The Effects of Spasticity on Motor Recovery After a Stroke.

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Background and Aim: *The rehabilitation process oriented to motor recovery after a stroke is a complex process associated with the appearance of positive and negative signs after motor neuron damage. The aim was to describe the effects of spasticity in the rehabilitation process after a stroke.* ***Methods:*** *Three reviewers, exploring the PubMed database, carried out a narrative review through a search plan. The terms MesH: Spasticity AND Motor Recovery AND Stroke were used; the search limits were reviews and clinical trials in humans and animals, published in the last 10 years with full texts in English and Spanish.* ***Result:*** *Seventy-one articles that met the search criteria were identified, and 21 were selected (16 clinical trials and 5 reviews), which were analyzed through the CASPE guide and used for the preparation of this review.* ***Conclusion:*** *Although there is broad scientific evidence, it is not conclusive, stating that spasticity is a source of functional commitment and disability, but it is not always harmful, and its effects can be beneficial and sometimes do not need treatment.*

Keywords: *Spasticity, Rehabilitation, Motor Recovery, Stroke*

Introduction

The rehabilitative process focused on motor recovery, specifically on the upper limb (UL) after a stroke, is a complex process linked to the appearance of clinical signs of upper motor neuron symptoms (MNS). These signs, under the Intern ational Classification of Functioning, Disability and Health, are dealt with on the basis of bodily structures and functions, associated to phenomena known as tonic "positive signs", such as spasticity, hyperreflexia, and phasic, such as clonus and spams; and on the level of activity and factors related to the individual and the environment, where paresis, fatigue and loss of dexterity are identified as "negative signs" (Table 1)[1]. The balance between positive and negative signs produced by MNS damage in general defines the impact over motor recovery and the quality of life after a stroke[2].

Accepted: 07/01/2020
Received: 17/07/2019

The author does not have any conflict of interests to disclose.

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Table 1. Positive and negative signs of MNS damage.(1)

Positive signs	Description
Spasticity	Abnormal increase in muscle tone during movement, dependent on the speed of muscle stretching.
Spasms	Sudden and involuntary muscle contraction, which can manifest itself in a pattern in flexion or extension
Clonus	Series of rhythmic and involuntary muscle contractions, due to a self-excitation of hyperactive stretching reflexes in distal joints (ankle and wrist).
Hyperreflexia	Exaggerated increase in reflex function, both proprioceptive and cutaneous, due to a lack of inhibition of the descending pathways.
Co-activation Muscular	It consists of the involuntary and simultaneous contraction of groups antagonistic muscles, due to a decrease in reciprocal inhibition mechanisms
Babinsky positive	Reflex response, after rubbing the sole of the foot. Observing hallux extension while the other fingers flex
Associated Reactions	Remote form of synkinesia due to failure in inhibition of spread of motor activity.
Negatives Signs	
Muscular Weakness (Paresis)	Reduced capacity to generate and maintain voluntarily the force (or torque), sufficient for effective movement or the task performance.
Waste of skill	Loss of coordinated and precise movements in the human body
Fatigability	Refers to the feeling of increased effort to perform a task motorboat.

Gracies, 2005, poses that positive and negative signs in UL are present at the same time and, together, lead to positioning and/or movement dysfunction that is aggravated by secondary adaptive changes in soft tissues[3].

From the total of people who suffer a stroke, about an 80% experience motor-sensory impairment[4]. The main determinants of this impairment are paresis and the positive sign known as "spasticity"[5], defined as a motor disorder characterized by a velocity-dependent increase on tonic stretch reflexes as the result of an abnormal intraspinal processing of the primary afferents[6]. Its range of prevalence is estimated to be between 4% to 43%, with a disability incidence of 2% to 13%[7].

Although spasticity is a source of functional compromise and morbidity, it is not always detrimental and sometimes it does not require treatment[8], as some of its effects may be beneficial during the rehabilitative process.

