Muscle Overactivity in the Upper Motor Neuron Syndrome: Pathophysiology

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KEYWORDS

• Spasticity • Muscle over-activity • Upper motor neuron syndrome • Hypertonia

KEY POINTS

- The positive signs of the upper motor neuron syndrome, including spasticity, all involve muscle overactivity. Of these, some are stretch medicated and some are not—the so-called efferent" phenomena.
- The positive signs, including spasticity, clonus, flexor and extensor spasms, associated reactions, cocontraction and spastic dystonia, do not necessarily co-occur and may vary in their pathophysiology.
- Muscle activity is modulated by descending motor pathways, both inhibitory and excitatory.
- Muscle overactivity in the upper motor neuron syndrome results from abnormal signal processing in the spinal cord because of altered supraspinal inputs and/or dysfunction of segmental spinal modulation.
- Rheologic features of the upper motor neuron syndrome involve changes in the physical properties of muscle and can also affect and be affected by weakness and hypertonia.

INTRODUCTION: ALL THAT GLISTENS IS NOT GOLD; ALL THAT CONTRACTS IS NOT SPASTICITY

Patients with upper motor neuron (UMN) syndrome often develop abnormal patterns of muscle activity as expressed clinically is negative signs (muscle underactivity) and positive signs (muscle overactivity). Although negative signs underlie clinical paresis of voluntary movement, positive signs present as a variety of involuntary phenomena, the most familiar of which are spastic stretch reflex behaviors. In day-to-day practice, clinicians often used the term, *spasticity*, as a catch-all term to describe the overlapping phenomena of muscle overactivity that is observed in patients with UMN syndrome.

Disclosure Statement: The author has nothing to disclose.
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Phys Med Rehabil Clin N Am ■ (2018) ■–■
https://doi.org/10.1016/j.pmr.2018.04.005 **pmr.theclinics.com**

The term, spasticity, however, is defined narrowly as "velocity dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome."[1] This definition only applies to a subset of the phenomena to which the term, spasticity, is often casually applied. These phenomena are perhaps more accurately discussed as the so-called positive features of the UMN syndrome. Pitfalls exist with this language as well, because it groups mechanistically different phenomena together that do not necessarily co-occur and may vary with respect to treatment. Additionally, this also implies that all of these features result directly from dysfunction of the upper motor neuron or pyramidal tracts. This is not entirely the case, because the pathology can have a much broader basis, from circuits which modulate the pyramidal tracts to plastic changes in the spinal reflex circuitry as well as pathologic changes in muscles themselves.[2,3] Semantics aside, in this article, the term, *muscle overactivity*, is preferred to understand and describe the presentation of the UMN within a larger pathophysiologic context.

CHARACTERISTICS OF THE UPPER MOTOR NEURON SYNDROME

Characteristics of the UMN syndrome have been described in terms of the duality of negative and positive signs but some investigators have also suggested that changes in the viscoelastic properties of muscle, also known as rheologic changes, should be considered a third sign[4] (**Table 1**). This article focuses primarily on the positive signs, which all involve increased muscle activity and mostly involve exaggerated spinal reflexes. The negative, positive, and rheologic signs, however, interact with one another, often in a perpetuating manner.[2,5]

Spasticity

As defined by Lance in 1980 (see above), spasticity refers to a velocity dependent increase in the tonic stretch reflex.[1] At velocities used to test tone in normal, relaxed muscle, tonic stretch reflexes do not contribute to muscle tone but rather tone is generated by the viscoelastic properties of muscle. In contrast, muscles of hemiparetic stroke patients demonstrate electromyographic activity in linear proportion to the stretch velocity imposed.[6] Of note however, spasticity is also a length dependent phenomenon, with a tendency toward an increase at longer lengths in the lower extremity and at shorter lengths in the upper extremity.[7–9] It has also been shown that there is an interaction between the length and velocity variables. For example, the

Table 1
Characteristics of the upper motor neuron syndrome

Negative	Positive
Weakness	Hyperreflexia and reflex irradiation
Loss of Dexterity	Clonus
Fatigue	Spasticity
Impaired motor planning	Positive babinski and other primitive reflexes
Impaired motor control	Extensor spasms
	Flexor spasms
	Positive support reaction
	Co-contraction
	Associated reactions (Sykinesis)
	Spastic dystonia