

# July 2018 Spasticity in Stroke



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Hello members.

We hope you enjoyed our topic of early mobilization. Stay tuned as we have a conversation with Julie Bernhardt to learn more.

Until then, let's start this month with a new topic. SPASTICITY. Our topics have been identified by Stroke SIG members.

The first abstract, article, and the clinical point of view is provided below. The focus is on defining and understanding what spasticity is. We will delve into assessment and treatments of spasticity in later weeks.

Enjoy.

[Front Neurol. 2017 Apr 3;8:120. doi: 10.3389/fneur.2017.00120. eCollection 2017. Spasticity, Motor Recovery, and Neural Plasticity after Stroke. Li S1,2.](#)

## Abstract

Spasticity and weakness (spastic paresis) are the primary motor impairments after stroke and impose significant challenges for treatment and patient care. Spasticity emerges and disappears in the course of complete motor recovery. Spasticity and motor recovery are both related to neural plasticity after stroke. However, the relation between the two remains poorly understood among clinicians and researchers. Recovery of strength and motor function is mainly attributed to cortical plastic reorganization in the early recovery phase, while reticulospinal (RS) hyperexcitability as a result of maladaptive plasticity, is the most plausible mechanism for poststroke spasticity. It is important to differentiate and understand that motor recovery and spasticity have different underlying mechanisms. Facilitation and modulation of neural plasticity through rehabilitative strategies, such as early interventions with repetitive goal-oriented intensive therapy, appropriate non-invasive brain stimulation, and pharmacological agents, are the keys to promote motor recovery. Individualized rehabilitation protocols could be developed to utilize or avoid the maladaptive plasticity, such as RS hyperexcitability, in the course of motor recovery. Aggressive and appropriate spasticity management with botulinum toxin therapy is an example of how to create a transient plastic state of the neuromotor system that allows motor re-learning and recovery in chronic stages.

Clinical Point of View

- Spasticity defined. Velocity-dependent increase in resistance during passive stretch, from hyperexcitability of the stretch reflex.
- \*Mechanism of spasticity. Poorly understood. Hypothesis. Imbalance between descending inhibitory and facilitatory regulation of spinal stretch reflexes because of cortical disinhibition, e.g. after stroke. There are 3 descending pathways Corticospinal (CST), Reticulospinal (RST) and vestibulospinal (VST). The stretch reflex involves afferent, spinal motor neurons, and efferent fibers) and it is regulated by descending supraspinal origins RST and VST (to a lesser extent). The CST is cortical and provides voluntary movement, if damaged, the RST has stronger signal.
- Motor recovery stages after stroke. 1) flaccidity, 2) spasticity, 3) increased spasticity with synergistic voluntary movement, 4) movement out of synergy and spasticity decreasing, 5) more complex movements and spasticity decreasing, 6) spasticity disappears, 7) full recovery of coordinated movement. Different states of recovery could reflect different pathophysiology.
- **Key point.** Spasticity and motor recovery are mediated by different mechanisms.
- Clinical point, focus on motor recovery for cortical plasticity and focus on reducing spasticity-maladaptive plasticity (from supraspinal RST input). Consider, as we push our patients for cortical reorganization via sustained activity-dependent, goal-oriented training programs focusing on motor re-learning. We also need to identify ways to reduce the input from the RST, The author discusses interventions to reduce the mal-adaptive plasticity, such as non-invasive brain stimulation, pharmacological agents (botox), auditory stimulation. How can we reduce the maladaptive plasticity?
- Keep in mind, spinal plastic changes correlate with severity of cortical injury. So integrity of CST matters. And while a time course of spontaneous recovery has thought to taper off at 6 months, we still have physiological processes we can intervene with.

\*For a great diagram of tracts and the possible mechanism interactions, see Li S, Francisco G. New insights into the pathophysiology of post-stroke spasticity. *Front Hum Neurosci* (2015) 9:192. doi:10.3389/fnhum.2015.00192

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