The purpose of this review is to describe the mechanisms that intervene in spasticity and how these affect, positively or negatively, motor recovery after MNS neurological damage

Methodology

In order to address the study subject, a search plan was carried out by three reviewers, who explored the PubMed database using the MesH terms: Spasticity AND Motor Recovery AND Stroke. The search term limits were reviews and clinical trials in humans and animals, published in the last ten years with complete texts in English and Spanish.

A total of 71 articles that met the required search criteria were found; 55 corresponded to clinical trials and 16 to reviews, which were analyzed according to the CASPE guidelines for reviews and clinical trials[9]. Finally, 21 articles were selected (16 clinical trials and 5 reviews), considering pertinence, relevance, and currency of the subject, which were used as the basis of the making of this review.

Development

Spasticity, a highly variable phenomenon.

Spasticity may begin early during the first few weeks or a year after the stroke[10]. The reported rates are 24% during the first week [11], 19% at three months, 22% to 43% between four to six months[12], and 17% to 38% at twelve months[13].Electromyography studies show that increases in muscular tone reach its maximum between the first and third month after the stroke[14].

Spasticity is a clinical sign, with a presentation that may differ depending on the localization and extension of the brain damage[15]. The injury by itself does not predict the intensity nor the impact it will have on spasticity[16], as it may change within the same day or after longer periods of time. In fact, it may vary and increase in situation of diverse nature, both of physiological and psychological origin[17]. Its extension may be variable, from an affectation of only some anti-gravitational muscle groups, up to a global manifestation. This way, three types of compromise are recognized: focal, regional or segmental, and generalized.

Likewise, the spastic manifestation may appear with typical patterns in upper[18] and lower limbs, each with specific muscles[19], affecting primarily the elbow (79%), the wrist (66%) and the heel (66%)11. Currently, the Hefter patterns are proposed for the classification of UL. The Hefter patterns refer to five patterns that describe the position adopted by the articulations of the shoulder, elbow, wrist and fingers after MNS damage, with pattern No. 3 (Figure 1) being the most prevalent one, which positions the shoulder in internal rotation/abduction, flexed elbow, neutral forearm, neutral wrist and flexed fingers[18].

From a functional perspective, spasticity produces a deficit in the interarticular coordination of spastic muscles, affecting the interaction between the two upper limbs and[20] the force generated by a limb that cannot be clearly perceived by the contralateral spastic member [21].

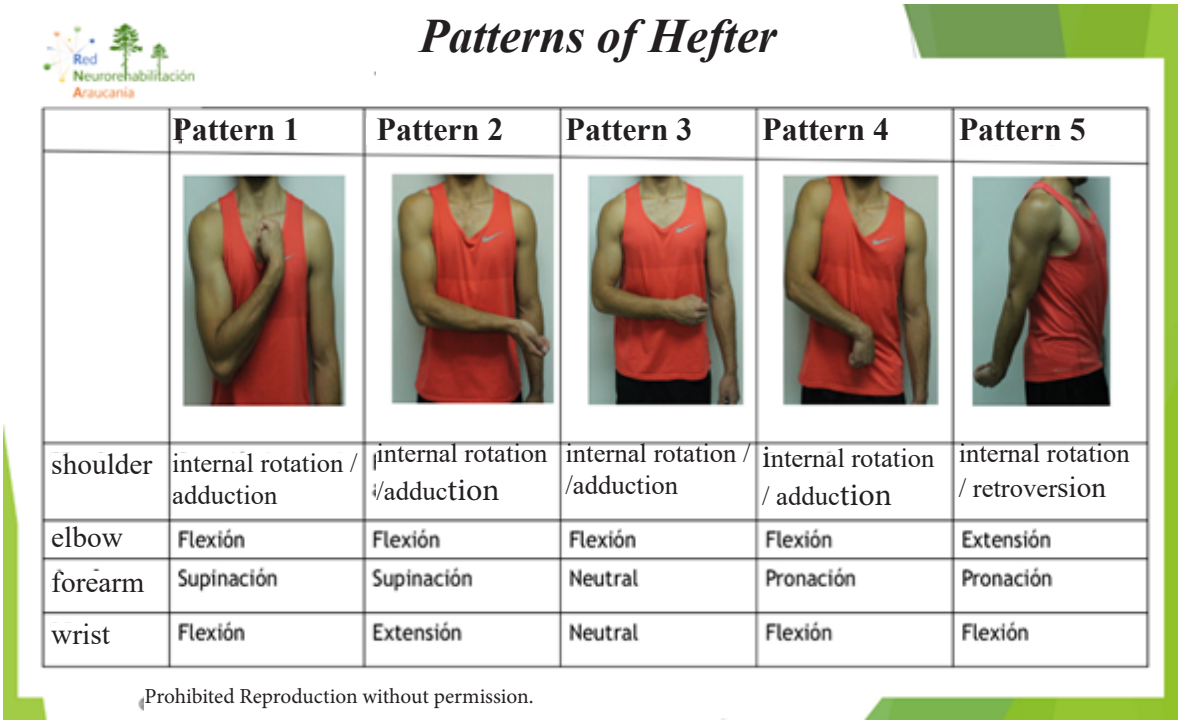
Although the scientific evidence is ample [22 23], there is still no consensus about the number of subjects who develop spasticity, as well as about their characteristics and determining factors.

