INTRODUCTION

Spasticity is a common complication after spinal cord injury (SCI) as a sign of damage of upper motor neurons. Spasticity may develop months or years after the acute injury and lead to increased loss of function and hospitalization [1,2].

The characteristics of the spasticity after SCI will be described in two parts: the first one (part 1) presents general and specific characteristics of the spinal spasticity with its clinical manifestations and repercussions and the way of assessing them; in the second one (part 2) the treatment will be described.

The most commonly cited definition of spasticity was created by Lance in 1980: "Spasticity is a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one component of the upper motoneuron syndrome" [3]. Nevertheless, this definition has limitations because the observed features of spasticity do not exclusively result from hyper excitability of the stretch reflex nor are they all velocity dependent like clonus, spasms or contractions of agonists and antagonists [4]. For this reason, the Ability Network (an international panel of clinical experts for developing management algorithms to guide and standardize the assessment, treatment, and evaluation of outcomes of persons with spinal cord damage and disabling spasticity) recommends adoption of the definition by Pandyan: "disordered sensory-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles" [5,6].

The prevalence of spasticity is about 65% of people with SCI [1]; during inpatient rehabilitation incidence is only 25-37% in traumatic SCI and 15-36% of non traumatic SCI [7-10], and in chronic SCI patients (more than 1 year post injury) spasticity is present in 65-93% [1,2,11,12]. Problematic spasticity has been associated with cervical levels and severity of the injury according with American Spinal Injury Association Impairment Scale as grade A though patients with grade C injuries had the highest prevalence of ongoing spasticity treatment and functional limitation [11,12].

PATHOPHYSIOLOGY OF SPASTICITY

Spasticity is generally due to a lowered threshold of phasic or tonic stretch reflexes; when inhibitory signals are lost due to spinal cord damage the segmental reflexes are released and become hyperactive [1].

The classical motor unit path is formed by lower motoneurons of 2 types: alpha motor neuron that projects to extrafusal skeletal fibers, and gamma motor neuron that projects to intrafusal muscle fibers within the muscle spindle [11,13]. The stretch reflex is a monosynaptic reflex that originates in the muscle spindles when there is any stimulus (not only stretching) and travels via a la afferent to the spinal cord where it synapses with correspondent alpha motor neuron (directly or via another interneuron) and finally contracts muscle fibers where stimulus has been produced. If the stimulus goes on too long, Golgi tendon organs can be elicited, and via lb interneuron’s can relax previous contraction. The hyper excitability of stretch reflex can explain spasticity after SCI [11,13].

The mechanisms underlying spasticity and its consequences can be classified in 2 groups:

- Alterations in spinal mechanisms [11,13,14]. Enhancement in the excitability of motoneurons and interneurons’ are most likely involved in the pathophysiology of spasticity following SCI. This enhancement is attributed to depolarizing current that does not inactivate with prolonged membrane depolarization (persistent inward currents) whose activation is regulated through the monoaminergic drive from the brainstem, mediated on the other through neurotransmitters like serotonin or nor epinephrine. Other mechanisms are fusimotor hyper excitability, axonal sprouting, reduction in presynaptic inhibition and
reduction in Ia- reciprocal inhibition. Induction of plateau potentials has been demonstrated as a very important mechanism developing spasticity in animal models but not in humans [15]. These mechanisms have a neurophysiologic correlation: the H max (maximum amplitude of an H reflex)/ M max (maximum amplitude of the compound muscle action potential) ratio shows the percentage of motor neurons that can be reflex activated by an electrical stimulus delivered to a nerve, being greater than normal in upper motor neuron lesions (greater still when spasticity occurs) and lower than normal in lower motor neuron lesions or spinal shock [16], though this increase in the ratio may take up to 2 to 6 months from the time the injury occurs, while recovery of F-waves and flexor reflex excitability appears at the end of spinal shock and may increase as spasticity develops [17]. Clinical features of spasticity depend on the greater or lesser loss of the ability to voluntarily modulate the level of activity of a given motor pool and the capacity of the interneuron's project to these motor pools to recruit the appropriate combination of motor pools [18].

