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TECNOLOGÍA EXPERIMENTAL YACHAY**

Escuela de Ciencias Biológicas e Ingeniería

**TÍTULO: STUDY OF AN ELECTRICAL REHABILITATION
TO REDUCE THE SPASTICITY IN THE MUSCLE**

Trabajo de integración curricular presentado como requisito para la
obtención del título de Ingeniera Biomédica

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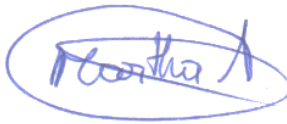
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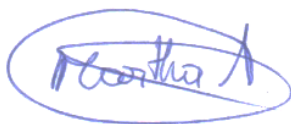
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Resumen

Cuando se producen lesiones graves en las extremidades que pueden conllevar a la falta de movimiento prolongado o continuo, se produce la atrofia muscular. Esta falla muscular se encuentra asociada a la espasticidad, la cual se puede definir como la alteración del control sensoriomotor derivado de una lesión neuromotora superior; caracterizada por una exageración de los reflejos de estiramiento debido al aumento del tono muscular y el clonus. Uno de los tratamientos más prometedores es la aplicación de electricidad, la Estimulación Eléctrica Funcional (FES), que utiliza pulsos eléctricos de baja energía para generar artificialmente movimientos corporales en pacientes con parálisis parcial o total de sus extremidades. El protocolo FES depende del tipo y localización de la espasticidad, por ello es necesario un análisis crítico detallado de la configuración de FES en términos de amplitud, forma de onda, frecuencia y voltaje, que considere cualquier aspecto de la patología. El conocimiento sobre la aplicación de FES en la rehabilitación de la espasticidad es disperso, incompleto y algo contradictorio. Por esta razón, el presente artículo presenta información conjunta de una manera integral para dar una actualización del potencial terapéutico real de FES en la rehabilitación de los músculos espásticos.

Palabras claves:

Espasticidad, Estimulación Eléctrica Funcional O FES, Estimulación Eléctrica, Rehabilitación, Terapias.

Abstract

The muscles of the limbs are kept in constant work, these are able to adapt to the efforts by their strength and power of work. When severe injuries occur, a prolonged or continuous lack of movement can occur, leading to muscle atrophy. One of the pathologies associated with this muscle failure is spasticity, which can be defined as the alteration of sensorimotor control derived from a superior neuromotor injury, characterized by an exaggeration of stretch reflexes due to increased muscle tone and clonus. One of the most promising treatments is the application of electricity, namely the Functional Electrical Stimulation (FES), which uses electrical pulses of low energy to artificially generate body movements in patients with partial or total palsy of his limbs. The FES protocol will depend on the type and localization of spasticity. It is critical a detailed analysis of the configuration of FES in terms of amplitude, waveform, frequency, and voltage, which consider any aspect of the pathology. The knowledge about FES application in spasticity rehabilitation is scattered, incomplete, and somewhat contradictory. For this reason, the present article joint information of a comprehensive way to give an update of the actual therapeutic potential of FES in the rehabilitation of spastic muscles. Future research avenues to improve the efficacy of FES in physical rehabilitation of spasticity are also provided.

Keywords:

Spasticity, Functional Electrical Stimulation or FES, Electrical Stimulation, Rehabilitation, Therapies.

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1. Introduction and justification of thesis:

Spasticity is one of the principal pathologies of any injury in the Central Nervous System (CNS), it disables or reduces the mobility range of the patient affecting in their daily activities. For this, is necessary find the best tool to the rehabilitation to recover the muscle function so that the patient recovers not only his functionality but also feels sure.

Spasticity is defined as the alteration of the sensorimotor control resulting from disturbances of the upper motor-neuron (UMN) system (Bacca, Patiño, Herrera & Barela, 2017). It is caused by abnormal nervous control exerted by UMN system on the distal spinal networks that directly control muscle movements. These lesions affect significantly the daily activities of the patient by the mobility reduction and the pain produced. The rehabilitation and reduction of the spastic muscle is something important to study to decrease the affections of neurological injuries.

The first sign of spasticity reduces the patient's independence in their daily activities, and causes significant pain, contractures and joint dislocations and ends with the limitation of voluntary movement of the limbs. The cure of this disabling illness relies on drug therapy and different rehabilitation strategies.

The different treatments of spasticity rehabilitation are divided in pharmacological, electrical rehabilitations, and physical treatment. The pharmacological treatment is used with the aim to reduce the side effects of spasticity like pain, depression and muscle tone. The physical treatment of spasticity is used to recondition the spastic muscle so that it returns to perform its normal functions. Finally, the electrical stimulation is aimed to reducing the muscle tone and to stimulate the nervous to recover their functionality helping a better recovery of the muscle (García Díez, 2013).

These treatments reduce the spasticity in the patient to restore their normal quality of life. The main problem with these treatments is that a single treatment is not enough for the rehabilitation of the spastic muscles. A combination of treatments rather demonstrates favorable results for recovery. The electrical rehabilitation like Functional Electrical Stimulation (FES) and Neuromuscular Electrical Stimulation (NMES) are among the most controversial treatments so far used for spasticity rehabilitation because, according to some

health professionals the treatment is not just useful, but also increase spasticity and worsen their symptoms. Suitable clinical methods to evaluate the therapeutic outcomes of the electrical stimulation treatments are lacking, which contributes to skepticism among health professionals and physicians of the therapeutic potential of this treatment in muscular spasticity. The most popular methods are analytical scales for spasticity (Le Cavorzin, 2013), which are conducted before and after to each treatment, to reliably capture therapeutic effectiveness. The present work gives a critical insight into the different ways of applying electro-stimulation as a treatment for muscular spasticity highlighting the advantages and disadvantages of each type of method, with a special emphasis in FES.

2. Problem and its background

According to Biering-Sørensen, Nielsen, & Klinge (2006), after the first year of spinal cord injury, about 70% of the people are spastic. Spasticity affects significantly the quality of life not only of the person with the pathology, but also to their relatives; for this reason, adequate treatment and rehabilitation are very important to allow the patient have some independence in their daily activities, which avoids the psychological troubles associated with the illness including depression and anxiety. Psychological distress interferes with spasticity treatment.

The grade of spasticity is hard to evaluate, which makes it rely on the subjective criteria of the physician. Therefore, there is not a unifying criteria or reference standards to which the progress of a patient undergoing treatment can be referred to. There is not a clear definition for spasticity because it is not produced by the disruption of a single physiological process, but rather by a chain of disturbances affecting neural networks at different levels as well as interconnected soft and hard tissues, resulting in an array of clinical signs (Sepúlveda, Bacco, Cubillos, & Doussoulin, 2018). To achieve the rehabilitation, or at least relieve spasticity, it is firstly needed to understand its etiopathogenesis and the diagnosis of severity before being able to evaluate the effectiveness of any treatment.

2.1. Muscle activity

The skeletal (striated) muscle is a contractile tissue and an active part of the locomotor system. It is what allows the function of moving and at the same time maintaining body stability; all this thanks to its ability to contract. The muscle consists of multiple individual cell fibers (myocytes), which are its basic functional unit. Muscle fibers are parallel to each other to produce an additive contraction force. To achieve this contraction, a number of myocytes are recruited depending on the strength developed by muscle fascicle. The approximate size of these cells is 12 cm long by 0.04 x 0.06 mm thick (Plonsey & Barr, 2007; Juarez & Montes, 2012). An active skeletal muscle can spend up to the 90% of its energy; without shortening, since it is in a constant muscle tension (i.e., *tonic* contraction). In contrast, the *phasic* contraction produces a shortening of the fibers (motion) which cannot be more than half of its length. A greater either tonic or phasic effort requires more blood supply (Juarez & Montes, 2012). The largest skeletal muscle groups are found in the upper and lower extremities of the human body. As a result of their permanent activity, the limb muscles adapt to the workload by increasing the number of myocytes, their thickness, and cell metabolism. The adaptation to workload is achieved through constant training during daily physical exercise. Physical inactivity as consequence of sedentary or paralysis produced by Central Nervous System (CNS) lesions leads to serious muscular injuries and even muscle atrophy. Some of these pathologies may be treatable with pharmacological treatments, physical therapy (i.e., rehabilitation), or both.

