

# Mind, trauma & muscle inhibition Part II: When muscle inhibition is not ignored. Evidence of effectiveness from the NBA.

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Robert Weissfeld

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**Abstract:** Part I of this series experimentally validated a hypothesis that muscle inhibition (the ongoing weakness of individual muscles clinically revealed through manual muscle testing) is sustained by trauma-induced maladaptive learning; a muscular form of PTSD (mPTSD). It demonstrated that about 90% of inhibited muscles would immediately strengthen, and 80% would remain strong over a period of weeks, when treated only with side-to-side eye movements, a part of EMDR, an accepted intervention for PTSD. This therapy has been theorised to gain its effects by interrupting reconsolidation (re-storage) of activated (recalled) and therefore destabilised memories. Currently, muscle inhibition is ignored and considered untreatable in most musculoskeletal specialties. The chiropractic subspecialty of Applied Kinesiology however, has been reversing muscle weakness for over 50 years, but because of a dearth of reporting and studies of the treatment, it has gone unnoticed in most muscle inhibition literature. A 20 plus-year 'demonstration' of correction of muscle inhibition in the NBA has left statistical and other evidence suggesting what is possible when the condition is routinely treated. Particularly striking is the finding that when treated immediately, mild to moderate sprain-strain injuries can recover in minutes, not weeks, putting into question universal assumptions we hold about the nature of tissue damage in injuries.

**Indexing Terms:** Muscle inhibition, PTSD, chiropractic, theory, NBA, Applied Kinesiology.

## Background

In Part I of this series on muscle dysfunction, we defined 'muscle inhibition' as the persistent weakness of individual muscles that is clinically discovered with manual muscle testing (MMT). (Weissfeld R, Part 1 2021)

'Muscle "weakness"', we reported, is a stated factor in diverse 'named conditions' like whiplash, (Jull, 2000; Prushansky et al., 2005) tennis elbow, (Coombes et al., 2012) runner's knee, (Ireland et al., 2003; Thomee et al., 1999) plantar fasciitis, (Allen & Gross, 2003) in addition to all manner of unnamed, non-specific pain syndromes like low back pain, (Cuthbert et al., 2018; Russo et al., 2018) and loss of performance for athletes. (Freeman et al., 2013; Mehran et al., 2016) Weakness of one or more muscles crossing a joint definitively establishes the finding of 'muscle imbalance', causing altered patterns of movement. It also suggests that the affected joint will be less stable. These factors of muscle inhibition may be what predisposes those with muscle inhibition to future injuries and osteoarthritis, (Michael V. Hurley, 1999; O'Reilly et al., 1997; Rice & McNair, 2010; Slemenda et al., 1997) and diminished athletic performance. Neurologists have described a phenomena of '*functional weakness*', '*weakness which is both internally inconsistent and incongruent with any recognizable neurological disease.*' (Stone et al., 2010)

... the use of treatments featuring reversal of muscle inhibition for over 20 years with one NBA team presented a unique opportunity to obtain data that is substantiated by third party reporting and retrospective analysis of independently gained statistics'



Because muscle inhibition persists long after seemingly successful healing, (Gabler, 2016; Rice & McNair, 2010) and is initiated by pain itself (Lund et al., 1991; Mense, 2008) along with

psychological (Stone et al., 2002, 2009, 2010) and organ stress (Hoffman & Mendel, 1977) it seems valid to state that the problem is ubiquitous. Nonetheless, it lacks a code among the 70,000-plus codes of the International Classification of Disease, the ICD-10. Diagnosis Code 'M62.81, Muscle weakness (*generalized*)' is one of those codes, but '*weakness of individual muscles*' or some equivalent, is not listed. (ICD10Data, n.d.) This means that it is not possible to epidemiologically study muscle inhibition, link it to other findings, or retrospectively determine if its treatment leads to better outcomes.

Researchers, who have been wondering about the nature of muscle inhibition since the 1800s, (Dehne, 1955) essentially consider muscle inhibition to be irreversible, '*stating that it 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; Weissfeld, 2020)

In the experiment reported in Part I, we applied the side-to-side eye movements of Eye Movement Desensitisation and Reprocessing (EMDR), an accepted therapy for post traumatic stress, as a treatment for muscle inhibition. The treatment is believed to eliminate maladaptive memories. When the eye-movements were performed immediately following the testing of a weak muscle, about 90% of inhibited muscles were immediately strengthened, with about 80% remaining strong an average of about 15 days later, leading to the conclusion that muscle inhibition is maintained by maladaptive learning. Similarity to post traumatic stress disorder led us to call the phenomenon muscular PTSD, or mPTSD.