- Plastic alterations in affected muscles [11]. Spasticity causes fibrosis, atrophy of muscle fibers, decrease in the elastic properties, decrease in the number of sarcomeres, accumulation of connective tissue, and alteration of contractile properties toward tonic muscle characteristics. Muscle shortening causes contractures, and then the capacity for development of active tension is less than that normally possible at longer lengths.

CLASSIFICATION OF SPASTICITY

In general, spasticity can be classified according to 2 criteria [11,19]:

- Place where located stimulus for spasticity is allows differentiating between intrinsic if stimulus emerges within Central Nervous System, and extrinsic if afferent input proceeds from other structures such skin, subcutaneous tissues, muscles or joints.

- Components of the stretch reflex. If there is no resulting movement tonic or static component is described, and if there is any movement phasic or dynamic component is described. The concept of tonic spasticity is the one that best agrees with the classic definition of spasticity.

So, we can distinguish 3 main types of spasticity [4,13,19,20]:

a. Intrinsic tonic spasticity. Hypertonia is an involuntary increase in resistance of muscle to passive stretch velocity-dependent.

b. Intrinsic phasic spasticity. Clonus is defined as an involuntary rhythmic muscle contraction that can result in distal joint oscillation, and it can be result of the sudden application of sustained stretch to a muscle too. Tendon hyper-reflexia is identified as an exaggerated muscle response to an externally applied tap of deep tendons.

c. Extrinsic spasticity. Spasms are involuntary and abrupt muscle contractions. Although one type of spasm, flexor or extensor usually predominates, they can coexist in the same patient simultaneously.

CLINICAL FEATURES OF SPASTICITY AFTER SPINAL CORD INJURY

Time - course of developing spastic impairments following injury

When a spinal cord injury comes abruptly (as occurs in traumatic or vascular injuries) there is an initial phase called spinal shock. Spinal shock is defined as temporary loss of spinal reflex activity occurring below a SCI and implies flaccid paralysis and loss of deep tendon reflexes below the level of the cord injury [21]. In first 3- 4 weeks following acute spinal transaction, the intrinsic properties underlying motor neuron excitability start to return [22,23]. If spasticity appears we can say spinal shock is over but there is no relationship between first signs of spasticity and time since injury beyond the spinal shock period [11].

As spasticity is part of the upper motor neuron syndrome, it cannot occur when the SCI feature is like a lower motor neuron syndrome. The anatomical segment above the conus medullar is named the epiconus, consisting of spinal cord segments L4-S1; lesions in this zone affect lower lumbar roots with sparing of reflex function of sacral segments. According with the International standards for neurological classification of spinal cord injury [24], neurological levels of injury above T10 always present as an upper motor neuron syndrome, and for that reason people with these injury levels are most likely to develop spasticity when their spinal shock is over, while people with injury levels below L3 do not develop spasticity [25,26]. Those patients with injury levels between T11 and L2 usually present with an upper motor neuron syndrome.

Differences between spasticity of cerebral and spinal origin

When spasticity is of spinal origin it has several specific characteristics among which the following stand out [1,27- 30]:

- Acute flaccid phase previous to spastic period is longer in patients with spasticity of spinal than cerebral origin.

- Most frequent presentation of spasticity when origin is spinal is generalized and diffuse while focal spasticity is more prevalent when origin is cerebral.

- As for spastic hypertonia, patients with SCI develop a more intense spasticity than those with a stroke or a brain injury, and clasp- knife phenomenon is more prevalent when origin is spinal. The muscles most commonly affected in SCI are extensor especially in lower limbs, while cerebral spasticity predominates in flexor muscles.

- As for intrinsic phasic spasticity ankle clonus is more prevalent when origin is spinal, and patellar clonus is rare if origin of spasticity is not cerebral.

- Extrinsic spasticity is more frequent in SCI patients; extensor spasms in lower limbs are the most prevalent spastic feature among people with SCI. Most important stimulus is hip extension (especially last 20 degrees), and most important receptors are hip mecanceptors; within cerebral origin spasticity most important receptors are knee mecanceptors.

