

Dig Harder! Win the War, not Just a Few Battles, on Effective Interventions for Multi-system Problems, in the Chronic Patient

Hickey CJ*

Department of Physical Therapy, California State University, USA

***Corresponding author:** Cheryl J Hickey, PT, MPT, MS, EdD, 2345 E. San Ramon Av MS MH 29, Fresno, CA 93740, USA, Tel: (559) 278-2625, (559) 278-3030; Email: wcherylba@csufresno.edu

Editorial

Volume 1 Issue 1

Received Date: October 04, 2018

Published Date: October 15, 2018

DOI: 10.23880/aphot-16000104

Editorial

The recently published application, regarding scapular dyskinesis, is a microcosm that illustrates an important dialogue about how today's therapists need to see chronicity as a multi-system interaction that requires multisystem treatments. Walker et al. used ES in a novel way to address scapular dyskinesis [1]. This application, (ES cueing with voluntary exercises) was used to address a multisystem defect versus, how stimulation is traditionally used in orthopedic practice, to address muscle hypertrophy or to gate pain. Perhaps if the orthopedic therapist considered ES cueing coupled with voluntary exercise, when treating a chronic, multisystem problem, in his or her orthopedic patients, they would see more consistent, replicable results in much shorter time frames [2].

An empty review of systems, for professional liability is one thing, however, the integration of system findings to understand the true presentation of prolonged musculoskeletal pathology is completely different. And, as the patient progresses in chronicity, lacking this integration may lead to less than optimal treatment outcomes. Given that we are on the threshold of 40% of the U.S. population falling to a chronic disease and, over 20% living with chronic pain [3,4]. The traditional single system, linear intervention may work for acute injuries but not for patients that enter the chronic state.

Consider this scenario, what if, when treating our "chronic orthopedic" patients therapists led with the idea

that the major impairment, driving the chronic pathology, has shifted to the neurologic system or even better to an inter-system dynamic? Considering questions like, how acute ankle sprains become chronic ankle instability (CAI); how acute shoulder pain becomes scapular dyskinesis; and, how acute low back pain becomes chronic low back pain are important considerations. Clinical issues like this are precursors to complicated multi-dimensional problems and these problems aren't suited to singularly constructed treatments. These interactive defects need multi-dimensional treatments. Accordingly, treatments that address things like weakness and tightness in muscles, based on functional impairments may still lead therapists to less than perfect results with chronic conditions? So why do therapists continue to see only marginal success with chronicity?

If impairments are acute, they may respond to the more-linear system approach and straight line interventions that follow but, perhaps, once the condition passes the tipping point (which may be different for every individual) and it became less of a musculoskeletal problem, and more of a poor neurological engram [5] (e.g., the mechanical defect is gone, the muscle has regained normal testing strength etc., yet we see the persistent movement deficits), the therapists will need to build multi-system interactive treatments, because now the label of "chronicity" also brings with it, all the social overlay and determinants that accompany the stigma of the chronic state, as well as the lack of adequate coping

mechanisms and all the personal circumstances magnified by the chronicity label itself.

In an additional publication under review, Walker et al. [2] indicated that in essence symptomatic dyskinesia and potentially other applications such as a chronic ankle instability CAI, show arthrogenic muscle inhibition in the musculature surrounding the chronic joint, after damage to its structure [6,7]. More importantly, these individuals demonstrate decreased alpha motor neuron excitability that leads to diminished muscle activation [7]. If this is one potential cause for the altered neuromuscular control and residual symptoms found in these patients [7,8], then perhaps the addition of ES, serves as a much more multi-system approach than it has historically been used for (strength and pain). They purport that positive outcomes seen with the use of ES in this new way are really addressing the more prominent neuro defect. This defect, is possibly less recognized and also not accessible by conventional treatment strategies. Specifically, ES cueing increases the excitability of the motor neuron pools observed in the lacking muscle, and increases the effectiveness of the altered descending corticospinal pathways, which is something that the patient cannot do voluntarily, without the electrical cueing [9,10]. Combining this with active movement, affects the joint by further increases afferent input which facilitate organizational change (neuroplastic changes) in the motor cortex at a quicker and more efficient way than voluntary contraction alone [11,12]. This happens because of the involuntary neurologic mechanisms including neuro volleying [13]. Walker et al.'s premise is grounded in Bergquist, et al.'s. observation that emphasizes plasticity of the central nervous system that can be accessed with the use of very specific ES parameters under laboratory conditions [13]. He suggests, ES at very specific parameter, will not mitigate neuro volleying but accentuate it when coupled with voluntary exercises. And these conditions may maximize neuroplasticity changes that create new movement patterns.

Using ES cueing and voluntary exercise as a multi-dimensional treatment versus using ES as a linear treatment to address pain or muscle hypertrophy will likely not get us to the same place. Although the multi-dimensional treatments are much hard to reason out or even see, they are the ones therapist must dig harder to find. When looking at issues that are chronic like scapular dyskinesia and CAI, is it possible that therapists are tending to treat in more single system defects, when what they are faced with is a chronic interactive systems

defect? Therapists will need to continue to ask the question; am I digging deep enough, for treatment strategies that affect multi-systems, in the face of complicated chronic problems?

References

1. Walker D, Hickey C, and Tregoning M (2017) The effect of electrical stimulation versus sham cueing on scapular position during exercise in patients with scapular dyskinesia. *Int J Sports Phys Ther* 12(3): 425-436.
2. Walker D, Hickey C The effect of electrical stimulation cueing and exercise on scapular position in a patient with shoulder pain and scapular dyskinesia: A case study.
3. National Health Council (2014) About Chronic Disease.
4. Center for Disease Control and Prevention (2018) Prevalence of Chronic Pain and High-Impact Chronic Pain Among Adults- United States 2016.
5. Monfils M, Plautz E, Kleim J (2005) In search of the motor engram: motor map plasticity as a mechanism for encoding motor experience. *Neuroscientist* 11(5): 471-483.
6. Hopkins JT, Ingersoll CD (2009) Arthrogenic muscle inhibition: a limiting factor in joint rehabilitation. *J Sport Rehab* 9(2): 135-159.
7. Eric DM, Riann MP, Carrie LD, Steven MZ, Christopher DI (2005) Arthrogenic Muscle Inhibition in the Leg Muscles of Subjects Exhibiting Functional Ankle Instability. *Foot Ankle Int* 26(12): 1055-1061.
8. Gutierrez GM, Kaminski TW, Douex AT (2009) Neuromuscular control and ankle instability. *PM R* 1(4): 359-365.
9. Nelson RM, Hayes KW, Currier DP (1999) *Clinical electrotherapy*. Prentice Hall.
10. Riann MP, Christopher DI, Mark AH (2004) The hoffmann reflex: methodologic considerations and applications for use in sports medicine and athletic training research. *J Athl Train* 39(3): 268-277.
11. Sugawara K, Tanabe S, Higashi T, Tsurumi T, Kasai T (2011) Changes of excitability in M1 induced by neuromuscular electrical stimulation differ between

- presence and absence of voluntary drive. *Int J Rehabil Res* 34(2): 100-109.
12. Sugawara K, Yamaguchi T, Tanabe S, Suzuki T, Saito K, et al. (2014) Time-dependent changes in motor cortical excitability by electrical stimulation combined with voluntary drive. *Neuroreport* 25(6): 404-409.
 13. Bergquist A, Clair J, Lagerquist O, Mang C, Okuma Y, et al. (2011) Neuromuscular electrical stimulation: implications of the electrically evoked sensory volley. *Eur J Appl Physiol* 111(10): 2409.

