Spasticity Management After Stroke

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KEYWORDS

• Stroke • Spasticity • Symptom management • Outcome measures • Rehabilitation

KEY POINTS

- Spasticity from stroke is classified as cerebral-origin spasticity, characterized by hyperexcitability of monosynaptic pathways, a rapid rise in excitation, and stereotypical postures involving antigravity muscle groups.
- The prevalence of spasticity after stroke can be as high as 46% in the chronic phase (over 3 months).
- Spasticity is most often associated with other neurologic impairments, in particular paresis, which complicate its evaluation and management.
- Treating spasticity after a stroke entails combining nonpharmacological and pharmacologic interventions.
- There is emerging evidence suggesting that some treatments for spasticity improve upper and lower extremity functional performance.

INTRODUCTION

Spasticity is a movement disorder, defined as a velocity-dependent increase in stretch reflexes[1] due to impaired supraspinal inhibitory signals. Recent observations suggest, however, that decreased homosynaptic depression (ie, impaired depletion of the release of excitatory neurotransmitters with repetitive afferent activation), rather than decreased presynaptic inhibition, is associated with poststroke spasticity.[2] In addition, defective supraspinal control of various spinal inhibitory and facilitatory circuits is associated with abnormal muscle contraction during voluntary movement.[3] Finally, changes in the rheologic and contractile properties of musculoskeletal soft tissue are frequently associated with chronic spasticity and in turn have been linked to increased spasticity.[4]

Cerebral-origin spasticity after a stroke differs from spinal-origin spasticity as encountered in spinal cord injury and multiple sclerosis. Cerebral-origin spasticity is characterized by

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- Hyperexcitability of monosynaptic pathways
- A rapid rise in excitation
- Stereotypical postures involving antigravity muscle groups. The hemiplegic posture commonly observed after a stroke consists of
  - In the upper extremity: shoulder adduction; forearm pronation; and elbow, wrist, and finger flexion
  - In the lower extremity: hip adduction, knee extension, ankle plantarflexion, and often pes varus

Spasticity is only one component of the upper motor neuron (UMN) syndrome. As a consequence, other features of the UMN syndrome most often accompany spasticity, such as weakness, loss of dexterity, and synkinetic movements. In one study, 100% of patients with spasticity exhibited limb weakness (vs 50% of patients without spasticity) when assessed over 3 months after a stroke.[5] Because other neurologic impairments are commonly seen after a stroke (eg, visual, sensory, and cognitive), it may be difficult to isolate the specific impact of spasticity on functional limitations. It has been stated that the functional limitations experienced by stroke survivors are mostly related to neurologic deficits other than spasticity.[6]

The prevalence of spasticity after stroke is difficult to ascertain in the absence of rigorous population-based studies. In a survey from the National Stroke Association, 57% of 504 stroke survivors reported tight or stiff muscles, suggesting the presence of spasticity.[7] Clinician-based determination of the prevalence of spasticity is most often conducted in hospital stroke units, with sample sizes ranging from 50 to 200 individuals. Prevalence values are between 4% and 27% in the first month after stroke and between 17% and 46% past 3 months.[8,9] Longitudinal studies generally report higher prevalence values at the chronic phase.[5,9,10]

PATIENT EVALUATION OVERVIEW

Identifying Spasticity

The diagnosis of spasticity is based on clinical features, in the context of a central nervous system disorder. Spasticity is usually associated with some or all of the patient complaints presented in **Table 1**. Even though these symptoms are suggestive of spasticity, they are not sufficient to establish the diagnosis. For example, difficulty moving a limb can be described as “stiffness” even when it is due to weakness. Pain can be related to spasticity (particularly if it is associated with spasms or occurs with passive movement), but central neuropathic pain (particularly after a thalamic stroke) or musculoskeletal pain may also be present.

The diagnosis of spasticity is confirmed by clinical examination findings, summarized in **Table 1**. Some of these signs are associated with passive stretch (resistance

Table 1
Patient complaints and examination findings associated with spasticity

Complaints	Clinical Signs
• Muscle stiffness or tightness	• Resistance to passive movement
• Muscle spasms	• Clonus
• Clonus (shaking)	• Spasms
• Pain	• Co-contraction of agonist and antagonist muscles
• Difficulty performing voluntary movement	• Spastic dystonia
	• Decreased passive range of motion
• Limb deformity (cosmetic or functional concern)	• Abnormal posture
	• Limb deformity