

THE UNRELENTING MISPERCEPTIONS OF SPASTICITY AND HYPERTONICITY

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The Problem

What is the misperception about this topic?

The misperception is one in which several leaders in Physical Therapy, including Dr. Carol Giuliani identified as stemming from the perception that spasticity was the cause of movement dysfunction in patients with neurologic diagnosis such as stroke and cerebral palsy. That perception led to a focus on interventions aimed at reducing spasticity. Dr. Giuliani and others contributed literature that importantly led to the 'resolution' of that specific problem. However, tremendous confusion still exists. According to Davidoff¹, the term "muscle tone" has been used imprecisely in neurologic literature, in part because it is defined so differently by the neurologic clinician and by the experimental neurophysiologist. While clinicians will correctly state the definition of muscle tone as the resistance to movement when the patient is relaxed and attempt to assess the magnitude of the resistance to passive flexion and extension of the joints in a limb, there is the unfortunate tendency to attribute impaired volitional movement ability to the tone state.

Historical Perspective

When in physical therapy history did the tone or reflexive state of a muscle become connected to movement?

In the momentous chapter written by James Gordon, EdD, PT, FAPTA in 1989 entitled 'Assumptions Underlying Physical Therapy Intervention: Theoretical and Historical Perspectives,' Gordon identifies that a remarkable process began in physical therapy through the 1950's and into the 1960's in which therapists began to study how the central nervous system (CNS) worked with the purpose of determining how to better treat patients with brain damage. As Gordon states: 'The result of this process – the development of several neurotherapeutic approaches – brought about a true revolution in the way neurological patients were rehabilitated.'² True remediation of movement abnormalities stemming from neuropathology was now considered possible as the CNS itself became the focus of treatment, not the peripheral expression of brain damage in the musculoskeletal system. The positive effect was that emphasis was now placed on trying to correct the underlying causes of movement deficits. However, the downside of this new slant became an overemphasis on neurophysiological explanations of movement

abnormality. In particular, the assumption that spasticity was a direct cause of disordered movement came to dictate clinical practice. The Brunnstrom Approach proposed six stages of sequential motor recovery after a stroke. Whether by design or by accident, the tone state became intricately linked to the movement state: Stage 1: Immediately following a stroke there is a period of flaccidity *whereby* no movement of the limbs on the affected side occurs. Stage 6: Spasticity is no longer apparent, allowing near-normal to normal movement and coordination. Multiple quotes by Bobath directly state the tone-movement connection: '...severe degrees of spasticity will make movements impossible...this indicates the intimate relationship between spasticity and movement and points to the fact that spasticity must be held responsible for much of a patient's motor deficit. How far have we come from a viewpoint that prescribed treatment of tone as a prerequisite to normal movement when current literature identifies that the paradox in relation to spasticity is that it has been defined in resting limbs, yet its clinical management is directed at the associated movement disorder. The rationale continues to be that sustained overactivity in some way limits limb performance.'³

The Unrelenting Link

Why does this problem continue to propagate?

We have narrowed down the cause for why this continues to pose such a significant challenge to clinicians to three main areas:

1) Terms are Used Interchangeably:

Even though the terms hypertonus, and spasticity are commonly used interchangeably, they refer to different forms of disordered tone. Hypertonia is resistance to passive movement and not dependent on velocity, Spasticity is directly related to the speed of the passive stretch. They are not interchangeable and not dependent on one another. Hypertonicity can be present without spasticity and vice versa. While the key problem is the automatic propensity to associate either or both with volitional movement ability, it is erroneous and imprecise to continually substitute two terms when they clearly result from two distinct pathophysiologic mechanisms.

2) Lack of Precision in Utilization of Terms

In 2009, Malhotra et al.,⁴ conducted a systematic literature search of 250 studies to determine whether there was a consistent definition and a unified assessment framework for the term 'spasticity.' The review established that not only was the term spasticity inconsistently defined but that often the measures used did not correspond to the clinical features of spasticity. Multiple studies apparently investigating the relationship between spasticity and movement fail to provide the definition or clinical inclusion criteria that clearly identify subjects with "spasticity."

3) Difficulty in disassociating observationally apparent tone impairment and movement dysfunction:

Understandably, it is challenging for any of us to feel excessive resistance in the quadriceps for example, and not suppose that the felt resistance must underlie the inability for a patient to flex the knee during the swing phase of gait. While multiple studies have shown an association between what may be spasticity or hypertonicity and functional ability, it is important to note that, at best, a *correlation* is demonstrated. No study has yet to demonstrate a causal relationship between spasticity or hypertonicity and functional ability.

Importance of Change

Why are we trying to change this now?