In Part I, we postulated that muscle inhibition occurred in a two-stage process. In the first stage, the motor control system (MCS) comes to associate the use of certain muscles with pain, stress or failure - trauma, in general - and then to avoid using those muscles. Logically, that avoidance would also translate to avoidance of the muscle synergies in which those muscles play a key role. This necessitates a second stage of adaptation that we generally know as rehabilitation: the MCS develops novel synergies that recruit new combinations of muscles, omitting muscles that were previously associated with trauma, to accomplish the movement tasks required of the body. This means that, as these new synergies are learned, the inhibited muscles may never again have the opportunity to act as prime movers, making muscle inhibition a life-long condition. Even if inhibition served an adaptive ('adaptive' generally indicates a beneficial change) purpose at its onset, it becomes maladaptive when those initial conditions pass.

Because pain itself may be a contributor to the development of muscle inhibition, and inhibited muscles may remain that way indefinitely, muscle inhibition is ubiquitous, certain to be present either as a cause or result of pain. Its resolution should therefore be a central goal of musculoskeletal research but instead the condition languishes in almost complete obscurity: the weakness of individual muscles is not among the 70,000-plus codes in the International Classification of Diseases, an attempt to exhaustively list all human maladies. (Weissfeld, 2021)

Here, in Part II of this series, we will look at a treatment protocol for muscle inhibition that has been around since the 1960s known as Applied Kinesiology (AK). Perhaps because of a dearth of peer-reviewed studies of the protocol, and perhaps because of its 'alternative' origins, AK's protocol for treating muscle weakness has gone unnoticed in muscle inhibition literature.

What is available for review, however, is retrospective documentation of a 20 plus-year 'demonstration' of correction of muscle inhibition in the NBA. While certainly this is not the same as outcomes of randomised, blinded studies, its NBA tenure has left a trail of statistical and other evidence suggesting what is possible when muscle weakness is routinely treated.

## Half a century of treatment of muscle weakness

In the early 1964 Michigan chiropractor George Goodheart was approached by a young man who couldn't pass an employment physical because he was unable to push forward with one of his arms. The patient had suffered from the problem as long as he could remember.

As stated in the paper *George Goodheart, Jr., D.C., and a history of applied kinesiology*, Goodheart 'corrected [the] patient's chronic winged scapula by pressing on nodules found near the origin and insertion of the involved serratus anterior muscle.' This finding led to the origin and insertion treatment, the first method developed in AK. Successive diagnostic and therapeutic procedures were developed for neurolymphatic reflexes, neurovascular reflexes and cerebrospinal fluid flow from ideas originally described by Frank Chapman, D.O., Terrence J. Bennett, D.C., and William G. Sutherland, D.O., respectively. Later, influenced by the writings of Felix Mann, M.D., Goodheart incorporated acupuncture meridian therapy into the AK system. Additionally, the vertebral challenge method and therapy localisation technique, both based on phenomena proposed by L. L. Truscott, D.C., were added to the AK system. (Gin & Green, 1997) The winged scapula was immediately resolved, and the patient could raise his arm normally.

Based on the acupuncture system of organ-meridian associations, Goodheart also postulated that each muscle was related to a specific organ. Putting all of these factors together revealed that each muscle had its own series of reflexes (acupuncture or other points on the body surface) that could be treated, and specific vertebral segments that might be adjusted, associated with its impairment. When the specific spinal adjustments are performed, and the reflexes, along with the muscle spindles and tendon attachments of the muscle are treated by manual massage, weakness of that muscle is immediately, and often permanently resolved. (Frost, 2013; Walther, 2000)

### Differentiating aspects of AK

If this canonical protocol for treating muscles was the only innovation of AK, it may have gained wider acceptance, but several related factors may have prevented that from happening. This original protocol evaluates individual muscles one at a time, a well-accepted use of MMT in orthopedic and neurological contexts.

