# Special Considerations and Assessment in Patients with Multiple Sclerosis

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**KEYWORDS**

• Multiple sclerosis • Spasticity • Botulinum toxin • Cannabinoids

**KEY POINTS**

- Multiple sclerosis is a progressive neurologic autoimmune disorder that may cause demyelinating lesions anywhere in the central nervous system.
- Spasticity is a symptom of multiple sclerosis that causes an increase in muscle tone, hyperreflexia, spasms, and pain.
- Spasticity in patients with multiple sclerosis can have profound functional implications that influence the quality of life for patients.
- Treatment objectives for spasticity in patients with multiple sclerosis should be clearly defined and individualized.
- Treatment of spasticity may include pharmacologic agents, physical therapy, botulinum toxin, cannabinoids, and modalities.

## INTRODUCTION

Multiple sclerosis (MS) is an autoimmune disorder associated with demyelination, axonal damage, neurodegeneration, and astrogliosis.[1] It is a progressive disease that can cause damage anywhere in the central nervous system (CNS). Spasticity is a symptom of MS, which is part of the upper motor neuron syndrome and is typified by increase in tone, muscle hyperactivity, spasms, and stiffness, among other complications. Spasticity is a disabling symptom of MS in more than 80% of patients, often leading to decreased function, pain, contractures, and skin breakdown.

## PATHOPHYSIOLOGY

The process of demyelination and remyelination, with exacerbation and remission, adds a dynamic component to the MS disease process. The pathology is unpredictable

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and individualized; accordingly, the associated spasticity can change significantly over time. The chronically damaged neurons of patients with MS are vulnerable to metabolic changes, stress, or inflammatory signals accounting for dynamic changes seen in patients with MS.[2] Similarly, the burden of the disease and function of patients can change. Continual reevaluation of the approach to treating spastic patients with MS by physicians, therapists, and patients based on individualized symptoms is critical to maintaining function, activities of daily living (ADLs), and quality of life.[3]

In the setting of central autoimmune damage in MS with neuroplasticity, there is a shift that increases the excitatory inputs to alpha motor neurons with disinhibition of reflexes. With compromise of the myelin sheath, there is ephaptic spread among axons of the action potentials distributing the excitation.

Patterns of spasticity in MS can vary and may be restricted to one limb or one side of the body. Alternatively, the spasticity can be more diffuse or assume a para or tetra pattern. A change in balance of the excitation and inhibition of alpha motor neurons can cause spasticity change throughout the day. A para-spastic pattern involving the lower limbs has important ramifications for transfers and ambulation.

## EPIDEMIOLOGY

The prevalence of spasticity in MS was studied looking at patient data from the North American Research Committee on Multiple Sclerosis (NARCOMS).[4] They found that 74% of relapsing remitting patients were spastic with 55% mild and 19% moderate or severe, and 90% of secondary progressive patients were spastic with 48% mild and 43% moderate to severe. The primary progressive group of patients revealed 80% spasticity with 50% mild and 30% moderate to severe. Men were more likely than women to be severely spastic, and the spasticity symptoms worsened with age and duration of MS. Risk factors for the development of spasticity included pain, motor impairment, and bladder dysfunction.[5]

## ASSESSMENT

Illomei and colleagues,[6] in 2017, published a report on the use of muscle elastography to evaluate spasticity in patients with MS. Elastography is real-time ultrasound that evaluates muscle fiber status and changes with treatment. Working with the understanding that the Ashworth scale has limitations in that it does not correlate well with disability and is not sensitive to change, they developed a muscle elasticity MS score. This scale is a muscle fiber rigidity imaging scale, which has strong correlation with the Ashworth scale, serving as an objective means to evaluate MS spasticity.

## TREATMENT

Detrimental effects of spasticity include decreased function, pain, increased burden of care, contractures, skin breakdown, and pressure wounds. This point is not to say that all spasticity needs to be treated. One must consider the negative effects of reducing tone, for example, patients who rely on stiff, spastic lower limbs for stability during transfers and standing. Immunotherapy and spasticity medications may have a negative effect on the MS disease process by increasing spasticity (immunotherapy) and by decreasing autoimmunity (spasticity meds).[7] Sometimes transient short-term treatment of spasticity is required, as when symptoms are worsened by a urinary tract infection or decubitus ulcer.

Treatment objectives and therapeutic targets should be clearly defined. The treatment protocol is most successful when multimodal, integrated, and individualized.