the state of the body sufficiently to prevent reconsolidation of that memory. Though the NET method used by Bablis and Pollard (2009) in the aforementioned treatment of hypothyroid assumed that beliefs and emotional factors needed to be consciously brought forth, it is possible that the brain's cognitively indecipherable programming might also be accessed and cleared in the same way.

All physiology is behaviour, (Litchfield, 2006) a reaction 'chosen' based on previously-developed algorithms to respond to inferred (logically-imagined) meanings about the current and predicted demands of the environment in the context of the current psycho-social and physiological state of the organism which is also inferred from its previous experiences. So there are actually at least three memory patterns that can be accessed. The first is the memory underlying the inferred meaning of an event, the second is the memory relating to the current perceived state of the body and mind as it relates to that meaning, and the third is the memory of the response that is brought forth like a template, from which a response appropriate to the current situation is created. To the extent that those factors can be activated in any moment, they may be subject to being eliminated by the blockage of reconsolidation, even without exact knowledge of their nature on the part of either the practitioner or the patient.

Herein, we putatively brought forth and erased memories containing beliefs that are not consciously knowable, or at least not available in the moment. MMT is subjective, so in future experiments, changes could be measured objectively with instrumentation. When tested in this way, muscles are essentially binary in their responses, providing immediate and clear feedback of success or failure of the treatment. (Caruso & Leisman, 2000)

The experimental treatment we applied is simple and assumedly safe enough to be easily adopted by experienced musculoskeletal practitioners of all stripe. At the very least, it could be clinically evaluated by practitioners with relative ease. While I do not find it to be quite as effective as the method that I have used for over 25 years, its rate of 80-90% of muscles rescued from inhibition is still significant, and this more limited technique could well be improved upon with clinical creativity.

Because muscle inhibition isn't even officially a condition, it has not been factored into most research on musculoskeletal conditions, and its treatment has been ignored; insurance will not cover treatment for a condition that does not exist either. This means that based on current literature at least, we have to guess at the benefits of treating muscle weakness.

The resolution of muscle inhibition can be a diagnostic aid as well. Whether the injury is acute or chronic, if correcting muscle inhibition using this simple method takes away symptoms, it means that those symptoms were related to the above mechanism. If it doesn't, other factors may be at play, including bony or soft tissue damage, infection, tumor, circulatory deficits, and so on.

The ubiquity of muscle inhibition means that the various forms it takes with each individual are never taken into account in investigations of musculoskeletal problems. This means that a

solution to muscle inhibition, routinely-applied, might necessitate a re-evaluation of much of what we have believed about the course and treatment of musculoskeletal problems. Overall, the treatment of muscle inhibition will be like what is called in technology a 'disruptive innovation'.

In coming parts of this series, we will look more deeply into this new understanding of muscle inhibition. In Part II we show the potential benefits of treating muscle inhibition, based on its 20-plus year application in the NBA.

In Part III, we examine muscle testing when done for the purposes of discovering muscle inhibition. We will conclude that the type of testing outlined for the above experiment may be superior even to 'objective' tests that are now applied in studies for determining whether a muscle is inhibited or not.

In Part IV of this series, we will go deeper into the pathophysiology of mPTSD that was generally laid out above. We will present evidence that it is result of associative learning in which use of particular muscles, (or movement in particular vectors) has become associated with stress, trauma, pain or failure, a kind of classical or Pavlovian conditioning. The development of new muscle substitution patterns is then operant or instrumental conditioning. We will show additional evidence to support the conclusion that muscle inhibition is governed by the cerebellum.

These are obviously radical conclusions, challenging and upending the very basis of practices that have predominated physical medicine probably since its inception. If these suppositions hold up, we can understand rehabilitation, as currently practiced, as being largely a process of developing new muscle synergies or substitution patterns, made necessary by muscle inhibition. We therefore propose that treatment of muscle inhibition should be the first procedure done when patients arrive in pain, after ruling out more serious problems. Such a practice will save huge amounts of time, money, pain and suffering.

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Cite: Weissfeld R. Mind, trauma & muscle inhibition Part I: Experiment and case history yield novel theory of muscular PTSD [Hypothesis]. Asia-Pac Chiropr J. 2021;1.3. URL www.apcj.net/weissfeld-hypothesis-mptsd-part-1/

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