This is different from what has been called '*muscle response testing*' (MRT), in which a single muscle known as an '*indicator muscle*' is tested repeatedly to determine responses to sequentially presented stimuli. (Jensen, 2018) MRT is has been shown in several studies to be both reliable and valid. It indicates a change in function of a muscle, usually from strong to weak, noted when the patient's or practitioner's hand is placed on an area of suspected involvement. (Pollard et al., 2006; Rosner et al., 2015) Another feature of MRT that has been and experimentally verified is that muscle tests change when stating a falsehood. (Jensen et al., 2016; Monti et al., 1999) Essentially, these results showed that muscle testing could be used to evaluate some kind of neurological response to information from the internal and external environment, a phenomenon that should be unsurprising to anyone familiar with the literature on motor priming. (Ocampo et al., 2012; Thomaschke, 2012; Toovey, 2018; Tucker & Ellis, 2004)

In the beginning though, the methods of AK were widely distributed. Much like amateur astronomers or programmers who experiment with the tools available, MRT was adapted in different ways by many individuals over successive decades. Some, taking muscle testing to be a veritable fortune-telling machine, made claims that that they were doing 'applied kinesiology', when in fact, professional clinicians who used AK were far more conservative in what they claimed. Nonetheless, this gave critics who didn't care about such distinctions easy targets to shoot at. Further, the lack of corporate profitability and major university affiliation meant there was little money available for research.

With the brand sullied, and limited and often ignored literature to refute irresponsible (and responsible) claims, any muscle testing done by alternative practitioners is still tarred by these same brushes. Still, today there are about 1 million individuals worldwide who are doing some form muscle testing that is ultimately derived from Goodheart's early work. (Jensen, 2015) In this series on muscle inhibition, however, we are limiting ourselves to the above-described protocol for reversing muscle inhibition, which is not dependent on the MRT effects.

### Expanding the number of testable muscles

Goodheart, in his investigation of specific muscles, utilised the book generally referred to as Kendall and Kendall, which describes tests for about 70 different muscles on each side of the body to establish the basic procedures of testing of individual muscles. (Kendall & Kendall, 1949) It remains a definitive text on MMT today.

In the late 1970's however, another innovative researcher, Oregon chiropractor Alan Beardall, a student of Goodheart's AK, more than quadrupled the number of testable muscles to over 300 on each side of the body, also finding the specific reflexes to correct each one. A runner himself, Beardall had many runners as patients, and found that just treating the muscles elaborated by Goodheart insufficient. In Beardall's expanded testing that he referred to as Clinical Kinesiology (CK) there are, for instance, 13 separate tests for the quadriceps, 6 for the hamstrings, and 42 tests for the shoulder, reflecting all the muscles that attach into the bones of the shoulder girdle. (Beardall, 1980; Clinicalkinesiology.com, n.d.)

In 1979, Craig Buhler, previously a student intern of Beardall's, joined the training and medical staff for the NBA's Utah Jazz. He further expanded and optimised the AK-CK protocols, calling it Advanced Muscle Integration Technique, or AMIT®. (Mercola, Joe & Buhler, 2013) For the record, I began studying with Beardall in 1981, but because he was onto other innovations, I never fully learned the 300 muscle tests, which were difficult to extract from books. I therefore finally took Buhler's eight month course, gaining AMIT® certification in 2013.

From his experience treating the same small group of top athletes before and after games over years, Buhler concluded that if the entire protocol, which takes about 10 minutes to complete for each muscle, is not followed, that the muscle corrections might not hold. From conversations with AK practitioners, it appears that many do not utilise the full protocol, only the aforementioned origin-insertion treatment, and many may not utilise testing of the expanded set of muscles. As we discuss the results attained by Buhler with the *Utah Jazz* players, it is with the understanding that this full protocol was being administered on the full set of muscles. It should also be understood that Buhler considered additional spinal and extremity chiropractic adjustments to be important to getting these results, and sometimes he used other tools derived from muscle testing and other diagnostic systems to address nutritional or other concerns. Still, the central aim of his treatments was assuring that muscles would be responding fully and normally to their owner's intentions to contract them, and the canonical AK protocol, with the tweaks added by Beardall, was his core therapeutic intervention.

### Treatment of muscle inhibition in the NBA

In 2000, about Buhler's 20<sup>th</sup> year with the team, the *Jazz* was the oldest team (1999-00 *NBA Season Summary* | *Basketball-Reference.Com*, n.d.) in the NBA. Yet, it was also the most injury-free, its players missing only an astonishing 11 games (Buckley, 2000) that year, about the number of games that would be missed from a sprained ankle, the most common NBA injury, for one player, given the expectation of at least 4 weeks of recovery time. For comparison, the league median is around 150 games missed to injuries per year, on each team. (Rautionmaa, 2019)

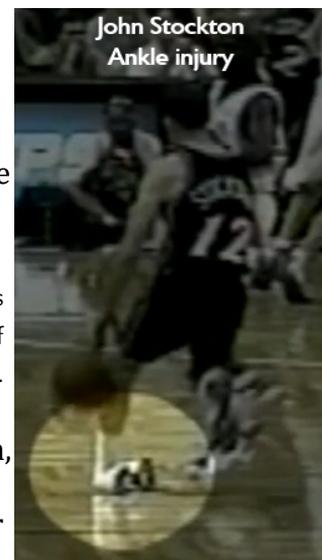
Using the NBA's data, the rest of the league operated as a control group for the treatment provided to the Jazz players. Of course, other unknown factors could play a role, so this data

cannot be considered determinative. Others might wonder if the whole treatment and training staff was just superior for over the various time periods noted.