Mechanisms that explain the phenomenon of spasticity

Spasticity does not constitute an acute sign, but rather a clinical characteristic that develops gradually and persists after a nervous system injury in an undefined manner, due to sprouts of undamaged nerve fibers, into the phenomenon of hypersensitivity by denervation and transsynaptic degeneration and the channeling of silent synapses, which leads to it being a plastic phenomenon[16. 24].

The literature proposes two models by which spasticity is explained from the physiopathological point of view.

Figure 1. Classification of "Hefter" upper limb patterns.



	Pattern 1	Pattern 2	Pattern 3	Pattern 4	Pattern 5
shoulder	internal rotation / adduction	internal rotation /adduction	internal rotation / /adduction	internal rotation / adduction	internal rotation / retroversion
elbow	Flexión	Flexión	Flexión	Flexión	Extensión
forearm	Supinación	Supinación	Neutral	Pronación	Pronación
wrist	Flexión	Extensión	Neutral	Flexión	Flexión

A spinal model: This model proposes three factors that result in an increase in the the muscle-spasticity tone, affecting the motorneuron through(1) changes in its afferent entrances, (2) in the reflex circuits that affect its excitability, and (3) in its intrinsic properties[25].

Despite these three mechanisms, only the loss of homosynaptic depression, referred as the decrease in synaptic efficiency between Ia afferent neurons and motorneurons due to a progressive reduction of neurotransmitters released by the pre-synaptic terminal, would be the factor that provokes this tone disturbance after a cortical damage [26].

Suprasegmental model: This model proposes that there is a disintegration of the mechanisms of suprasegmental control that regulate the spinal structures and the reflex arc involved in muscle tone.

The scientific evidence highlights the role of the dorsal and medial reticulospinal tracts, and of the spinal vestibule tracts through their inhibitory and down-regulating action over the stretch reflex[27]. An MNS injury that produces an imbalance of these downward influences, mostly of the TRED, would be the main cause of anomaly in the stretch reflex, and therefore, in spasticity[17].

Spasticity and the factors that regulate motor recovery

Motor recovery follows some relatively defined patterns, depending on the type and the extent of the brain damage, and also on how the positive and negative signs affect movement. Generally, this interaction has a negative impact in the motor functioning of the patient. Given the latter, it is essential for the rehabilitative process to include a team capable of discerning effectively the contribution of these positive and negative

signs within the process.

Longitudinal studies have proposed that motor rehabilitation has predictable stages during the first six months, regardless of the type of intervention[28]. During this period, there is a process of spontaneous recovery, which peaks at six weeks and then decreases at six months. Nevertheless rehabilitation protocols based on repetitive practice and enriched environments promote nervous plasticity and motor recovery in chronic stages (>1 year)[29 30].