2.2. Spasticity

Spasticity is defined as the alteration of the sensorimotor control resulting from disturbances of the upper motor-neuron system (Bacca, Patiño, Herrera & Barela, 2017). It is characterized by an exaggeration of stretch reflexes caused by an increase in muscle tone and uncontrolled repetitive and involuntary contractions (Chang, Ghosh, Yanni, Lee, Alexandru, & Mozaffar, 2013). Based on the clinical symptoms, spasticity is diagnosed by the presence of:

- a) Increased muscle tone: exaggerated response to muscle stretch and decreased control of the stretch reflexes.
- b) Spasms: involuntary, repetitive, and sustained movements involving multiple muscle groups and joints.
- c) Clonus: rhythmically involuntary contractions following a sudden muscle stretching (see Figure 1).

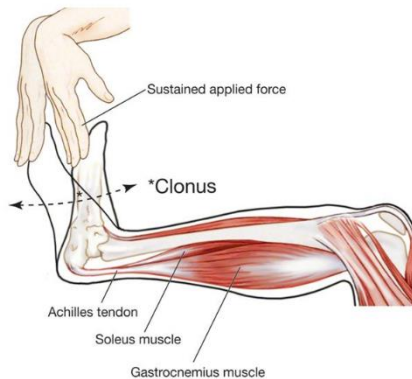


Figure 1. Illustration of ankle clonus in a person with spasticity, which consists of rhythmic contraction - relaxation cycles of the ankle extensor muscles responding to sustained stretch. (Source: Medical Images).

2.2.1. Etiogenesis

Spasticity is caused by abnormal nervous control exerted by upper motor-neuron system on the distal spinal networks that directly control muscle movements. Neural lesions interrupt the nerve pathways between the upper and lower motor control systems, thus leading to defective motor adjustments of the muscle-skeleton system. Spasticity is the hallmark of the so-called Pyramidal Syndrome to which *rigid* palsy is associated (García Díez, 2013). Lesions affecting the pyramidal tracts of the CNS produce palsy, increased muscle tone and clonus (*spasticity*) of largely the limb muscles and, as opposite to disturbances of extrapyramidal system (e.g., basal ganglia) affecting the trunk and causing rigid palsy and posture abnormalities (*stiffness*).

The pyramidal pathways (see Figure 2) include the anterior corticospinal tract, lateral corticospinal tract, corticobulbar, and tectospinal tracts, which are collectively known as

the pathways of the Upper Motor Neuron system or UMN that constitute the nerve afferents to the spinal motor circuitries. Physiologically speaking, spasticity is a motor disorder caused by injuries of the UMN that give rise to the descending pyramidal tracts. These tracts control, through the activity of alpha-motor neurons sited in the spinal cord, thousands of muscle fibers of two types: I and IIa. Fibers I (slow-twitch, anti-gravity fiber) are responsible for the slow, tonic contractions necessary for postural maintenance, while Fibers IIa (fast-twitch, anti-fatigue fiber) are responsible for the rapid contractions involved phasic movements of the limbs (Bolaños, Arizmendi, Carrillo, Rivera & Jiménez, 2011).

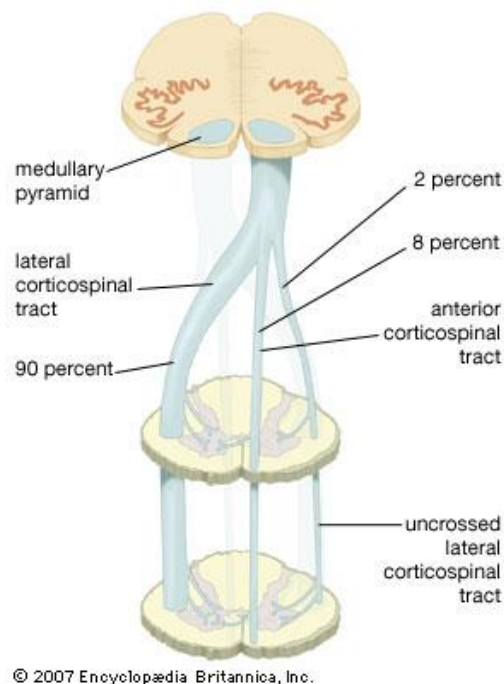


Figure 2. The decussation of the medullary pyramids and the formation of the corticospinal tract in the spinal cord. (Source: Encyclopedia Britannica)

The UMN syndrome presents both positive and negative movement symptoms. The negative symptoms include weakness, rigid palsy, and loss of dexterity. As to positive symptoms, they are found muscular hyperactivity, excessive muscle contractibility, and any kind of movement not showing a normal pattern. Spasticity then falls into the positive symptoms of UMN syndrome. This syndrome not only involves the pyramidal tracts, but also adjacent nerve fibers (Sheean, 2002). Lesions of other structures, like the lateral

reticulospinal tract (extrapyramidal) and the anterior (pyramidal), produces a clinical condition similar to the UMN syndrome. Indeed, the lesion of the lateral reticulospinal tract mostly produces spasticity by inhibiting the control of the flexor activity (Mukherjee & Chakravarty, 2010).

2.3.Spasticity and stiffness

Spasticity is often confused with stiffness; the difference between these concepts is the location of the injuries. While the injury location of spasticity is the pyramidal pathway, the injury location of stiffness is the extrapyramidal pathway (basal ganglia). One way to differentiate the stiffness from spasticity is the flexion pattern of the body. Stiffness produces flexion in trunk and limbs, while spasticity pattern involves flexion in upper limbs and extension in lower limbs, with the presence of Babinski signal. Also, it is important to understand that spasticity only occurs during muscle extension (not at rest), and stiffness occurs during the resting state (Aznavurian, & Aguilar, 2006).

2.4.Spasticity symptoms and characteristics

The first signs of spasticity reduce the patient's independence in their daily activities, causes significant pain, contractures and joint dislocations, and ends with the limitation of voluntary movement of the limbs, the spasticity symptoms are divided in positive and negative. The positive symptoms of UMN are the consequences of the activity of the pyramidal elements either less affected by the lesion or that did not lose its function completely, whilst the negative symptoms are due to components of the pyramidal syndrome that are lacking. The principal problem of spasticity is that it produces motionlessness, social isolation, pressure abscesses, infections, and other effects due to the physical disability (Bhimani & Anderson, 2014). The type of dominant motor symptom present in the patient will depend on the grade of severity and location of the injury.

2.4.1. Severity grade of spasticity

Spasticity has three levels of severity:

- a) Mild spasticity: clonus, increased muscle tone, and muscular spasms with a minimal affectation of joint mobility and limb functionality.
- b) Moderate spasticity: decrease of the movement range with occurrence of muscle contractures and a difficult march that forces the patient to use external assistance for daily tasks.
- c) Severe spasticity: excessive muscle tone that severely reduces mobility and increases the occurrence of muscle contractures (Quiñones, Paz, Delgado, & Jiménez, 2009). It disrupts sleep and has psychological consequences.

2.4.2. Localization injury of spasticity

There usually exist three specific lesions:

- a) Cortical injury, internal capsule or trunk injury above the core prepuberal bulb produce moderate spasticity with dominant estrogenic activity.
- b) Partial spinal cord injury causes significant spasticity with predominant motor tone of the extensor muscle.
- c) Complete spinal cord injury leads to high variety of symptoms as a result of the location of the lesion rather than its etiology (Rey Redondo, 2015).




2.4.3. Upper limb pattern




Each of these symptoms usually appears according to a pattern of muscular alteration. In the case of the upper limbs they are shown in the *Table 1*.

Table 1

Patterns of spastic upper limbs and their characteristics (adapted from Quiñones, Paz, Delgado, & Jiménez, 2009; Rey Redondo, 2015 and Latinoamericano De

Especialistas & Esquenazi, 2012).

