Chronic muscle imbalance = unstable joints = injuries & pain: A neurobehavioral model leads to new treatment options.

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When the logical surgical and/or physical medicine procedures have failed to completely relieve musculoskeletal disorders (MSD) and no obvious reason exists, it can be a frustrating situation for physician and patient. Recent understandings, however, suggest that a novel therapeutic principle and method may provide an answer for the riddle of why some patients fail or incompletely respond to appropriate treatment.

Surprising clinical research shows that basing treatment on principles of therapy for post-traumatic stress disorder (PTSD) reduces recovery time for acute injuries or surgery. It also suggests a way to evaluate and treat for subclinical deficits that may predispose athletes to injuries while also addressing non-recovery that may otherwise go unexplained. It is applicable to both athletes and non-athletes.

This therapeutic principle has its roots in a treatment, Clinical Kinesiology, developed in the 1980's.[1] The NBA's Utah Jazz throughout the 1990's, were known as the most injury-free team in the league, when a student of this method was one of the team physicians. For over a decade, John Stockton, Karl Malone and the rest of the team missed few games due to injury. Over 18 years of regular-season games, Malone played in an amazing 1,434 of a possible 1,444 games,[2] and Stockton in 1,409 of a possible 1,431 games[3] and both played into their 40's, a feat accomplished by only nine other players in history.

Coincidence? Let's review the four prevailing physical medicine approaches, and then we will reveal a fifth, newer principle and some current neurophysiological research background that may explain those results.

The four general varieties of ortho/rehab treatment are.

1. Therapy directly to the joints or muscles *where the pain or dysfunction is located*. This includes hands-on myofascial work, ultrasound, electrical stimulation, laser, heat, cold, external applications of various healing or anti-inflammatory creams, stretching, acupuncture, joint manipulation, trigger point and other injections and rehabilitation exercises.
2. Treatment to *physiologically related areas*, such as nerve blocks, treatment of ankle instability for knee pain, acupuncture to distant area of the body that is known to influence the region of the problem, or spinal manipulation to the area of the spine from which the nerves to the involved area arise.
3. Foundational or postural *supports*, such as arch supports, braces or wraps of the effected areas.
4. Proprioceptive or postural rehabilitative training, including balance boards, Swiss ball and other exercise based treatments that work with balance and coordination and neuromuscular integration.

The neurobehavioral injury

Before we look at this new variety of treatment, we must first understand some principles of what is considered a psychological or neurobehavioral disorder, post-traumatic stress disorder, (PTSD) and the relationship of PTSD to motor planning and execution. PTSD is actually a disorder of the contents of memory, wherein memories of trauma, and adaptations made both to the trauma and to sequela to the trauma, are firmly encoded in memory due to, among other things, increased arousal via norepinephrine (NE)[4] that accompanies trauma. Unlike other memories that will include the beginning, middle and end of the experience, and will confer a sense of completion, trauma memories will hold the unresolved activation of the trauma, including feelings, emotions and, particularly for this discussion, somatic and motor patterns of the trauma. These uncompleted behaviors are then carried out to the future, and act as conditioned triggers for behavioral strategies, including motor strategies, that continue to respond as if the trauma was awaiting completion.[5],[6]

While PTSD is generally considered to arise from threats to physical or emotional survival, a similar but more restricted phenomenon seems to result from more 'mundane' physical injuries like typical athletic injuries, such as strains, sprains, tears and fractures. In this *musculoskeletal PTSD*, (mPTSD), not only will the initial stage of injury increase memory consolidation via pain-stimulated increase in NE, causing the failed motor pattern to be encoded to memory, but subsequent antalgic postures and compensatory movement strategies will also essentially be practiced (and perfected) and thus also added to the traumatic learning.[7],[8]

Based on a *predictive equation* which calibrates all that is known about the internal and external environment, including present sensory information, results of similar previous motor activity, and learned expectations of the environment, anticipated action is simulated with muscles inhibited to prevent movement.

This *predictive equation* factors in all aspects of mPTSD including past adaptations, compensations and memories of pain or tissue failure into the decision process. It chooses not only which movement strategies will be enacted, but also the choice of which muscle activations or synergies it can use to accomplish the upcoming movement. The integrated activity of a number of brain areas (including basal ganglia, cerebellum and motor cortex, brainstem, and extrapyramidal areas that control primitive postural reflexes[9]) plan and silently execute each movement before it is actually made based on the results of past movement.[10] The inclusion of mPTSD into the predictive equation is like adding a virus into a cell, altering the behavior of the cell.

Therefore, even after the trauma has passed, and tissues have

had time, conservative treatment (or surgery) to effect repair, the learned motor behaviors will continue to manifest in the musculoskeletal system as altered function. This altered function is a part of the myriad factors that equate into the complex orchestration of motor and postural strategies.[11]

Thus, mPTSD causes musculoskeletal dysfunction (MSD). The MSD may manifest as continuing symptoms and dysfunction in the injured area, in other areas that were altered as part of whole-body postural adaptive strategies, or as subclinical dysfunctions in muscle activation and balance that nonetheless may take more energy to enact or are engaged at the expense of motor control thereby increase risk of future injury or joint wear because of poor joint balance.[12]

Can we erase trauma memories?