Over the last 10 years of the century however, it was incidentally documented by data analysts looking at the whole league that *Jazz* players missed significantly fewer games to injury than the rest of the league's teams. (Wilczynski, 2011) An unpublished analysis by Buhler further demonstrated that *Jazz* players missed about half as many games per year as the average of the top 10 teams, over a multi-year span. (Buckley, 2000; Dorausch, 2002; Jarvis, Kelly B, 1998; Mercola, Joe & Buhler, 2013; Stockton, 2009)

During Buhler's tenure with the team, future hall-of-famers John Stockton and Karl Malone, who played together on the team from 1985 through 2003, were on their way to being the least injured star players over the last 40 years according to the statistical organisation 538.com. (Phillips, 2017) They are also numbers 9 and 11 respectively, on the all time list of oldest players in the NBA. (*List of Oldest and Youngest National Basketball Association Players*, 2020)

The most striking examples of the effects of this treatment however, were from its use on fresh injuries. One in particular had videographic documentation. In one game, Stockton was dribbling the ball upcourt when his ankle gave way in an ugly injury. His foot was turned in at 90 degrees, with his lateral malleolus touching the floor after stepping on the defender's foot. (Figure 1)



**Figure 1:** John Stockton's ankle injury. He was back on court after about 10 minutes of treatment, and completed the game.

Based on the appearance, he was fortunate nothing had torn or broken, as x-rays determined. Yet, he was back on the court after 10 minutes of treatment, playing out the game. In a YouTube video, (Buhler, n.d.) Buhler discusses how he treated that injury. He states that in addition to resetting the alignment of the bones of the ankle, he treated several divisions of Stockton's peroneus muscle, known to be negatively affected by ankle inversion sprains. (Palmieri-Smith et al., 2009)

Buhler also believes, as he implies in the video, that because of his previous treatment of Stockton, his tissues were more resilient and flexible, unrestricted by the usual defensive tension that occurs when the brain adapts to the instability caused by muscle inhibition. According to the logic of the reflex treatments, inhibited muscles have reduced circulation, meaning less resources reaching them. The presence of muscle imbalances change joint postures (alignments) and movement patterns, perhaps leading to more stress on ligaments as well.

Still, if this were the only instance of this kind of recovery, it could be dismissed as simply a one-off oddity. But others witnessed the regularity of such results as well. In 1998, team owner Larry Miller was recorded stating: *'If Dr. Buhler can get to a sprained ankle, it won't swell. Getting to the players quickly is the key.'* (Jarvis, Kelly B, 1998) Stockton stated in his autobiography that *'Under [Buhler's] care, sprained ankles stabilised and became pain-free in days, not weeks or months. Tendonitis went away with only a few treatments. It's not an overstatement to say that Dr. Buhler was largely responsible for me enjoying a durability and longevity that few players have come close to matching.'* (Stockton & Pickett, 2013, pp. 176–177) Other *Jazz* players, Olympic and NFL athletes have made similar statements in testimonials. (*Testimonials*, n.d.)

## Conclusion

Practitioners of Applied Kinesiology have been reversing muscle inhibition since the 1960s, but such treatment has not been widely accepted, for the reasons we have shown, most prominently perhaps because outcome studies of the treatment of inhibition have not been done. The use of treatments featuring reversal of muscle inhibition for over 20 years with one NBA team presented a unique opportunity to obtain data that is substantiated by third party reporting and retrospective analysis of independently gained statistics. While this obviously lacks the value of randomised, blinded, controlled trials, it puts forth an intriguing suggestion of what may be possible when muscle inhibition is routinely treated.

In Part III of this series we will take a new look at the phenomenology of muscle testing, and how that plays into the evaluation of muscle inhibition. In Part IV, we will look into the psychoneurophysiology of mPTSD, evaluating potential mechanisms for the ongoing weakness of individually tested muscles.

Robert Weissfeld

DC

Private practice, Denver

[drweissfeld@gmail.com](mailto:drweissfeld@gmail.com)

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