Brunnstrom empirically describes seven states of motor recovery associated with tone disturbance (Figure 2), emphasizing three states: flaccid, spastic (emergent, evident, and decreasing) and total recovery (voluntary control of movement)[31].

During the evolution of an MNS syndrome, a subject may progress from a recovery state to another in a variable manner, but always in an orderly fashion and without omitting any stage. Nevertheless, recovery may be stopped in any of these stages, which is associated with rehabilitative opportunities, negative neuroplasticity and the psychosocial characteristics of the subject[32]. These stages of motor recovery and their relation with spasticity are used in clinical practice and were validated in the last few years[33].

As previously mentioned, the appearance of spasticity is highly variable, a phenomenon that is related to the changes of neural plasticity within the nervous system after the damage [25]. In this way, the neuronal plasticity provides a base for motor recovery[34]. This background shows that spasticity and motor recovery are mediated by different intrinsic and extrinsic mechanisms of the subject, the development of spasticity being a milestone in the course of recovery.

The effects of spasticity in motor recuperation

The impact of spasticity in recovery may be variable, and may not be identified in the acute stage. However, in sub-acute and chronic stages, spasticity is associated with hypertonia, where resistance to passive movement affecting UL is caused by adaptive changes in soft tissues, producing pain, tendon retractions and muscle weakness (being a non-neural factor caused by biomechanical changes) and associated positive signs (neural factor), such as spasticity, which in some cases may limit and in others facilitate the rehabilitative process[35].

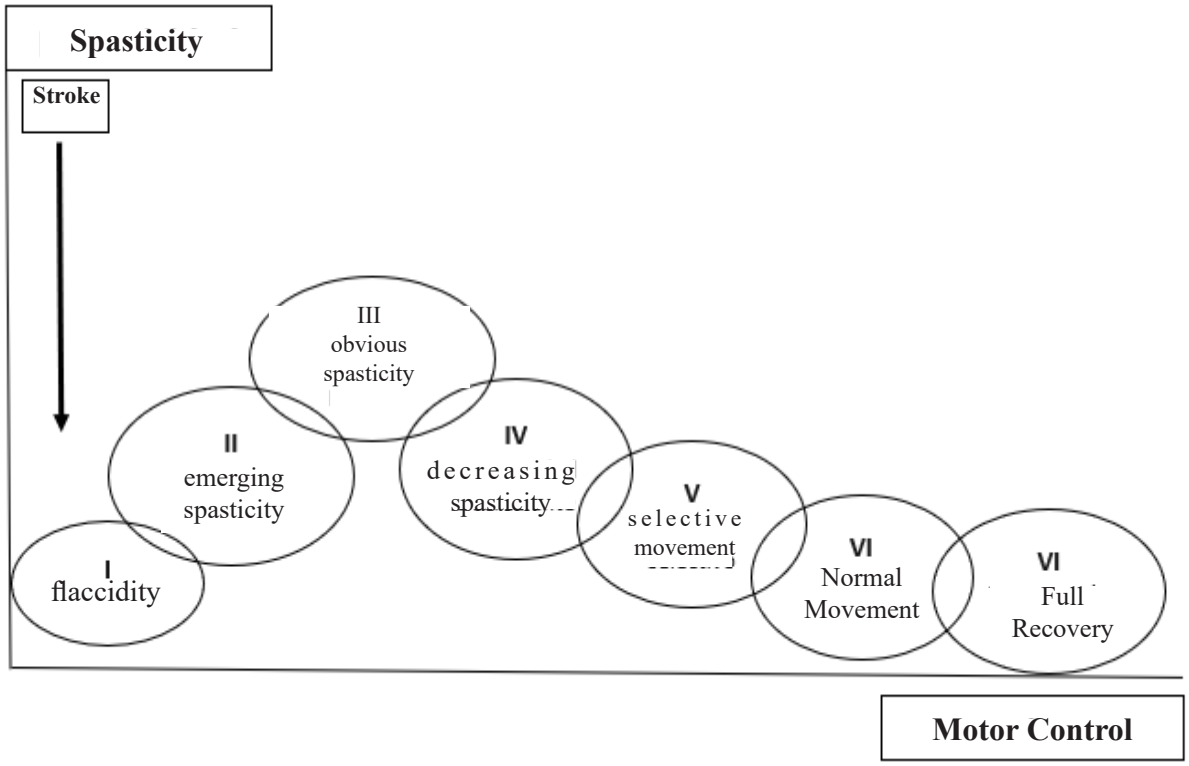
The abnormal increase in tone may result in an exaggerated static position, disuse, and muscular atrophy. By losing balance reactions and protections, and with paresis and fatigability[36] restricting the activities and participation of the subject, the situation generates a significant impact in the daily functioning and quality of life[37]. Increasing the direct costs of healthcare during the first year after the stroke[38].