Assuming that adhesions and other structural issues are also addressed, we are left with a paradox of sorts; how do we treat an injury that is now but a ghost, an ongoing memory of injury and the adaptations to injury that is no longer there? To understand the answer we need to look at the mechanics of memory and the results of a pilot study.

Until recently, memory was considered indelible. The only way to change memory was thought to be an additive process of consolidation (formation) of a new memory that is different than the previous memory, a process known as extinction.[13],[14] Many therapies work with extinction. Repetitive exercises to strengthen inhibited muscles or train more appropriate activation patterns, repeated joint manipulations and proprioceptive exercises may all be examples of therapeutic approaches that train the body into reasonable or better alternatives to traumatically acquired adaptation through extinction. But extinction does not remove the adaptation, it leaves the risk of its re-emergence. A thorough muscle activation evaluation can detect extinction as a state of abnormal facilitation of muscles throughout the body, with some over-facilitated (unable to weaken to appropriate stimuli), and some inhibited.

Around the year 2000 a new theory about the permanence of memory was beginning to seep into the literature, shaking established assumptions. Further research shows that, once activated, conditioned fear memories in the amygdala,[15] (the center of PTSD memory) and hippocampus and motor memories in the cerebellum[16] (likely the primary area for post-traumatic motor conditioning) may become unfixed from storage and thus subject to being lost if they are not reconsolidated. (Consolidation is the formation of memory, so reconsolidation means to store again.) This means that, in fact memory is not indelible, it just appears that way, because it generally seems to re-store itself as much the same memory that was present previous to activation. It is likely that norepinephrine (NE) production will be triggered along with the activation of traumatic memory, fostering reconsolidation of the memory. Blocking NE production or reception is known to decrease both memory consolidation and reconsolidation and has been suggested as a treatment for PTSD.

Pilot study of mPTSD

To test the hypothesis, a pilot study to see whether side-to-side eye movements used in Eye Movement Desensitization and Reprocessing (EMDR) which may have its effect via reduction of NE,[17] would strengthen inhibited muscles. Rated as "probably efficacious in the treatment of civilian post-traumatic stress disorder" by the American Psychological Association, EMDR is the most studied of a group of similar treatments in which fairly trivial sensory input has the effect of immediately reducing the intensity level of psychological pain,[18],[19] but only when applied simultaneously or immediately following patient focus on the pain.

In the study, the side-to-side eye movements, applied for about 20 seconds, strengthened all muscles when applied immediately after challenging (testing) the muscle. One of the subjects was followed for several months afterwards:

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 (routinely tested and corrected along with the knee in such cases) 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. 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 (an expected EMDR result when applied to the image). 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.

Defining a new mechanism

Had it been a chicken bone instead of a finger that the subjects had been following with their eyes, the claim of resolving a decades old knee dysfunction in 5 minutes by waving a finger in front of a subject's face would only seem a bit more preposterous. But from our earlier discussion, we may now ponder with some understanding the actual neurophysiology that resulted in such immediate symptomatic and behavioral change. As in working with the psychological aftereffects of PTSD, wherein activation of a stressful thought or feeling, simultaneous with or immediately followed by the eye movements will diminish the affective intensity of a thought or feeling, the study suggested that challenging (contracting) inhibited (weak) muscles, followed by the same eye movements will re-facilitate (strengthen) those muscles.

In the case of SM, above, the re-emergence of the image of the mangled knee supports the conclusion that the muscle inhibition was part of the post-traumatic adaptation to the earlier injury.

More often however, the incitement of emotion or images from the challenge of a muscle is not externally observable and remains unconscious. Yet it is known that motor activity can also trigger the memory of previous contexts in which the movement was undertaken.[20] Challenging a weak muscle therefore will stimulate the context, however unconsciously, of associated traumas, and this arousal in turn may increase NE levels, inciting reconsolidation of the memory of the trauma in (among other areas) the amygdala, and its associated adaptive motor pattern likely in the cerebellum.

The eye movements of EMDR, among other effects, may decrease NE levels,[21],[22] thereby breaking the automatic loop of reconsolidation following aberrant muscle activation, effectively erasing the adaptive muscle inhibition.

When the trauma-induced adaptive muscle patterns are no longer elicited in activity, the latent pre-trauma movement and postural strategies may resume control.[23] Thus, it is possible to immediately restore function without any local therapies or specific rehabilitative input.

No therapy rendered

Trauma activation (muscle challenge)

⇨Memory rendered labile

⇨ Norepinephrine increase

⇨Reconsolidation of trauma memory

Therapy rendered

Trauma activation (muscle challenge)

⇨Memory rendered labile

*⇨Therapy rendered, **NO** NE increase*

*⇨**NO** reconsolidation, aberrant muscle activation pattern not reinstated*

Making rehab more efficient

As an analogy, elimination of mPTSD can be compared to rebooting a computer to clear its memory of programs that are no longer running but have left traces in the memory that are now in conflict with currently running programs. Because mPTSD alters *functional control* of body-wide muscular and postural mechanisms, appropriate therapies may sometimes be less effective than hoped if mPTSD is not first addressed. In fact, in the absence of underlying joint damage, the failure of appropriate treatments actually increase the odds that addressing mPTSD will help to put them on track. Local therapies, strength or proprioceptive training will be made more effective following treatment of mPTSD. Reducing fibrosis, increasing range of motion, or correction of joint subluxation will also be more effective when the body is not locked into in a persistent centrally mediated adaptation that alters joint mechanics.

