Common Etiologies of Upper Extremity Spasticity

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KEYWORDS

• Spasticity • Spastic hypertonia • Upper extremity • Motor neuron syndrome • Cerebral palsy • Spinal cord injury • Stroke • Cerebrovascular accident

KEY POINTS

- Spasticity is a motor disorder characterized by increased muscle tone and a hyperexcitable stretch reflex.
- The most common causes of upper extremity spasticity include stroke, traumatic brain injury, multiple sclerosis, spinal cord injury, and cerebral palsy.
- The underlying pathophysiology of spasticity may vary, but the clinical manifestations are somewhat predictable and include elbow flexion, forearm pronation, wrist flexion, and thumb/digital flexion.
- The management team should understand the cause of upper extremity spasticity in order to formulate an optimal treatment plan.

INTRODUCTION

Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (ie, muscle tone) with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome (UMNS).[1–4] Upper motor neurons originate in the motor region of the cerebral cortex or brain stem and carry information to the lower motor neurons, which innervate skeletal muscle. Damage to the upper motor neuron results in several clinical findings that encompass the UMNS. Some of the positive features include spasticity and dystonic hypertonia, hyperreflexia, spasms, and clonus, whereas negative features include paralysis, weakness, loss of dexterity, and muscle fatigue.[4]

Data regarding the incidence and prevalence of spasticity are scant, but the clinical impact is undeniable. Many patients with spasticity require life-long medical management and substantial assistance with activities of daily living. Upper extremity surgeons may be involved in the management of patients with spasticity in order to address joint deformities and functional deficits. In this article, the authors discuss the epidemiology and pathophysiology of the most common causes for upper extremity spasticity.

PATHOPHYSIOLOGY OF SPASTICITY

The upper motor neuron pathways originate in the brain stem or cerebral cortex. These pathways include the corticospinal (pyramidal) tract; these tracts, along with descending pathways originating in the brain stem, can directly or indirectly influence the excitability of the anterior horn cell.[4] All of these structures may play a role in the pathophysiology of positive symptoms of UMNS.

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The fundamental disruption leading to spasticity is in the muscle stretch reflex,[5] as evidenced byan increase in resistance during passive stretchor movement of a joint. Initial paralysis followedby aberrant motor behaviors, such as spasticity, is a result of the adaptive changes of the brain andspinal cord after damage to centralmotor pathways.The correlation between spasticity and paralysis is clinically relevant, as each manifestation results in some form of functional impairment anddisability. Recognition of the simultaneous findingsof spasticity and paralysis is also important, as they require different treatment strategies.

There is no single pathophysiologic mechanism that accounts for all aspects of spasticity. Paresis, soft tissue contracture, and muscle hypertonia are the 3 major mechanisms of motor impairment. Further, several conditions are part of spastic hypertonia, including dystonia, rigidity, myoclonus, muscle spasms, clonus, posturing, and spasticity.[6] Clinically, isolated stretch-related spasticity will be velocity-dependent and able to be tested with passive stretches. It is often assessed with examiner-dependent tools, such as the Modified Ashworth Scale, that reliably rates resistance to passive movement on a 5-point scale.[6] If left untreated, spasticity may evolve into muscle and joint contractures with limited motion and loss of function. The most common clinical manifestations are elbow flexion, forearm pronation, wrist flexion, and thumb/digital flexion (Fig. 1).[7,8] Optimal management of these deformities requires an understanding of the underlying cause of upper extremity spasticity and is highly individualized.

EPIDEMIOLOGY OF SPASTICITY

Upper extremity spasticity may result from several different conditions. Whereas there has been a significant amount of research into the treatment of clinically significant spasticity, the studies on the incidence and prevalence of spasticity have been limited. Most of these studies rely on patient surveys. In addition, there are differences in clinical assessment measures and diagnostic definitions,[9] which make it difficult to estimate the prevalence of spasticity.[10] Despite these limitations, existing research indicates that 17% to 38% of patients with a cerebrovascular accident (ie, stroke), 34% of patients with traumatic brain injury (TBI), 67% of patients with multiple sclerosis (MS), 68% to 78% of patients with spinal cord injury (SCI), and 85% of patients with cerebral palsy (CP) have spasticity.[10–20]

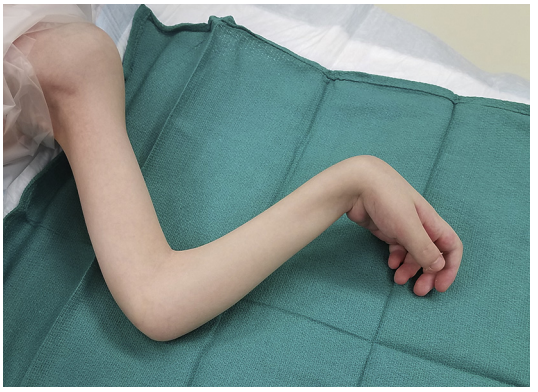

Fig. 1. Typical clinical manifestations in patients with cerebral palsy.

CAUSES OF UPPER EXTREMITY SPASTICITY

Cerebrovascular Accident (ie, Stroke)

A stroke is the sudden onset of neurologic deficits secondary to an acute decrease in blood flow and resultant brain hypoxia. It is a major cause of morbidity and mortality worldwide and ranks as the second-leading cause of death behind ischemic heart disease. Nearly 800,000 new cases are reported annually in the United States, where approximately 2.6 million men and 3.9 million women live with stroke.[21] Spasticity affects up to 38% of patients following a stroke.[10,11,14,18,20] Further, it is the number one cause of paralysis in the United States.[22,23]

The acute decrease in brain perfusion can be caused by 2 different mechanisms: occlusion of blood vessels (ie, ischemic stroke) and blood vessel rupture (ie, hemorrhagic stroke).[24] Hemorrhagic strokes are much less common and may be caused by hypertension, aneurysm rupture, arteriovenous malformation, anticoagulants, or tumor bleeding. The neurologic deficits present in patients who have had a stroke will depend on the area of the brain affected.[25] For example

- Unilateral ischemic injuries secondary to occlusion of the *middle cerebral artery* will result in contralateral hemiplegia. Upper extremities will be more affected than lower extremities. This lesion results in the classic clinical picture of upper extremity hemiplegia, facial palsy, and speech difficulties.
- Unilateral ischemic injuries secondary to occlusion of the *anterior cerebral artery* will result in contralateral motor deficits to the lower extremity, with little to no effect on the contralateral upper extremity.
- A central lesion to the *posterior cerebral artery* can cause contralateral hemiplegia.
- Occlusions of the *basal ganglia* result in contralateral hemiparesis.