The inconsistency in terminology and the inaccuracy in linking movement capability with the tone state must be resolved. We are not the first to try to alter the perceptions that exist. Leaders in physical therapy have been presenting and writing about this issue for years. As stated earlier the work of a number of individuals including Drs. Carol Giuliani, James Gordon, Pam Duncan and Steven Wolf all but resolved the unrelenting clinical notion that tone needed to be treated in advance of movement retraining but the tendency to sweep all movement abnormality under the tone 'rug' continues to exist.

The True Clinical Importance

When do spasticity and hypertonicity matter?

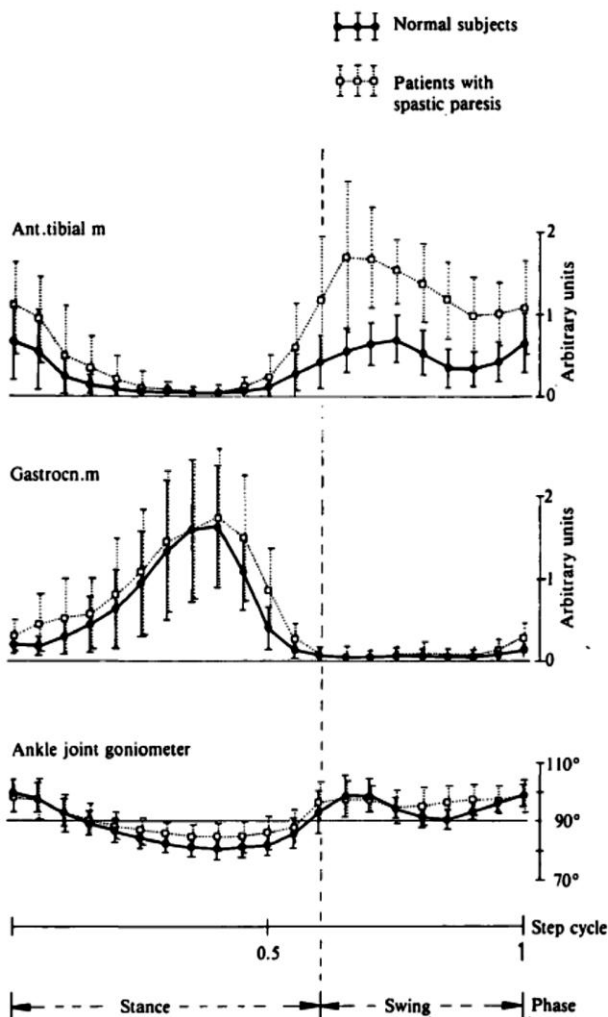
Considering that spasticity is a sign of an upper motor neuron lesion, the diagnosis of stroke and the finding of spasticity is redundant information. However, the finding of spasticity following whiplash as a result of a car accident is important information as it establishes that there has been damage to the spinal cord and/or CNS. Since Landau in 1974, multiple experiments have supported the view that spasticity is not related to volitional movement capability and yet according to O'Dwyer, Ada and Neilson 'the continued interest in mechanisms of and therapeutic interventions for spasticity suggest that it retains a focus that is out of step with its theoretical importance.'⁵ Given that the definition of muscle tone includes both a neural and mechanical component, it is not surprising that hypertonia has been associated with contracture.⁵ The relationship between hypertonia and abnormal movement is then through the potential loss of mobility due to abnormal stiffness of the passive tissues. This launches the contention that the important objective in treating hypertonia is maintaining range of motion.

The Evidence

Picture common gait deviations following stroke. You won't have to think long before hypertonia or spasticity enter your thoughts. For example, consider your patient walking with their knee fully extended and their heel off of the ground throughout stance. During swing, they have limited toe clearance due to limited knee flexion and excess ankle plantarflexion. Hypertonia in the calf and quadriceps is often thought to cause this gait pattern. We confidently state this person has an "extensor pattern" or "spastic hemiplegia." Is this really true? What does the scientific literature say?



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Dietz V, Quintern J, Berger W. Brain. 1981;104(3):431-49.

Averaged surface EMG and ankle position from 20 unimpaired individuals and 10 people with hypertonia and spasticity due to spinal lesions. Note there is no calf activity during swing in the group with hypertonia and spasticity, despite the presence of excess ankle plantarflexion..

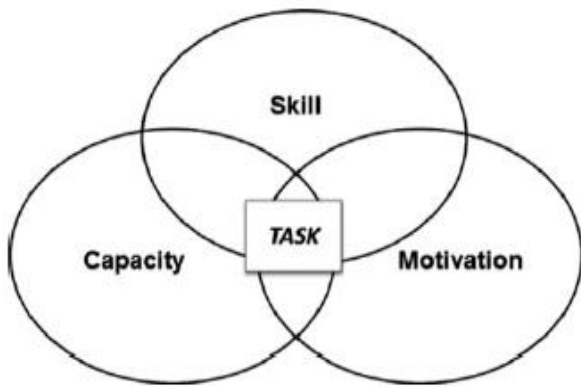
Do antagonist hypertonicity or spasticity interfere with movement?

