Myth # 1: The definition of spasticity as presented by JW Lance (1980) has been proven to be valid.

According to Lance (1980), spasticity is "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ("muscle tone") with exaggerated phasic reflexes, such as tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome."

One element in this definition that compromises clarity is the parenthetical ("muscle tone"), which is actually defined as the degree of muscle tension during rest or the degree of resistance to passive stretch at any velocity.¹

A group of clinicians who called themselves SPASM spent three years investigating the literature on the issue of spasticity and its assessment and management. In one of their publications led by Pandyan (2005), they suggest that if Lance’s definition of spasticity is valid, then it should be possible to demonstrate the truth of the following four resulting myths. I have contributed to the evidence that the four myths lack factual certainty:

1) The increased muscle activity seen on EMG during the imposed stretching phase results exclusively from increased activity in the stretch reflex pathways - often described using the terms “hyperexcitable” or “exaggerated”.

   - One of the main features of physiologic, nonreflexive soft tissue adaptation in calf muscle contracture is the stiffening and proliferation of a connective tissue matrix throughout the perimysium and endomysium of the shortened muscle. Peripheral nerves and blood vessels are contained within neurovascular tracts that are connected to the periphery of muscles along most of their belly lengths. Each neurovascular tract has branches through which the major blood vessels and the collagen fiber reinforced nerves enter the muscle at specific locations. The intramuscular connective tissues are the intramuscular part of the neurovascular tract since they also embed nerves as well as blood and lymph vessels (Yucesoy et al 2007).

   Peripheral nerve entrapment and the formation of peripheral nerve cell adhesion molecules have been reported in transformed muscle that is considered to be "spastic" (Barber et al 2011, Booth et al 2001, Butler DS et al 1991, Castle et al 1979, Huijing 2007).

   - Frascarelli et al (2005) performed nerve conduction and needle EMG tests in 29 patients with "spastic" CP and severe limb deformities, 11 of whom showed abnormal nerve conduction in 32 of 400 sensory or motor nerves, indicating one or more entrapment neuropathies. Severe joint contractures and deformities can cause nerve damage, possibly as a result of the stretching, angulation, or compression mechanisms in the anatomic fibro-osseous passages where nerves are particularly susceptible.

   - Stecker et al (2011) state, “Clinically, the presence of spontaneous EMG activity is one of the factors used in determining when there is a significant injury to a nerve.” p. 7 They add, “The presence of EMG activity mainly occurred during stretch at the higher force levels and during recovery after a severe [nerve] stretch injury.” p. 8

2) The increased muscle activity during imposed stretching will contribute to an increase in resistance to passive movement.

   - Several studies indicate that it is not possible to support this statement with confidence. “Although one would expect to be able to measure reliably the muscle activity contributions to stiffness from tonic stretch reflex activity (this will probably involve long latency polysynaptic pathways), it is surprising to note that it has also not been possible. The key confounding factors are likely to be inertial components from the limb segments, changes in the visco-elastic properties of soft tissues and joints, abnormal voluntary muscle activity, abnormal involuntary muscle activity resulting from phenomena other than stretch reflex hyperexcitability and the patient’s cognitive and/or perceptuomotor abilities.... spasticity-related muscle activity may contribute to increased joint stiffness. However, under routine clinical or research conditions, the exact relationship between spasticity-related muscle activation and increased stiffness is yet to be modelled reliably.” p. 95

3) **Velocity dependent increase in the resistance to passive movement is exclusive to spasticity.**

- Abundant evidence refutes this myth. The velocity-dependent change in stiffness is a characteristic response of soft-tissue structures (e.g. muscles, tendons, ligaments, etc.) and their normal viscoelastic properties.

4) **Spasticity is a pure ‘motor disorder’.**

- Though spasticity is an abnormal motor phenomenon, current evidence indicates that it would be wrong to treat it as a pure motor disorder. Stretch reflex activity is influenced by activity in cutaneous and proprioceptive pathways – as is shown in the beneficial effects of electrical stimulation and lycra garments – and is modulated by the higher centers in the nervous system. It is also possible that disordered feed-forward, corticospinal modulation of reflex activity, under both active and passive conditions, may also contribute to spasticity.

**At this time, it is clear that spasticity is not:**

- A movement disorder. It is hyperreflexia. The movement disorder is due to corticospinal dysfunction with impaired reciprocal inhibition.
- Distinctly evident in EMG activity observed during rapid passive elongation of resting muscles and soft tissues.
- The source of soft-tissue stiffness – diminished extensibility due to physiologic transformation and peripheral nerve adhesion following a history of tonic recruitment in shortened state.

**References:**