- Influence of posture. SCI patients show shortening of
muscles due to prolonged postures not considered as spasticity, the most frequent affected muscles are hamstrings that are shortened by keeping the sitting in the wheelchair. However, stroke patients can maintain a posture due to spasticity when they develop spastic dystonia, whose principal example is elbow flexion posture.

- Spinal spasticity is more exacerbated by visceral diseases.

**Triggering factors**

The most important factors are posture and bowel and bladder issues [31]. The presence of these exacerbating factors can produce that a patient with a SCI and normal or decreased tone can present with intense or frequent spasms.

Supine position is associated with a more intense hypertonia and greater ease of causing spasms than sitting, the most frequent situation related by patients with SCI to suffer spasms is during transfers (above all transfers to bed) [32,33]. There is an activation of involuntary contractions in high soleus and tibialis anterior during transfers from wheelchair to bed [3,4].

Bowel issues are related with spasticity, both the constipation maintained and the moments of evacuation and faecal impact; the former are related with hypertonia and the others with dynamic symptoms [35,36].

Most frequent bladder issues related with spasticity are detrusor hyperactivity, urinary tract infection or obstructions [35,37].

Other factors like pregnancy, cold, circadian rhythm, skin conditions (pressure sores, ingrown nails), menstrual cycle, mental stress, and tight clothing also increase. Acute, serious infections (sepsis) and syringomyelia may cause both increased spasticity and a sudden absence of spasticity [2].

**Clinical influence of spasticity on the SCI patient**

Spasticity interferes in the patient causing functional disadvantages but can also provide some advantages. Spasticity can limit the ability for positioning, transfers, mobility, basic activities of daily living and can interfere with sleep, occupation and social participation. Disabling spasticity can also lead to fatigue, pain, increased risk of pressure ulcers and infections, and all of these effects can increase the number of primary care visits and hospitalizations [6,11]. For those reasons there can be a negative self-image and a decreased quality of life [38]. However, sometimes spasticity may increase stability in sitting and standing, facilitate the performance of some activities of daily living and transfers, increase muscle bulk and strength of spastic muscles (thereby helping prevent osteopenia), and increase venous return diminishing the probability of deep vein thrombosis [11], and muscle contraction by spasticity can accelerate fracture healing [39].

**ASSESSMENT OF SPASTICITY AND ITS CONSEQUENCES**

The International Spinal Cord Society (ISCOS) provides recommendations on how to assess various groups of symptoms, collected as basic dataset, but spasticity does not have a specific item and is only mentioned in the dataset corresponding to musculoskeletal disorders [40].

**Differential diagnosis**

The first point is to make a correct diagnosis of spasticity. We must distinguish spasticity from other tone disorders; the resistance to passive movement found in extrapiramidal rigidity, paratonia or simulation are not velocity-dependent [1,20].

**Measurement of tone**

Usually the severity of spasticity is identified with the intensity of the tone, commonly measured with the Modified Ashworth Scale (MAS, Table 1) [41]. MAS is considered as the most adequate way to measure the tone despite having demonstrated in some studies problems of reliability [1,11]; being a likert-type scale may have problems of validity but its reliability and inter-rater and test-retest agreement have been reported [42,43]. Tardieu scale is based upon the pendulum test; it consists of performing stretching of a muscle between 2 points at 2 speeds. It adheres more closely to Lance’s definition of spasticity and some studies have identified the Tardieu Scale to be more sensitive than other measures to change following treatment with botulinum toxin [44]. Inter-rater and test-retest agreement for its scores is substantial to almost perfect but inter-rater reliability of some measures for the hip adductor and knee extensor muscles are poor; these facts suggest its utility as a complementary tool for informing treatment decisions in patients with SCI, keeping MAS as the main scale for tone assessment [45].

**Assessment of dynamic phenomena**

Although there is no correlation between the frequency and intensity of the phasic symptoms and the evolution of spasticity, they are collected in the physical examination [43]. Frequency and intensity of spasms can be measured, but only frequency measure is validated through Penn Spasm Frequency Score (Table 2) [46]. There is a specific scale for phasic components assessment in SCI: Spinal Cord Assessment Tool for Spastic Reflexes (SCATS, Table 3) [47], that has a great inter-rater agreement and even shows a significant correlation between SCATS clonus scores and MAS scores for lower limb joints [48].