Signs and assessment of mPTSD

Whether classified as muscle strain, sprain, tendonitis, bursitis or capsulitis, mPTSD, to the extent that it exists, will always be a part of the etiology or sequella of the condition. Anything that changes movement patterns runs the risk of being learned and implemented on an ongoing basis. One sign of mPTSD is the absence of logical explanation; the absence of x-ray or MRI findings of structural lesions, injuries that don't resolve as expected, or pains that come on for unknown reasons (i.e. 'just woke up with it' or 'just turned my head'.) Ruling out mPTSD as a cause of pain or dysfunction by a short course of treatment is logical when no other reason found for pain but may be more efficient if applied before other treatments to directly address the memory component of the pain.

If underlying metabolic (i.e. fibromyalgia, RA) or structural instability, (debris in joint, meniscus, disk or ligament damage) is present, treatment that addresses mPTSD may fail to make significant change or improvements will be temporary, and will fail until the body has lost its reason to adapt.

MPTSD is a functional pathology, thus it may be noted by functional testing, namely manual muscle testing. The presence of isometric muscle weakness – the inability of muscles to lock without movement to moderate manual pressure – is a reliable indication of the presence of mPTSD. It manifests as muscle imbalances that are not in expected neurological patterns, such as weakness of muscles innervated by multiple spinal levels, weak muscles crossing asymtomatic joints or muscles remaining locked in strength despite pain.

Addressing pain and dysfunction

Reduction of mPTSD will often immediately return to grade 5 of muscles that had been grade 3-4 moments earlier. This is often accompanied a reduction in pain that may have been referred to as tendinitis or bursitis. Tendinitis for instance, is considered be a misnomer, as it is not usually an inflammatory process.[24],[25] Utah Jazz team doctor Craig Buhler who utilized similar methods that addressed adaptation, hypothesizes that tendonitis "is caused by abnormal stress patterns being placed on the muscle that attaches to the tendon."

The effects of past injuries, Buhler continues, affect "gait or movement patterns… and change the stress on particular tissues," which predisposes individuals to what is known as tendinitis. "If certain muscles that support the foot and ankle apparatus, say, aren't working efficiently, then that's going to put abnormal stresses on certain parts of the Achilles tendon, creating an overuse injury of that tendon."[26] This is substantiated clinically by the immediate reversal of symptoms of tendinitis that is often observed when removing mPTSD patterns.

This logic may also apply to prevention of more severe injuries such as Achilles tendon ruptures. A common finding in mPTSD is the loss of the reverse myotatic reflex, a spinal reflex which should reduce contractile forces in a muscle if that muscle spindles or golgi tendon organs are overstretched. In clinical examination, a muscle should weaken if it's belly is stretched longitudinally, or it's tension is stretched away from its insertion. Failure of this reflex indicates that some form of adaptation is present, and is known as over-facilitation.

MPTSD may occur in both weight-bearing and non-weight bearing joints and create body-wide patterns of over-facilitation and inhibition that will shift as appropriate treatment is applied. Such treatment may benefit in recent injuries as well, reducing commonly assumed healing times, and even immediately reduc-

ing pain. The body may default to overreactions to injuries, as evidenced by the effectiveness, particularly in the short term, of ice or anti-inflammatory medications.

Treating mPTSD

The purpose of the eye movement study was not to suggest eye movements as a primary treatment for mPTSD, rather to establish that similar mechanisms may be format both emotional and muscular conditioning. Other methods are apparently more effective for treating mPTSD.

Following an in depth evaluation of the muscles supporting the involved joint(s), joint manipulation, acupuncture, (usually 10-30 seconds of microcurrent applied to several few points) and manual muscle therapies will rapidly restore function to muscles that are imbalanced. Though the treatments are common, the choice of which location to treat is not based on established parameters of treatment from the perspective of various disciplines. Instead, treatment is directed at the disruption of the larger pattern of adaptation rather than the specific area of complaint, which often proves to be symptomatic because it is a part of a larger pattern of imbalance. When muscle imbalance is addressed in this fashion, the frequent finding of immediate return to pain-free function leads to a conclusion that many dysfunction patterns may not be associated with tissue damage at all, rather they are due to maladaptive muscle imbalances.

Muscle weakness and joint imbalance, in this model, is due not to local tissue damage or swelling (though these may be present,) instead it represents the strategic adaptation to a past trauma or pain, one that may no longer exist. We may call this condition generally, *maladaptive syndrome*.

When treating mPTSD, the area of dysfunction itself is often best addressed only after its relationship to the rest of the body has been restored. The key is to restore the latent neurological 'program' of pre-trauma function. Then rehabilitation can progress, the past ghosts exorcised.

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