Special Considerations in Assessing and Treating Spasticity in Spinal Cord Injury

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KEYWORDS

• Spasticity • Assessment • Treatment • Management • Spinal cord injury

KEY POINTS

- The effects of spasticity after spinal cord injury may be easier to understand when looking at functional neurologic levels.
- The assessment of spasticity should include both subjective descriptions from the individual and objective findings of the examiner.
- Spasticity treatment should be guided by the presence or absence of problems/difficulty stemming from the effects of spasticity on the individual.

SPINAL CORD INJURY AND SPASTICITY

Spinal cord injury (SCI) is a condition that affects the spinal cord either by trauma or intrinsic pathologic conditions, such as multiple sclerosis or compressive epidural abscess. As the SCI has multi-organ sequelae, one of the more functionally disabling consequences is the compromise onto the musculoskeletal system. When the corticospinal tract is affected, upper motor neuron syndrome ensues. The incidence of spasticity affecting persons with SCI may approach 80%.[1] The incidence is affected by timing after injury as well as level and severity of injury. In general those with tetraplegic SCI report more difficulties with spasticity.[2] Those with incomplete tetraplegia have the highest incidence of spasticity.[3] Although spasticity can affect activities of daily living (ADLs) in individuals with SCI[4] with an associated decrease in quality of life[5] in no small part due to pain and insomnia for some,[6] it has also been observed to decrease life expectancy.[7]

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Many define spasticity as "a motor disorder characterized by a velocity-dependent increase is tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome."[8] Others may ascribe to a modified definition, whereby various components of spasticity are separated into subdefinitions: "(1) intrinsic tonic spasticity: exaggeration of the tonic component of the stretch reflex (manifesting as increased tone), (2) intrinsic phasic spasticity: exaggeration of the phasic component of the stretch reflex (manifesting as tendon hyper-reflexia and clonus), and (3) extrinsic spasticity: exaggeration of extrinsic flexion or extension spinal reflexes."[9]

Special considerations in the pathophysiology of spasticity in SCI should be noted. As with other causes of upper motor neuron syndromes, the spinal reflexes below the neurologic level unaffected by injury are intact. However, at the level of injury, affected motor neuron and spinal roots may lead to denervation of corresponding muscles and consequent flaccidity. That is, at the affected spinal segments, spinal reflexes may be abated and spasticity mitigated. For incomplete injuries, at the level, the muscles may be unaffected with volitional activity or partially affected with some mild tone. However, depending on the muscles involved, the volitional muscles, although unaffected by increased tone, may be functionally affected by the antagonist muscle having unopposed and uncoordinated muscular hyperactivity. For example, elbow flexion by biceps brachii and brachialis muscles may be impeded by spasticity of the elbow extension muscle of the triceps.

For persons with SCI, spasticity may have both a positive and negative impact on their lives. Incomplete injuries, with some preserved sensations, may experience spasticity as uncomfortable and painful.[10] It is commonly noted that spasticity may adversely affect ambulation. Additionally, a decrease in daily activity functional independence, especially in transfers, is commonly observed.[4] Nonintuitively, spasticity has been observed to have either no impact[11] or a negative impact on bone mineral density preservation for persons with SCI[12] just as hemiplegic stroke survivors.[13] Similarly, arterial insufficiency is noted with concordant limb spasticity.[14]

Although nocturnal spasticity is often reported, it can generally be secondary to 3 causes. Nocturnal spasticity that occurs when a person first goes to bed into the supine or recumbent position is primarily due to extension of the flexed muscles, for example, hip flexors, from the sitting to the supine position, exciting the reflex arc. This spasticity often triggers a flexor response and is often transient, although recurrent, depending on the body position in bed and has been noted to correlate with insomnia.[6] For those with the inability to spontaneously reposition different parts of the limbs, a second contributing factor to increased nocturnal spasticity is prolonged recumbency in one position leading to otherwise undetectable discomfort, that is, increased nociceptive stimuli, resulting in increased reflex activity and spasticity. Shorter-acting half-life of medications, such as baclofen and tizanidine, may reach subtherapeutic levels by mid to late portions of sleep with manifestations of increased spasticity hours before completion of intended restful sleep. Lastly, subjectively reported spasticity refractory to all attempts at antispasmodic medication titration may need to have a differential diagnosis broadened to include considerations for periodic limb movements (Levy).

On the other hand, spasticity has beneficial effects for persons with SCI. Muscle mass maintenance is noted with those with increased spasticity.[11] By association, there is noted improvement in lipid profile and glucose metabolism with increased spasticity.[15] Interestingly, although spasticity has been noted to improve standing posture for some,[2] others use spasticity as sentinel for tissue injury or pathological condition below the neurological level, such as urinary tract infection, stool