<u>Thumb included in the palm</u>	
<p>Affected muscles: Flexor long thumb, short flexor thumb, short flexor of thumb, adductor pollicis and first interosseous dorsal.</p> <p>Functional alteration: Impediment to hold things</p>	
<u>Shoulder in adduction / internal rotation</u>	
<p>Affected muscles: <i>Pectoralis major, latissimus dorsi</i>, greater round and long head of the triceps.</p> <p>Posture: Elbow in flexion, right hand and forearm on the anterior part of the thorax.</p> <p>Functional alteration: Limitation for personal hygiene, dressing, reaching objects, shoulder pain.</p>	
<u>Elbow in flexion</u>	
<p>Affected muscles: Anterior brachial biceps and long supinator. The radial extensor of the carpus and the pronator may also be affected.</p> <p>Functional alteration: Limitation for dressing, manipulation of objects such as cane or crutch, altered symmetry and speed of walking</p>	
<u>Forearm in pronation</u>	

<p>Affected muscles: pronator round and pronator squared.</p> <p>Functional alteration: limitation to reach objects and perform activities that require turning the palm of the hand upwards.</p>	
<p><u>Wrist flexion</u></p>	
<p>Affected muscles: radial flexor of the carpus, anterior ulnar, palmar minor, common flexor <i>digitorum profundus</i>, deep flexor <i>digitorum profundus</i> and, in cases where there is ulnar deviation, ulnar posterior.</p> <p>Functional alteration: limitation for dressing, hygiene, reaching objects, alteration of activities with both hands</p>	
<p><u>Closed fist</u></p>	
<p>Affected muscles: superficial common flexor of the fingers and deep flexor <i>digitorum profundus</i>.</p> <p>Functional alteration: pain, limitation for daily activities, release and grasp objects.</p>	

2.4.4. Spasticity phases

Spasticity has genetic, congenital, or acquired (the most common) origins and does not only imply injured neuronal pathways, but also processes unleashed afterwards, such as the outbreaks of unharmed nerve fibers as result of the plasticity of the brain (Sepúlveda, Bacco, Cubillos, & Doussoulin, 2018). Spasticity develops gradually since the denervation and subsequent trans-synaptic degeneration lead to the hypersensitivity of the remaining healthy neural. Spasticity phases are described as follows (Figure 3):

Initial phase: it is characterized by an increased muscle tension by exacerbated muscle stretch reflex, when lengthened passively.

Vicious Activity phase: when not treated from the beginning, spasticity produces muscular imbalances in certain muscle groups, largely in the elbow, wrist, and finger flexor muscles of the upper limbs.

Muscular Retraction phase: if the vicious activity persists over time, agonist and antagonist muscle groups will grow unequally leading to a lack of accommodation of the sarcomeres and an abnormal structure of myocytes. This phase is characterized by the resistance opposed by muscles to stretching.

Phase of osteoarticular deformities: It corresponds to the last phase of the spasticity evolution and is due to the lack of treatment for spasticity in previous phases. It is more evident during the childhood as it coincides with the high rate of bone growth. According to the *Delpech Law*, the compressive and traction stimuli on growing cartilages and bones are altered by spastic deformities of the muscles, thus evolving to the characteristic osteoarticular deformities of the limbs (Oreja, Montalban, de Andrés, Casanova, Muñoz, García, & Fernández, 2013; Rey Redondo, 2015).

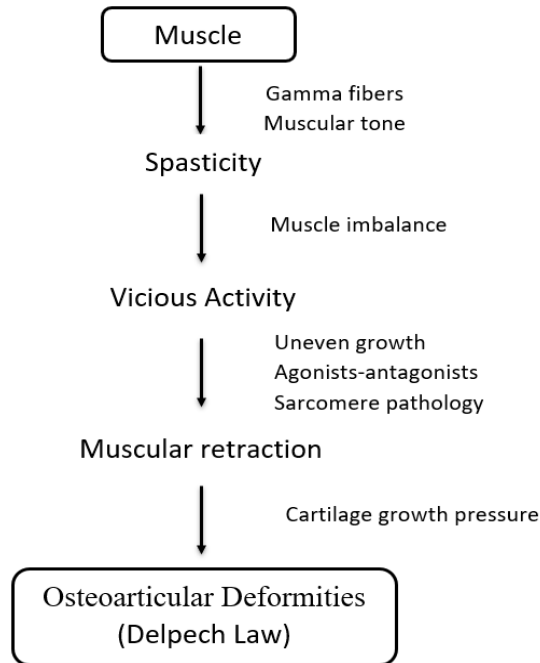


Figure 3. Spasticity phases (adapted from Vivancos, Pascual, Nardi, Miquel, de Miguel, Martínez & García, 2007).

2.5.Spasticity Treatment

The dominant symptoms in every clinical case will determine the specificity of the treatment to be given. It varies from pharmacological therapy to physical rehabilitation and even electro-stimulation. The health specialist uses a flow chart (Figure 4) to select the best therapeutic strategy (Soriano, 2012; Le Cavorzin, 2013). Only if spasticity compromises the life quality of the patient, the physician performs a pormenorized clinical evaluation of illness progression and the grade of spasticity, which is classified as either chronic or transitory. Physical therapy is recommended only if spasticity is transitory. When chronic spasticity, a combination of focal (applied to the specific site) or generalized (pharmacological and physical) therapies are needed pending on the extension of the affected area. Among treatments to reduce discomfort and improve motor restrictions, they are found the systemic administration of drugs with central mechanisms of action like baclofen (an alpha-2 agonists injected via intrathecal), the local injection of the Botox (a toxin from *Clostridium botulinum*), rehabilitation by the electrical stimulation (Chang et al., 2013), and physical

therapy. A combined treatment of the “Botulinum Toxin type A” and physical rehabilitation is a widely used first-choice treatment.

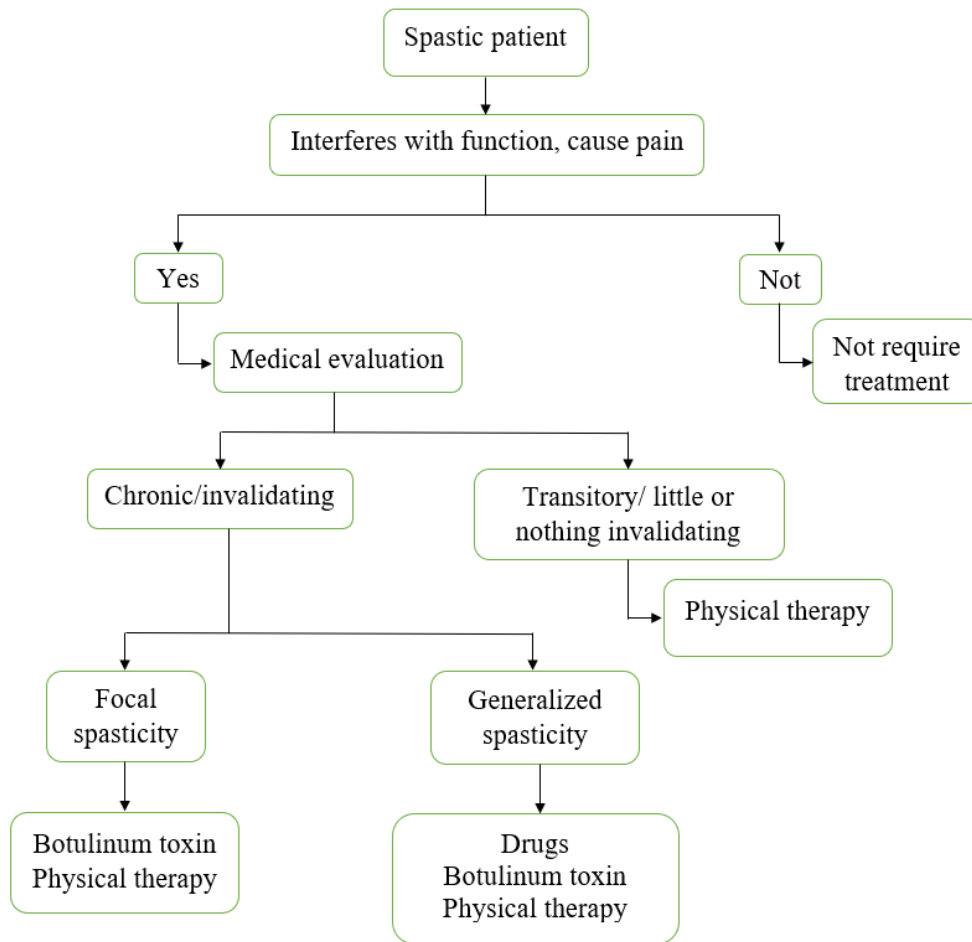


Figure 4. Spasticity therapies (adapted from Soriano, 2012; Le Cavorzin, 2013).