During swing, the ankle dorsiflexor muscles act as agonist muscles to assist with toe clearance. The calf muscles act as antagonists to this motion. If calf muscles are activate during swing, ankle plantarflexion and limited toe clearance could result. However, evidence for this is lacking.⁶⁻¹⁰ In multiple studies, excess calf activity during swing has not been found to cause limited toe clearance. Rather, limited ankle mobility is consistently considered to be the problem.

In addition to gait, studies of voluntary arm movement show no evidence of excess antagonist activity that would interfere with movement.^{3,11-13}

Do agonist hypertonicity or spasticity interfere with movement?

During stance, the calf muscles act as agonist muscles to control forward progression of the tibia. Inappropriate, or "excess" calf activity could cause the ankle to plantarflex and the heel to rise from the ground. Again, evidence for this is lacking and limited ankle mobility is the more likely cause (Dietz 1981, Berger 1984, Berger 1988, Dietz 1991, Sinkjaer 1996).⁶⁻¹⁰



Winstein C, et al. *J Neurol Phys Ther.* 2014;38(3):190-200.

I can see the muscle contracting when it shouldn't be. How is hypertonia not to blame?

The discussion thus far has focused on inappropriate muscle activity during gait as a proxy for hypertonia or spasticity. These are, of course, passive phenomena. However, spasticity and hypertonicity enter the dialogue of gait every day in the clinic, which necessitates this discussion. Excess muscle activity during voluntary movement is too often mislabeled hypertonia when it truly represents compensatory behaviors.

When the demands of a task exceed a person's capabilities, abnormal muscle recruitment and movement are likely unavoidable (e.g. Winstein 2014).¹⁴ However, this is *not* hypertonia or spasticity. For example, people *without* upper motor neuron lesions (e.g. shoulder impingement syndrome) display compensatory maneuvers much like people with stroke.^{15,16} Nevertheless, the presence of stroke obscures clinical reasoning and any aberrant muscle activity is quickly attributed to spasticity or hypertonia without considering biomechanical variables. For example, the person with stroke who walks with their knee fully extended and their heel off the ground during stance may be purposefully compensating for weak calf muscles, which are unable to eccentrically control forward progression of body weight. By avoiding ankle dorsiflexion altogether, demand is significantly reduced.

What is the relationship between contracture and hypertonia?

As mentioned above, limited joint mobility is often part of the clinical picture surrounding hypertonia. This *does* directly impair movement. The person who walks with their heel off of the ground likely has limited ankle dorsiflexion range of motion (ROM). Did hypertonia cause this? Can chronic activation of a muscle lead to contracture?

The answer is unclear due to inconclusive evidence, but, in general, muscle contracture often occurs early due to immobilization, and later, inappropriate motor unit activity may contribute to limited joint mobility.^{3,5,13,17-19} This suggests clinicians need to aggressively manage contracture in the acute stage of recovery from neurologic injury. Maintaining joint mobility may prevent the stiff-legged gait pattern we have been discussing from emerging in the first place.

How seriously should we interpret studies that do show a connection between hypertonia or spasticity and movement capability?

Year after year, studies are published reporting significant correlations between some measure of spasticity and some measure of voluntary movement capability. However, as is well-known, correlation does not equal causation. Authors of these studies do not forget to state this, but clinicians are quick to assume causation and correlation are one and the same. Clinical assessments performed while the participant is fully relaxed cannot yield information about voluntary movement capability. Strong correlations may exist, but that is all. Voluntary motor control in the setting of a specific functional task should be the focus of assessment and treatment. This is the art of physical therapy and the path to becoming a better clinician.

Clinical Impact

What role does PT play?

Therapeutic goals are dependent on setting and patient presentation. Factors that contribute to this include: shortened lengths of stay, importance of reducing risk of falls, patient support systems, time since onset of neurological injury, and many more. The following list is created to explore potential situations in which treatment plans may inadvertently be supporting development of aberrant motor control that can impact a patient's functional mobility.

Conditions that potentially promote poor motor control:

- Utilization of early open chain movement that exceeds demand of movement capacity and therefore drives increases in co-contraction and impaired ability to access fractionated or graded muscle forces.
- Utilization of assistive devices and bracing to promote stability rather than mobility (quad canes,

knee immobilizers, etc). These may inadvertently cause a patient's active co-contraction of agonist and antagonist to promote joint stability.

- Learned compensations that are the unintended consequence of transfer training towards the "uninvolved" side and minimizes opportunities to load the "involved" side.
- Recommendation during stair training to go "up with the good, down with the bad" which again supports learned non-use of involved extremities.

"However useful to clinical diagnosis may be the increase of excitability at anterior horn cells and, to some extent muscle spindles, these phenomena have little more relation to the patient's disability than does the insertion of the rectal thermometer in pneumonia."