Alibiglou, 2008, states that the consequences of spasticity may affect the work and productivity of the individual: 89% of the subjects report a total or partial inability to work as the result of their spasticity[39].

Although spasticity is a source of functional compromise and morbidity, it is not always completely harmful and sometimes does not need treatment8, because some effects could be beneficial, as, for example, in the quadriceps to stabilize the knee during the support phase and, with this, promote verticalization and gait rehabilitation. This aspect requires to take into consideration the final impact that spasticity has before looking for therapeutic strategies to reduce it[40].

The influence of spasticity in the function of UL and the necessity of treatment have been questioned[41]. Muscular weakness and biomechanical changes in soft tissues are proposed as the main causes of functional limitations in UL[42].

Figure 2. Brunnstrom motor recovery states after a stroke (31).



Bhadane et al., 2015, recently concluded that there is a strong correlation between the angle of rest of the articulation of the elbow and the severity of spasticity through clinical and biomechanical measurements, affecting motor function[43].

The Hypertonicity Intervention Planning Model (HIPM), which corresponds to a process structured through clinical reasoning and decision-making, to maintain the long-term function of US affected by hypertonicity[44], was created with the goal of guiding professionals in the area through the complex process of choosing the most appropriate intervention.

The model is divided in groups that correlate with the terms mild (HIPM 1), moderate (HIPM 2a), moderate-severe (HIPM 2b) and severe (HIPM 3), and are used clinically to describe the amount of hypertonicity in one limb and the degree of functional limitation experienced by the individual on a daily basis.

For example, if spasticity is low but negative signs predominate (HIPM1), it should not be intervened. However, if the person has moderate to severe disability in UL (HIPM 2b or 3), but positive signs predominate, intervention is proposed[3].

In the chronic stage, the limb may be affected by mechanical stiffness associated with hypertonicity rather than spasticity, which seems to decrease over time, affecting the therapeutic decision since they respond to different types of intervention[45].

When relating spasticity to motor recovery, it is important to focus on three topics related to the functional impact it causes: (1) the clinical pattern of motor dysfunction and its origin, (2) the subject's ability to control

the muscles involved in the pattern, and (2) differentiation between the role of muscle stiffness and contractions[46].

Conclusion

Spasticity is a phenomenon associated with different neurological pathologies and continues to be a point of division among the leading experts in the area of neurorehabilitation; this considering that, in some cases, it is associated with loss of function and disability, and in others, it favors trunk control, UL function, standing, and ambulation.

Thus, some therapeutic approaches aim to inhibit spasticity and others to maintain or facilitate it. The indication to decrease spasticity through different therapeutic strategies, such as botulinum toxin[47], intrathecal pump, oral medications (escitalopram)[48], and physical therapy[49] [50], does not show conclusive results and is still under research, even if its inhibition is justified to prevent an incipient contracture or to reduce the regional pain associated with a shoulder dislocation.

Studies are still needed to help establish the prevalence and affected musculature when spasticity is established, as well as therapeutic guidelines for the rehabilitation team regarding when to inhibit or facilitate this positive sign of MNS damage in order to promote motor recovery.

In conclusion, spasticity and motor recuperation are mediated by different mechanisms and stages after an MNS injury, which must be considered when preparing the therapeutic plan of each patient.

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