2.5.1 Botulinum toxin type A (BTXA).

This toxin inhibits of the release of the neurotransmitter acetylcholine at the neuromuscular plaques, thus producing the permanent stimulation and contraction of myocytes (Quiñones, Paz, Delgado, & Jiménez, 2009). BTXA is the purified (crystalline) form of one of the eight exotoxins produced by the bacteria *Clostridium botulinum*. It is used for the focal treatment of the spasticity of specifically affected muscle combined with

physiotherapy to obtain the maximal results, as well as an adjuvant treatment for others anti-spastic drugs. BTXA reduces pain, muscle tone, improves passive movements of the muscles, and prevents and even reduces bone deformities in infantile spasticity (Oreja, Montalban, de Andrés, Casanova, Muñoz, García, & Fernández, 2013). Given its tropism for skeletal muscles, the route of administration is a deep intramuscular injection. Reliable pharmacological effects appear within the next 5-7 days, reaching the maximal therapeutic effects within 3-4 months. In bulky muscles, it should be injected in up to 6 points to prevent the spread to unaffected muscles (Mutuberría, Parra, Camejo, & Valdés, 2005). The advantage of this toxin is the durability of its effects. As a shortcoming, it is not recommended in cases of ankylosis and when uncontrolled systemic diseases.

2.5.2 Physical therapy

It is aimed at inhibiting the excessive tone and facilitating normal movements for the patient to regain normal mobility. It consists of the following interventions:

- *Postural treatment* through correcting postural adjustments, it tries to avoid sudden stretches and side effects that aggravate spasticity (Murie, & Imaz, 2015).
- *Kinesiology* consists of joint mobilizations, stretching of the spastic muscles and neuromuscular facilitation using of passive and slow mobilizations. It prevents joint stiffness and bone deformities.
- *Ferula and orthotics* are adapted accessories helping patients control their spastic muscles and recover mobility.
- *Massaging* stimulates cutaneous mechanoreceptors thus reducing muscle tone in a rhythmic, deep and smooth way.
- *Myoelectric feedback* provides patient's awareness of the variations in electrical activity in their spastic muscles under treatment that should or will be attempted to be voluntarily controlled. It requires surface electrodes that indicate patient's myographic activity.
- *Vibrotherapy* manually regulated electric vibrators that are applied to the myotendinous joints on stretched muscles.

- *Cryotherapy* treatments based on inducing cold by ice, cold water, dry ice, cryogen, or methyl chloride for a 15 min-period on spastic limbs to reduce pain and muscle tone.
- *Thermotherapy* is the application of heat by irradiation (infrared rays), contact (hot water, fluid therapy) and conduction (high frequency electrotherapy) to reduce spasticity.

2.6.Spasticity evaluation following treatment

It is generally accepted that spasticity is easy to diagnose, but difficult to quantify, which is essential for the therapeutic effectiveness of treatments (Biering-Sørensen, Nielsen, & Klinge, 2006). Several protocols have been developed for the clinical evaluation of spasticity, which vary from not specific like the analytical scales, to specific like the pendulum test, goniometer and the electromyogram.

2.6.1. Analytical Scales

The most widely used are the analytical scales for spasticity (Le Cavorzin, 2013), where an observer assigns a number to the degree of spasticity depending on the characteristics described for each while the patient is found in relaxation to assess their passive movement. The following is a brief description of the most widely used scales in the evaluation of spasticity:

- Ashworth scale (Table 2): based on the resistance to passive mobilization around a joint.
- Modified Ashworth Scale (Table 3): It evaluates the resistance to passive mobilization of the limb in cases of hypertonia (increased muscle tone).
- Penn scale (Table 4): It relies on frequency and facility of production of muscle spasms, both flexors and extensors in response to stimuli.
- Oxford scale: It scores muscle injury levels or muscular strength.
- Held-Tardieu scale: similar to the Modified Ashworth scale, but more complex as it integrates the velocity dependence of stretching.

Their main drawbacks are the inaccuracy of the scoring which is biased by the observer's subjectivity. These scales do not provide an accurate estimation of the motor disability because other symptoms are likely to interfere with the magnitude of the functional loss.

Table 2

Adapted from Quiñones, Paz, Delgado, & Jiménez, 2009.

Ashworth Scale	
Muscle tone evaluation	
1-	No increase in muscle tone
0	Discrete increase in tone with minimal resistance to passive movement
1+	Discrete increase of tone with resistance in all passive movement
2	Decrease in range of movement greater than 50% and less than 100%
3	Mobility range limited by less than 50%
4	Severe limitation of mobility

Table 3

Adapted from Quiñones, Paz, Delgado, & Jiménez, 2009.

Modified Ashworth Scale	
Muscle tone evaluation	
0	No increase in muscle tone
1	Slight increase in muscle tone manifested by the action of << grabbing and releasing >> or by a minimum resistance at the end of the range of motion, when the affected limb is moved in flexion or

	extension.
1+	Slight increase in muscle tone manifested by the action of << grab, followed by minute resistance >>, through the remaining movement arc.
2	Marked increase in muscle tone, manifested through most of the movement arc, but the affected limb is easily moved.
3	Considerable increase in muscle tone. Passive movement is difficult
4	Severe limitation of mobility

Table 4

Adapted from Quiñones, Paz, Delgado, & Jiménez, 2009

Penn Scale	
Spasm evaluation	
0	No spasms
1	Spasms included only by stimuli
2	Spasms that occur less than once every hour
3	Spasms that occur more than once every hour
4	Spasms that occur more than 10 times per hour

2.6.2. Goniometer

A more precise tool to carry out analytical evaluations of the mobility grade based on the articular balance (see Figure 5). The measurements are conducted on the dorsal flexion of the wrist, palmar flexion of the wrist, radial and cubital deviations. This procedure describes abnormal positions of groups of bones using medical, orthopedic, therapeutic and diagnostic devices.



Figure 5. Classic and digital goniometer (Source: [Taboadela, 2007](#); [Apemedical](#)).

2.6.3. Pendulum Test

Other procedure, which is widely used in order to evaluate the spasticity of the quadriceps muscle, is the “Pendulum Test”. It consists of an objective estimate of the mechanical features of limbs responding to movement or stretching. This test describes the movement of the leg following a free drop under gravity from a horizontal position. Yeh and colleagues (2016) designed an experimental device for this test with a three-axis accelerometer sensor, internal three-axis gyroscope, and a Bluetooth-based wireless device. Figure 6 shows the movement of leg from start position to resting stage (top) and the recording (called “goniogram”) of the measurement of knee (bottom).

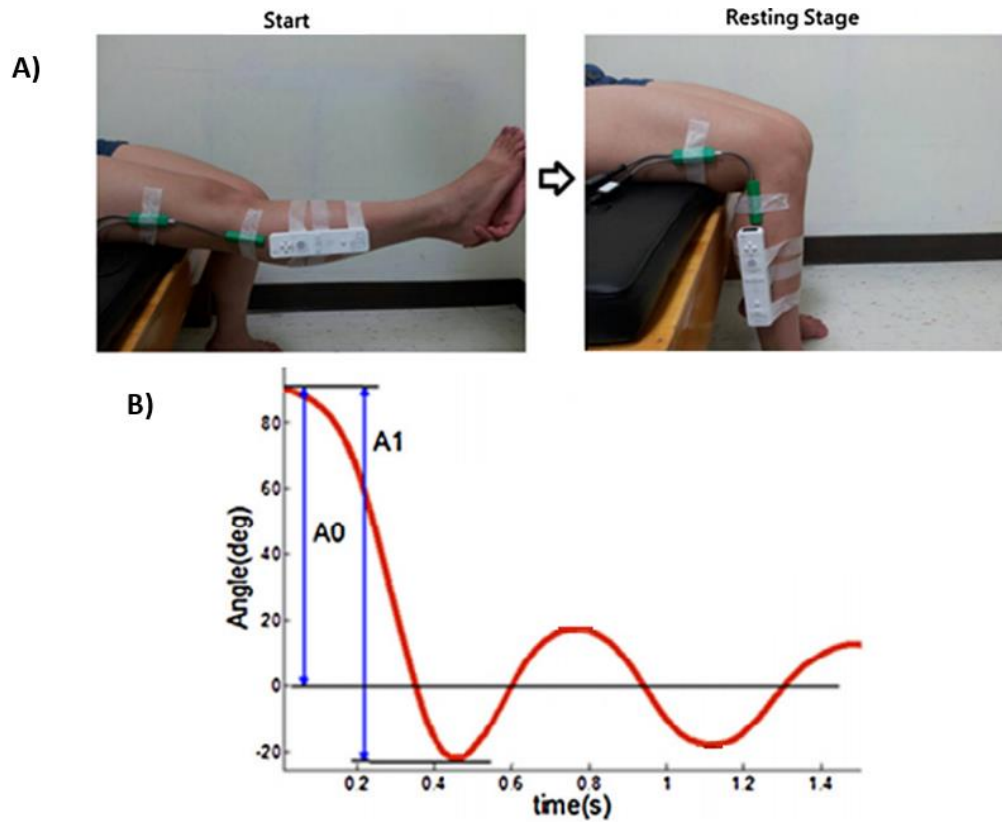


Figure 6. A) Photos of the movement of leg from the start position to resting stage. B): Goniogram and the spasticity parameters. (Adapted from: [Yeh et al., 2016](#)).