- William M. Landau, MD 1970

The Art in Clinical Practice

How do I explore one's capacity for movement?

Start with a movement, any movement. Assessment should begin with the initial conditions of the movement and end when volitional effort is terminated. Various movement analysis systems have been proposed to assist in a systematic breakdown of simple and complex movements. The art of our profession arises when clinical decisions are made about how to assess further and intervene to promote ideal movement. Too often we rely on an impairment model with a formulaic approach to improving movement. Instead, our challenge can be to develop a deeper understanding of why a patient moves in such a manner. What role is gravity playing on the system? How can I modify the environment to limit compensation? What is the most critical element of the movement deviation that dictates subsequent movement?

The central nervous system is a frontier yet to be conquered. Lesions within it can cause an infinite amount of dysfunction to motor planning and control. Within each patient, the physical therapist must search for and alter the unique limitations in order to emancipate true movement capability. Key points we feel provide assistance in this process:

- Don't be afraid to problem-solve with the patient. Discover the movement capacity with your patient.
- We are the experts in movement, but that does not mean you are expected to have every answer. Merely having the right questions is at times enough.
- Expect it to be challenging. The skill of being artistic and creative can be a difficult process.
- Generate movement hypotheses and create movements that will change the demand of the task and provide insight into your patient's limitations. Test these hypotheses repeatedly.

Future Directions

Where do we go from here?

As numerous authors have concluded, the focus of future research should attempt to discover preventative techniques to thwart development of aberrant motor control during the rehabilitative process following upper motor neuron injuries and to equip clinicians with the tools to directly impact functional mobility with efficient utilization of medical management.

Clinicians, Regarding Clinical Reasoning:

We are the front line defense against further propagation of the misperception of spasticity and hypertonicity. Be critical and creative when evaluating human movement. Do not let the presence of a single impairment limit creativity and the pursuit of ideal movement. Be the voice of a profession defined by the ability to improve the way the human movement system operates by

discussing this need for change with medical personnel, colleagues, and patients.

Clinicians, Regarding Patient Education:

Empower them by providing factual education about their role in improving the quality of movement versus allowing spasticity and hypertonicity be the sole cause of disability. Discuss how practice variables of challenge, intensity, and task specificity will dictate the rate of recovery.

Researchers:

Address clinical questions that advance our understanding of how to assess and intervene when abnormal motor control is present, as opposed to less meaningful research into correlational evidence between movement and hypertonicity or spasticity.

Mobility Management

What role does hypertonicity play in contracture?

In examining the range of motion across a patient's rehabilitative process the risk of contracture is large. Potential substrates that are altered in the presence of contracture and atrophy are; loss of sarcomeres, accumulation of intramuscular connective tissue, increased intramuscular fat content, and degenerative changes at the myotendinous junction.¹⁸

This is likely the most important way in which spasticity or hypertonicity interferes with movement and yet it is indirect, at best. The agonist muscle must overcome passive tension of the antagonistic soft tissue in order to achieve the desired movement.

Our Question to You:

If the evidence consistently demonstrates that spasticity and hypertonicity are not the direct cause of movement dysfunction, why do we persist in assessment and intervention of these impairments with relation to movement dysfunction?

"If therapists can identify motor dysfunction using concepts of motor control and direct their intervention to the problems of control, they will be using a scientific approach to practice."

- Carol Giuliani, 2002²⁰

In the clinic tomorrow...

- Resist the urge to associate a patient's movement dysfunction to spasticity or hypertonicity as they are merely passive entities with weak correlational connect to volitional movement
- Challenge yourself to manipulate the manner in which your patient is interacting with the environment in order to extract true performance capacity.

Important Terminology

Tone: The tension attained at any moment between the origin and the insertion of a muscle. The tension is determined partly by mechanical factors (connective tissue and viscoelastic properties of muscle) and the degree of motor unit activity.²¹

Hypertonicity: The sensation of [increased] resistance felt as one manipulates a joint through a range of motion, with the subject attempting to relax.²²

Spasticity: a velocity-dependent increase in the tonic stretch reflex with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome.²³

Spasm: Persistent increased tension and shortness in a muscle or group of muscles that cannot be released voluntarily.²⁴

Contracture: An abnormal, often permanent shortening, as of muscle or scar tissue, that results in distortion or deformity, especially of a joint of the body.²⁴

Motor Learning: A set of processes associated with practice or experience leading to relatively permanent changes in the capability for responding.²⁵

Motor Control: The study of how our neuromuscular system functions to activate and coordinate the muscles and limbs involved in the performance of a motor skill.²⁶

“We need to teach ourselves the analytical skills necessary for making a sophisticated biomechanical and functional evaluation of our patients’ movement disorders.”

– James Gordon, 2000²

Suggested Readings:

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