**Functional assessment**

Goniometric measurements are performed to evaluate results of contracture or loss of motion. Neuropsychologic assessment is not correlated with spasticity [27]. The interference of spasticity with activities of daily living can be measured with general scales like Functional Independence Measure [49] or specific scales for SCI like Spinal Cord Independence Measure version III [50]. Ability for walking can be also measured with general scales such as theFunctional Independence Measure version III [50].

<table>
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<th>Table 1: Modified Ashworth Scale.</th>
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<tr>
<td>0. No increase in tone.</td>
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<td>1+. Slight increase in tone with a catch, followed with minimal resistance throughout the remainder (less than half) of the ROM.</td>
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<tr>
<td>2. Marked increase in tone through most of the ROM, but limb is easily moved</td>
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<tr>
<td>3. Considerable increase in tone; passive movement difficult</td>
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<td>4. Limb rigid or contracted</td>
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Table 2: Penn Spasm Frequency Score.
0. No spasms.
1. Mild spasms brought on by stimulus
2. Infrequent spasms (occurring < 1 time/ hour)
3. Spasms occurring > 1 time/ hour
4. Spasms occurring > 10 times/ hour

Table 3: Spinal Cord Assessment Tool for Spastic Reflexes.
0. No reaction
1. Mild: clonus < 3 seconds
2. Moderate: clonus lasts between 3 and 10 seconds
3. Severe: clonus > 10 seconds
Flexor spasms in response to pinprick on foot plantar surface with leg and hip in full extension
0. No reaction
1. Mild: extension of great toe or <10 degrees of hip/ knee flexion
2. Moderate: 10 to 30 degrees of hip/ knee flexion
3. Severe: >30 degrees of hip/ knee flexion
Extensor spasms of quadriceps muscle after extension of leg from a position of hip/ knee flexion
0. No reaction
1. Mild: spasms last < 3 seconds
2. Moderate: spasms last between 3 and 10 seconds
3. Severe: spasms last > 10 seconds

Table 4: Spinal Cord Injury Spasticity Evaluation Tool.
-3. Extremely problematic
-2. Moderately problematic
-1. Somewhat problematic
0. No effect.
1. Somewhat helpful
2. Moderately helpful
3. Extremely helpful
Evaluation of spasticity effects on components of daily life over a 7 days period: showering, dressing/ undressing, transfers, sitting positioning, preparation of meals, eating, drinking, small hand movements (writing, use of computer), household chores, recreational activities, enjoyment of social outings, standing/ weight- bear, walking, stability/ balance, muscle fatigue, flexibility of joints, therapy/ exercise routine, manual or power wheelchair use, lying positioning, change positions in bed, getting to sleep, quality of sleep, sex life, annoyance feeling, embarrassing feeling, social comfort, physical comfort, pain, concern with feeling, concern with getting injured, concern with accidentally injuring someone else, concentration, control over one’s own body, need for help.

Table 5: Patient Reported Impact of Spasticity Measure. Evaluation of spasticity effects on life experiences over a 7 d. period.
0. Never true for me.
1. Rarely true for me.
2. Sometimes true for me.
3. Often true for me.
4. Very often true for me.

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Assessment of the impact on the quality of life
Interferences of spasticity with leisure, sleep, physical activity and quality of life can be measured with a general health scale like Short Form Health Survey- 36 [54], but specific scales for impact of spasticity on SCI patient’s quality of life are preferred. Spinal Cord Injury Spasticity Evaluation Tool (SCISET, Table 4) is a scale to know how the patient estimates spasticity to influence on 35 components of his life [55]. Patient Reported Impact of Spasticity Measure (PRISM, Table 5) evaluates not only influence of spasticity on SCI patient quality of life but effects of patient life on spasticity [56].

Subjective assessment
Usually patients can describe their spasticity with a number rating scale [6,11], but they can use SCISET or PRISM scales as self-administered inventories or even they can describe their spasticity through specific signs such the duration and intensity of involuntary agonist- antagonist muscle coactivity during everyday tasks [34].

Changes after therapy
Assessment of these changes will be addressed in the next part (Part 2), corresponding to the treatment.

REFERENCES
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