The application of the “pendulum test” is simple and only requires the minimum of cooperation by the patient. Spasticity level can be captured with a free pendulum movement or with an isokinetic machine. The parameters measured with the pendulum test are the reflection index (or RI) and the normalized reflection index (or RIn). The RI is defined as the quotient between the value of the maximum angular excursion corresponding to the first roll and the difference between the angles between start and end. This quantifies the degree to which the extensor muscles slow down the movement of the joint: the greater the bricking, the lower the RI. The RIn is obtained by dividing RI by the constant M, which will depend on the measurement system and the age of the subjects. A $RIn > 1$ indicates a non-spastic member, while a $RIn < 1$ indicates the presence of a spastic member ([Lin, Ju, & Lin, 2003](#)). The RIn can even differentiate spasticity from stiffness.

2.6.4. Electromyogram (EMG)

Another important tool is the electromyogram (EMG). Data recollection relies on the activation of the stretch reflex and measurements consist of grading the stretching of the muscle (stretching reflection), the tendon tap (T-reflex), and the responses to the electrical stimulation of the peripheral nerve supplying the muscle (H-reflex). If any of these responses present a value highly over what is considered the reference (normal) levels, subject is diagnosed as having spasticity. However, the method presents three problems. Firstly, the amplitude of the responses measured in the EMG depends on factors such as electrode placement, skin resistance, subcutaneous fat, and muscle atrophy. For these reasons, EMG data present great both between-session and inter-individual variability. Second, given the variable responses, it is difficult to obtain of reference values of spasticity for comparison purposes. Finally, EMG studies in spasticity are scarce and therefore reference information is limited. (Biering-Sørensen, Nielsen, & Klinge, 2006)

2.7. Electrical Stimulation

The electrical stimulation is a less invasive alternative to acupuncture, where through high frequency currents with pulse trains, since it has been shown that the own spinal system depresses the excitability of the motor neurons after applying cutaneous stimulation. When applying electrical stimuli, the afferent information would activate synapses, but since the excitatory ones are near their level of saturation they would not modify their activity, instead the inhibitory synapses would be activated producing a state tending to the original balance (Spaich, & Tabernig, 2002). There are three different types of electrical stimulation: TENS (Transcutaneous Electrical Nervous Stimulation), NMES and FES. TENS seeks to improve tissue health or voluntary function by inducing physiological changes that remain after stimulation. Neuromuscular electrical stimulation (NMES) excites the motor nerve fiber through an electrical impulse, to stimulate the muscular motor plate and cause the muscle contraction. In contrast, FES intervention seeks to enable the function by replacing or aiding the voluntary capacity of a person (Cabrera Naranjo, & Hoyos Aguilar, 2015). In the clinical practice, there is the belief that the electrical stimulation is harmful, then this treatment is not offered to many patients as rehabilitation tool (Spaich and Tabernig; 2002).

2.8. Functional Electrical Stimulation (FES)

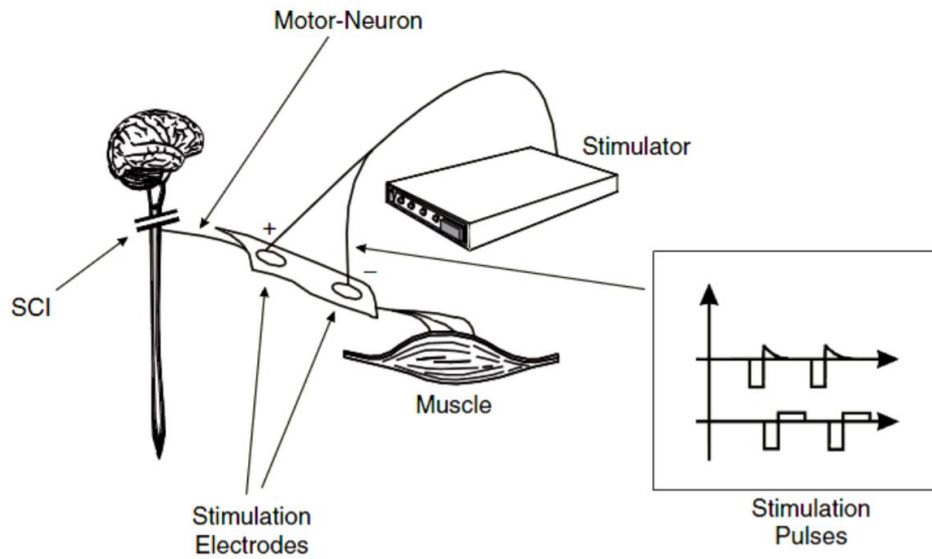


Figure 7. Basic configuration of FES with surface stimulation electrodes causes a muscle contraction by stimulating electrically the motor-neurons that are attached to the muscle (Source: Ahmad, Ibrahim, Hanafi, Rahman & Jamil, 2015).

FES uses electrical pulses of low energy to artificially generate body movements in patients with body palsy. This electrical charge is able to stimulate both the motor and sensory nerves, producing a direct muscle contraction. This only happens in the nerves or points where the union between the nerve and the muscle occurs. The FES is applied to neurons, which encode and transmit a series of impulses known as action potentials. The nerve signals are modulated in frequencies between 4 and 12 Hz. The electrical stimulation artificially caused this action potential by changing the electrical potential through the cell membrane (Plonsey, & Barr, 2007). The FES is a less invasive alternative to acupuncture. Through the cutaneous stimulation of high frequency currents applied in form of pulse trains, the own spinal system is able to depress the excitability of the motor neurons. When applying FES, the afferent information would activate synapses, but since the excitatory ones are near their level of saturation they would not modify their activity, instead the inhibitory synapses at the spinal cord would be activated producing a state that tends to re-establish to the original balance between flexor and extensor muscles (Spaich, & Tabernig, 2002). FES devices are named neuro-prosthetics since they work out as a substitute for the lost neurological function (Cabrera Naranjo, & Hoyos Aguilar, 2015).

The functional electrical stimulation (FES) treatment is also useful to monitor the progression of the rehabilitation. For example, Ahmad, Ibrahim, Hanafi, Rahman, & Jamil (2015) suggest a set of combined devices (the FES system, see Figure 7) consisting of a goniometer sensor, a PC (Personal Computer) based in an analysis system, the FES, an isolator, and surface electrodes. The process of the FES system begins to measure movement ranges of muscles using a goniometer sensor that recollects the experimental data. Once stored, the data are processed to develop an equation of motion that creates a dynamic model serving as a reference for the treatment. The PC based analysis system in turn goes to control the stimulator to apply the correct electrical pulses for rehabilitation. This proof of concept is difficult to apply due to the technical challenges inherent to the recollecting and processing of data (Ahmad, Ibrahim, Hanafi, Rahman, & Jamil, 2015).

The benefits of FES are the muscle strengthening, the enhancement of limb functions, the improvement of the range of motion (ROM), and the rehabilitation of voluntary responses. FES is used since 90's in the rehabilitation of lower limbs or muscles with injuries that derivate of spinal cord injuries (SCI) injuries. With the FES treatment is sought the rehabilitation of spasticity in muscles with atrophy after upper motor nerve spinal cord lesion, stimulating contractions that produce and generate power, force, and motion till a correct recovery of the muscle function. (Ahmad, Ibrahim, Hanafi, Rahman & Jamil, 2015). The electrodes are placed on or near the nerve fibers of the lower motor neurons that control a specific muscular group. Accordingly, with the appropriate electrical stimuli, they provoke action potentials in the axons, and the force of the resulting muscle contraction can be regulated by modulating the stimulus parameters. A functional movement of the limb can be produced by coordination of several electrically activated muscles. (Peckham, & Knutson, 2005).

