Spasticity and stroke: pathophysiology and management rules

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Introduction: Stroke is a common cause of morbidity and mortality. In fact, it is the leading cause of disability in the elderly. Spasticity occurs in up to 27% of patients in the acute phase of stroke and in up to 46% three months after the event.

The Central Nervous System (CNS) and intentional movements: The CNS is a coupling of increasingly complex systems regulated by themselves. Higher centres usually command lower centres, which, in turn, control the automatic and primitive behaviours, postural and tonic reflexes, associated reactions and balance.

Spasticity: Spasticity is an increase in tone (elastic hypertonia) that arises from pyramidal tract/upper motor neuron (UMN) injury, by the absence of its inhibitory action on the spinal reflexes and by a low threshold for myotatic activity. Lance (1980) put forward the most frequently accepted definition: spasticity is a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex. Spasticity is not only velocity-dependent, but it is also length-dependent. For example, in the quadriceps, the tonic stretch reflex is greater when the muscle is short, than when it is long. It is indeed a component of the UMN syndrome (UMNS), but functional limitations after stroke are more closely related to overall neurological deficits, other than spasticity. The UMNS includes positive signs, such as spasticity, hyperreflexia, clonus, muscle spasms, Babinski reflex, synergistic movement patterns, antagonist-agonist co-contraction, spastic dystonia and associated reactions; and negative signs, such as muscle weakness, dexterity decrease and fatigability.

How does an UMN lesion cause spasticity? Each patient has injuries that affect different routes to varying extents, leading to adaptations in neural networks of the spinal cord. However, in all of them normal cortical-spinal tract functioning is absent. Different spinal mechanisms, such as membrane potential, reciprocal inhibition and presynaptic inhibition, may have different roles in different patients. Spasticity is likely to be caused not by a single mechanism, but by a complex chain of interrelated changes in different networks. Changing the balance between the above inhibitory spinal routes and excitatory pathways in the spinal cord, leading to a disinhibition of the stretch reflex. Spasticity appears only days to weeks after a central neurological injury. This delay between the acute lesion and the onset of spasticity is more than a phenomenon of mere disinhibition and suggests plastic changes in the CNS. An initial period of shock is followed by a transition period with the return of reflexes, not yet hyperactive. There appears to be a rearrangement, corresponding to neuronal plasticity in the spinal cord, and probably in the brain. Afferent fibres can grow and transform inhibitory and excitatory synapses. There is denervation hypersensitivity due to the upregulation of receptors. Furthermore, changes
in the rheological properties and in the contractile soft tissue and musculoskeletal system (intrinsic hypertonia) are often associated with chronic spasticity and, in turn have been associated with increased spasticity.

**Signs and symptoms of spasticity:** Spasticity signs include muscle stiffness or spasm, muscle spasms, clonus, pain, difficulty performing voluntary movements or deformity of the limbs (cosmetic or functional concerns). Spasticity symptoms include the resistance to passive movement, twitching, co-contraction of agonist and antagonist muscles, spastic dystonia, decreased range of passive movement, abnormal posture and/or limb deformity.

**Spasticity pattern of cerebral origin:** This pattern observed after a stroke differs from spinal-origin spasticity as found in spinal cord injury and in multiple sclerosis. Cerebral-origin spasticity is characterized by a postural stereotype involving antigravity muscles: the upper limb presents a flexor pattern: depression of the scapula, internal rotation and adduction of the shoulder, forearm pronation, elbow, wrist and fingers flexion; the lower limb presents an extensor pattern: extension, adduction and internal rotation of the thigh, knee extension, plantar flexion and foot inversion. Spasticity is often classified according to the distribution of the affected body areas as focal, multifocal, regional or general. It is important to identify the distribution of spasticity since it has definite implications for treatment.

**Rules of spasticity management:** Spasticity does not always need to be treated. In fact, it may aid the patient to walk or perform other activities of daily living (ADL), maintain muscle mass and bone mineralization, and decreased oedema and the risk for deep vein thrombosis. However, it can interfere with mobility, exercise and range of motion, reducing the support and swing of gait and lead to contractures. It can also interfere with ADL and patient care, including hygiene. Moreover, pressure sores and sleep disturbance occur and can cause pain. In the treatment of spasticity, we may consider factors such as a chronical status, spasticity severity and distribution, location of the central lesion, patient comorbidities and caregiver availability. Treatment of spasticity should include three major classes of goals: technical (increasing the range of motion, reducing the tone or reducing spasm), functional (improving ADL, reducing pain, facilitating care, improving limb positioning and gait), and preventive (preventing contracture, skin maceration, and skin ulcers). Spasticity treatment should be performed by a multidisciplinary team that includes physiatrics, neurologists, nurses and caregivers, therapists, and should be always based on the person as a whole.