2.8.1. Characteristics of Electrical Stimulus and Current Flow

When talking about of current flow for an electrical stimulus, it is referred to the waveform of this current and who are the parameters necessary to it application. There are many types of electrical current, the use of these depending on the type of therapy to be applied. In the

case of physiotherapeutic treatment, the electrical stimulation consists of the administration of either direct current (continuous unidirectional flow of ions) or an alternating current (uninterrupted bidirectional flow of ions) (Bellew, Michlovitz & Nolan, 2016).

For electrotherapy it is used an alternative type of electrical current known as *Pulsed Current* (or pulsatile), which is the most common electrotherapy to improve muscle strengthening and activity. It consist of a series of pulses can stimulate frequently the muscle fibers, which subsequently produces tetanic contraction. It is either the uni- or bidirectional flow of ions that periodically ceases for a period of time before the next electrical event. Its waveform shape has an impact in the tolerance and the effect size in the skeletal muscle during the stimulation (Bellew, Michlovitz & Nolan, 2016). When consecutive pulses are followed by another series of consecutive pulses, the current pattern becomes a burst (see Figure 8). Alternatively, an uninterrupted generation of pulses at a fixed frequency generates what is called a train of pulses, that is, a series of bursts.

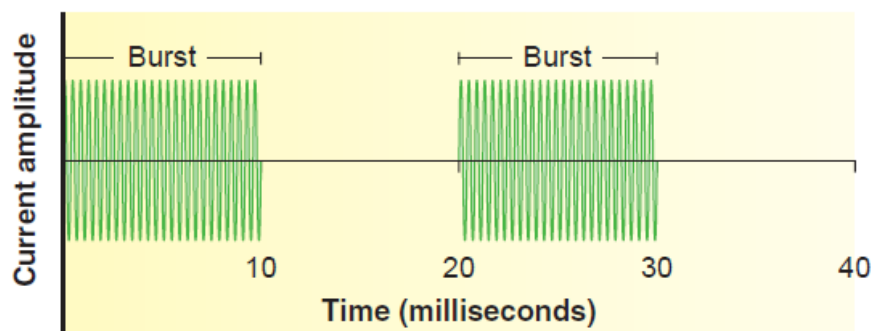


Figure 8. Representation of train of pulses (Adapted from: Bellew, Michlovitz & Nolan, 2016).

Regarding to the waveforms, they are described as follows:

- a) Monophasic: they have single phase and correspond to a unidirectional pulse from baseline to either positive or negative.
- b) Biphasic: they have two phases and are bidirectional wave with one positive phase and one negative phase. Biphasic waves can be symmetrical or asymmetrical.
- c) Polyphasic: they are bidirectional waves with three or more phases in bursts.

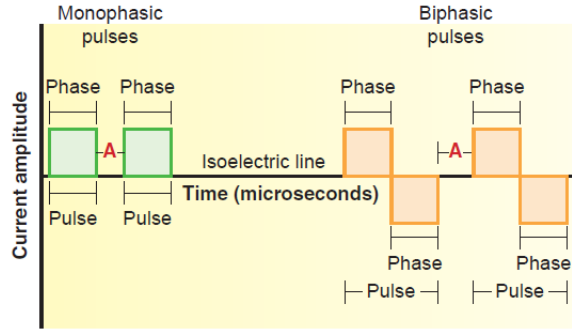


Figure 9. Representation of monophasic current and biphasic current (Adapted from: Bellew, Michlovitz & Nolan, 2016).

In the case of the pulse current, this can be monophasic (a monophasic pulse deviates from the isoelectric line in just one direction, depending on what direction the current initially flows before ceasing; see Figure 9). Most of the ES amplitudes are at mA scale. The charge of a single phase is called *phase charge* and the accumulated charge in all phases of a pulse is called *pulse charge*.

Scott, Causey & Marshall (2009) found that the magnitude of physiological effect in NMES is determined by the phase charge parameter, it depends of the current amplitude and pulse duration. The phase charge of each phase can help to determine if the wave is balanced or unbalanced. Also, the phase charge influences the recruitment of nerves, of this configuration depends the biological response to the treatment. If the phase charges are unbalanced and asymmetrical, it then results in a net current charge, which means that the wave in one direction has a current greater than the other.

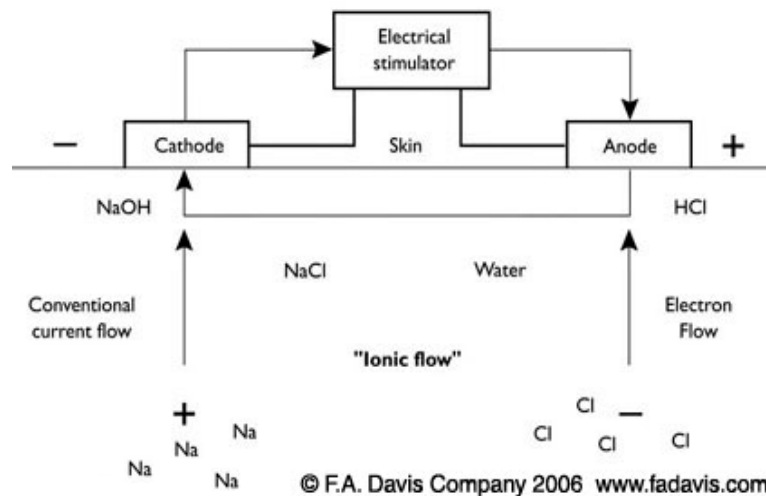


Figure 10. Representation of ions movement to electrodes (Adapted from: “Foundations of Electrical Stimulation, Physical Therapist Assistant Program at Lane Community College”).

The ions’ movement across the tissues produces flow of electrical currents that travel throughout the body. The main body electrolyte (NaCl) releases Na⁺ and Cl⁻ ions when an electrical stimulus is applied so that the Na⁺ cation migrates toward the cathode and the Cl⁻ anion moves toward the anode (Figure 10). In the cathode, Na⁺ reacts with water to form Na(OH) to cause a local alkalosis (pH > 7.4) of the extracellular fluid. In the meantime, Cl⁻ triggers an acidic reaction with water to produce hydrochloric acid (HCl) that acidifies the milieu around the anode. If either direct current or monophasic pulsed current are applied, these two ions will accumulate in the electrodes. If the applied current is biphasic, the two ions move forward and backwards between anode and cathode, a flow of ions very effective in the electrode-skin interface.

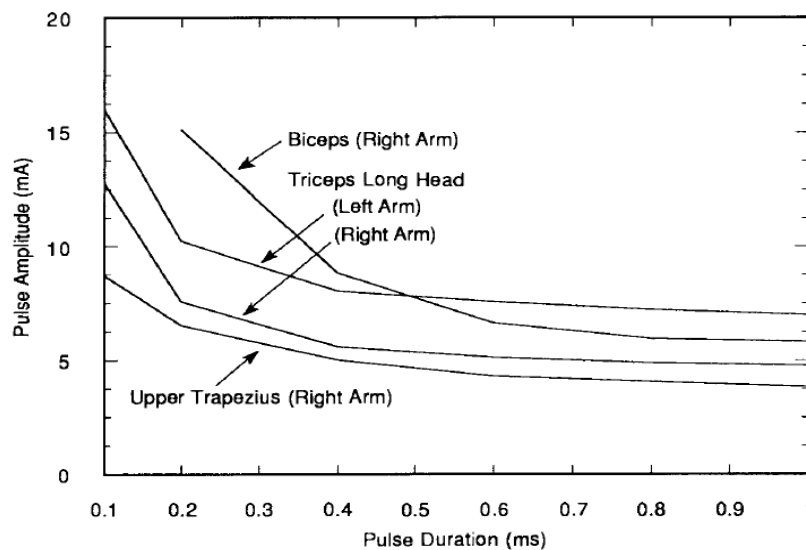


Figure 11. Representation Strength-duration curves for surface-electrode threshold stimulation of four upper-extremity muscles. (Source: Sweeney, 1998).

Electrothermal effects will depend on the amount of produced current in the tissues, the tissue impedance and duration of current flows. The stimulus must have the suitable current amplitude (or strength) and pulse duration to provoke the desired potential action. These two

parameters must have a non-linear relationship in order to elicit the action potential: S-D (strength-duration) curve without causing skin redness. Figure 11 shows the S-D curve; it should be noted that the action potential is not triggered below the curve.

2.8.2. Electrodes

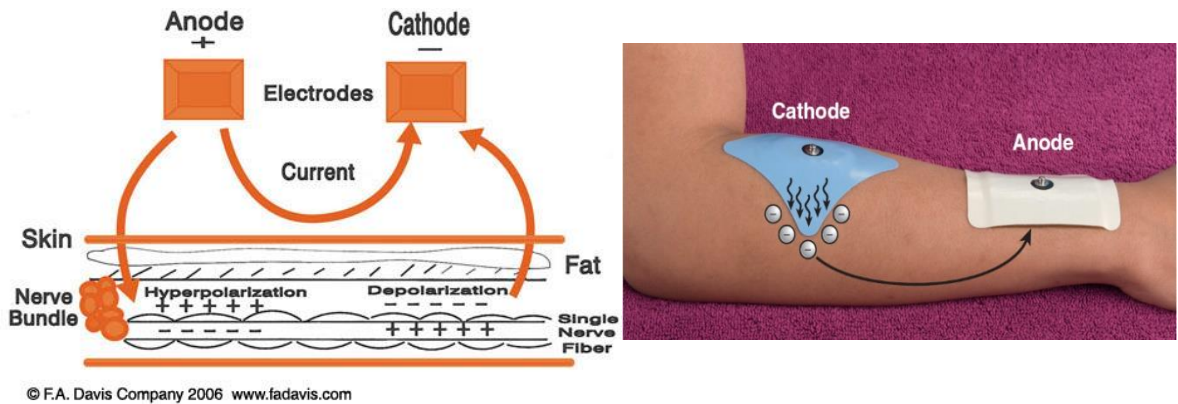


Figure 12. Left: Representation of the current in the body (“Foundations of Electrical Stimulation, Physical Therapist Assistant Program at Lane Community College”). Right: Representation of the electrons flow (Source: [Bellew, Michlovitz & Nolan, 2016](#)).

In the case of the application of electrodes (see Figure 12), because the same charges repel and opposite charges attract, a flow of charges takes place in the body. In the configuration of two electrodes, there are an anode (positive electrode) and a cathode (negative electrode). The conventional current flow is from anode to cathode (Fig 12, left) and the movements of electrons is from cathode to anode (Fig 12, right).

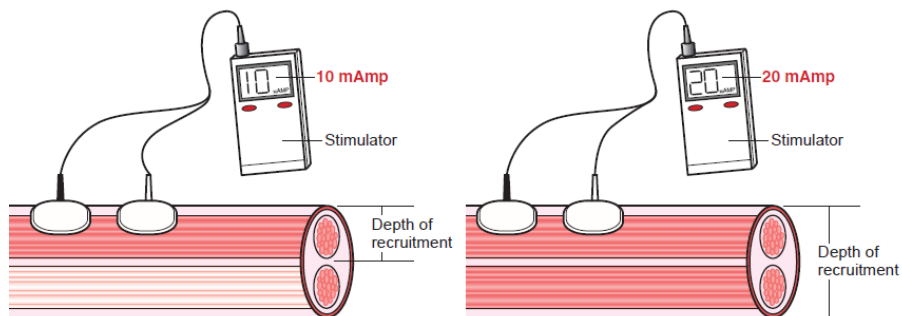


Figure 13. Fibers will be recruited before smaller-diameter fibers (left). Increasing the amplitude of the stimulus will recruit more fibers (right) (Source: Bellew, Michlovitz & Nolan, 2016).

Table 5

Potential treatments in function of the objective (Foundations of Electrical Stimulation, Physical Therapist Assistant Program at Lane Community College).

Treatment Goal	# Leads and Electrodes	Monopolar	Bipolar	Quadpolar
Muscle (motor) Stimulation	One lead per muscle with both electrodes on the same muscle, two leads if it is a larger muscle or if the device has more than one head		X	
Sensory Stimulation	One or two leads depending on the size of the area; use as many electrodes as possible to sensory stimulation			X
	One lead if only one lead and two electrodes fit in the treatment area One lead with one electrode at the spinal nerve root and the other in the sensory area	X	X	
Delivery of Medication	One lead and one electrode in the treatment area and the other more proximally placed on soft tissue	X		

The order of recruitment will be affected by the fiber depth in such a way that the first fibers to be excited will be those close to the electrode. However, the largest fibers will still be excited before the smaller. The way to stimulate the deeper fibers is by increasing the amplitude of the stimulus (see Figure 13). Some recommendations about the lead, electrodes and shape of wave are presented for different goals are in Table 5 (“Foundations of Electrical Stimulation, Physical Therapist Assistant Program at Lane Community College”).

3. Objectives:

3.1.General objective

The goal is to provide a critical overview of the therapeutic potential of FES for the treatment of muscle spasticity, with a special focus to the characteristics of the FES regime.

3.2.Specific objectives

- To understand the principal features of spasticity affecting patient's daily life.
- To review the advantages of FES over other therapies in the treatment of spasticity.
- To suggest recommendations about the most feasible waveforms of FES to be applied in the recovery of spastic muscles.
- To critically analyze the therapeutic suitability of the different FES protocols.

4. Methodology of the study.

The bibliography was browsed on the Google Scholar platform primary using the keywords "spasticity" and "functional electrical stimulation (FES)". The terms "electrical stimulation" and "rehabilitation of spasticity" were used in a secondary search. The search was limited to reliable scientific sources like articles, reviews, thesis written by medical professionals, and some handbooks of Neuropathology and Bioelectricity. Google Scholar platform redirect the search to data bases like Taylor and Francis online, Elsevier, SciElo, Wiley Online Library, NCBI, Sage journals, etc.

The published scientific reports covered the period from 1995 to 2019, and were written in Spanish, English, and Portuguese. They were identified up to seventy documents that fulfill the selection criteria, but with the criteria exclusion of repeated and not relevant information was selected only fifty-eight articles, and sixty of which presented information relevant to the theme of this thesis. The results of investigation are detailed according the

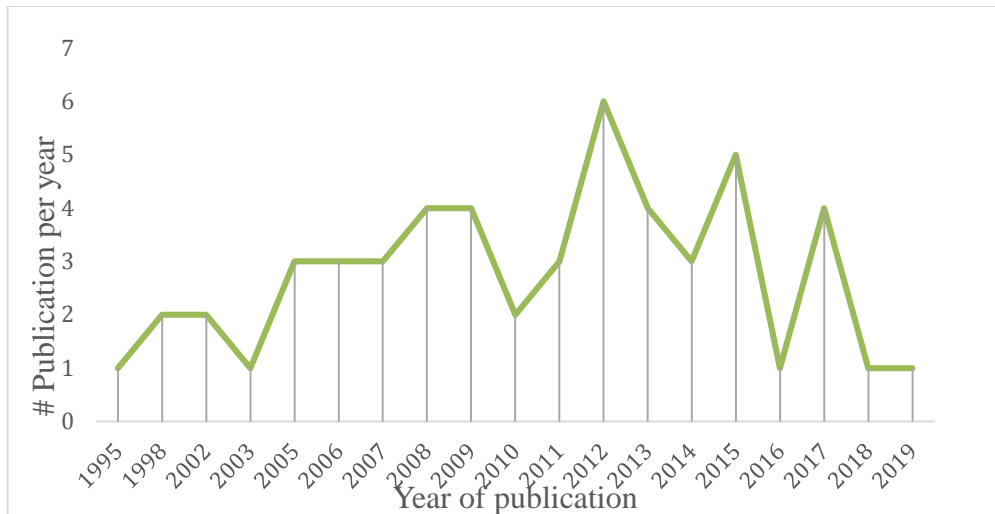
data base and the quantity of articles presents in each one about the searched theme in the Table 6.

Table 6

Description of data bases of search.

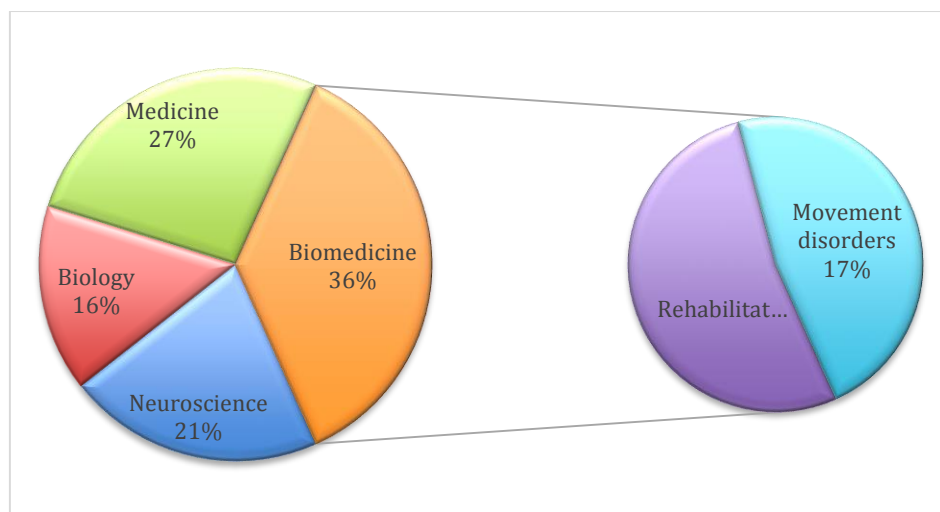
Data base	Articles
Elsevier	11
Sage Journals	4
Wiley Online Library	3
NCBI	3
SciElo	3
Spring	2
AHA Journals	2
Others	30

To the databases was identified two articles of Nature Journal, two articles of Gait & Posture journal, Clinical Rehabilitation journal present four articles, Movement Disorders journal have two articles and six articles of Neurology of different countries like Mexico, Brazil and Europe. All the collected information was conveniently used to explain the basic characteristics of spasticity and electrical stimulation treatment. It was also incorporated information about the spasticity in patients with Central Nervous System injuries.



Graphic 1. Number of publications per year

For the analysis of the data also was identified the years of publication of the documents used in the present work to indicate the grade of interest in the scientific field about the theme, it was showed by the Graphic 1. It also was compared with the type of journal by scientific area used for the publication to understand the principal areas related with this theme. According to this information was possible observe an increase in the interest of the theme, especially in medicine, neuroscience and biomedical engineering areas (see Graphic 2). The comparison of these data also goes to contribute in the analysis of the documents used in the present work to a better comprehension of the theme.



Graphic 2. Percentage of publications per scientific area

5. Presentation, analysis and interpretation of data

5.1. Electrical stimulation progress

Based on the scrutinized bibliography, the therapeutic potential of electrical stimulation in spasticity rehabilitation is still controversial. The following work is a brief overview of the recent progresses made in that particular field. For these, it was analyzed publications focused on electrostimulation as the therapy for the reduction of spasticity that give a complete description of the protocols and types of therapies used.

Motta-Oishi, Magalhães, & Mícolis de Azevedo (2013) challenged the hypothesis that some neuroplastic effects within some spinal cord circuitries may account for the NMES-induced improvement in spasticity. NMES effects in promoting neuroplasticity has been reported in the brain after stroke, but have not within the spinal cord. The protocol used to this paper to bear out the hypothesis involves experimental and clinical studies that are designed to evaluate the excitability levels in different spinal tracts. NMES parameters of stimulation used of these articles are left for exploration, its means that the response to the protocol could be delivered under different frequencies and/or intensities of NMES stimulation, also protocols involve the H-reflex assessments to lower and upper limbs. To do this, neural transmission in the spinal circuitries need to be evaluated before and after the exposition the NEMS treatment. Then of realized studies in the present article, they probe that H-reflex measurement show a satisfactory reliability, but for this was necessary to evaluate between 10 to 15 patients and no more, to discover the differences in the modulation of the vertebral pathways compared to the control subjects. This report also suggests that it is possible to compare the animal test and human test only in the cases to the spinal cord protocols, for the data recollection.

According to Bakhtiary, & Fatemy (2008) the Bobath inhibitory technique jointly with electrical stimulation may help to reduce de spasticity. The Bobath inhibitory technique tries to inhibit the spasticity to improve the voluntary movement in the limbs, it is also known like neurodevelopmental technique (NDT) used to the treatment of stroke patients. Its technique is applied to the treatment of movement disorders and posture. The Bobath technique is based in the reorganization of the brain by means of peripheral sensory calculations and it is used

to replace the lost functions of affected zones of the brain with the healthy parts, that means that is to say that the affected limb acts according to the opposite limb less affected in order to have a lifestyle more balanced. The results of the studies realized by this group showed that the NMES with Bobath techniques are more effective in the spasticity reduction.

Such as the previous work, in order to determine if FES actually produces an improvement in the physiological conditions of the patient with spasticity, others were carried out. Also, the efficiency of the FES application is analyzed against methodologies using this treatment combined with another treatment, for example electrical stimulation with stretching exercises. This is the case of the work of Sahin, Ugurlu, & Albayrak (2012). As presented before, NMES is an electrical stimulation in the motor nerve fiber to cause the muscle contraction. These authors found that this combination of techniques was more effective than the use of only stretching exercises. One reason was the fact that NMES treatment could be applied to strengthen the antagonist muscles and this alleviated the spasticity. In their research, the authors worked with 50 patients with forearm flexor spasticity following a stroke. Finally, these authors concluded that more studies about the clinical efficacy of electrical stimulation for spasticity are necessary.

According to Yıldızgören, Nakipoğlu Yüzer, Ekiz, & Özgirgin (2014), an improvement of the effectiveness of the treatment of wrist range motion, spasticity, and hand functions is achieved if the treatment with conventional exercises (such as those ones consisted of neurophysiologic exercises using the *Bobath* approach) includes NMES. They worked with 24 children with unilateral spastic cerebral palsy. They compare the wrist extension angle and Zancolli scoring of wrist spasticity between two groups, a control group and the NMES group. The control group was exposed to conventional exercises and static volar wrist-hand orthosis, while the NMES group was exposed to the same condition with the addition of a 30 min of NMES sessions. The Zancolli classification system classifies the spastic hand according its deformities. In the Figure 13a, a comparison of the extension angle is made between NMES group and control group, and it shows that the extension angle of the hand spastic patients improved after NMES treatment, allowing a better wrist mobility. When comparing wrist spasticity evaluated according the Zancolli classification (see Figure 13b),

a reduction in the Zancolli score was observed in the NMES group, which demonstrated the effectiveness of the NMES treatment in the wrist.

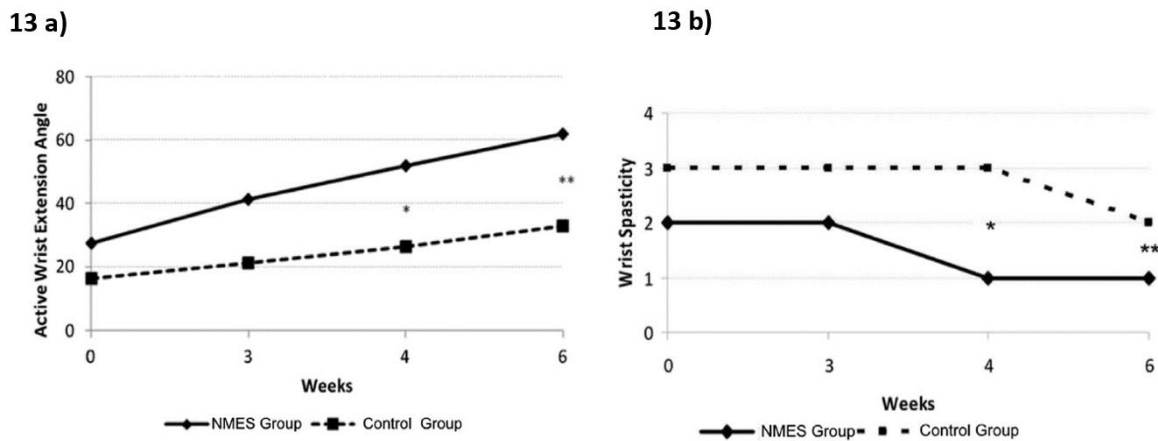


Figure 13: a) Comparison of the wrist extension angle changes between NMES group and Control group; b) Comparison of the Zancolli wrist spasticity changes between NMES group and Control group. (Source: Yıldızgören, Nakipoğlu Yüzer, Ekiz, & Özgirgin, 2014)

Other combination of therapies, such as short term cycling training and FES, were studied by Lo, Hsu, Hsueh, & Yeh (2012) in 20 stroke patients. They found that the physical training produced a reduction in the spasticity of the leg, with or without the application of FES, for example in reductions in muscle tone. However, in the cases of in the case of patients with higher muscle tone, the cycling training plus the use of FES produced additional benefits. Again, in this work the authors recommend more long term studies in order to check the improvements of the FES-cycling training combination.

Another work that supports the thesis of combining FES with physical exercises is the Sabut, Sikdar, Kumar, & Mahadevappa (2011), where they explain that the combination of FES and conventional rehabilitation has better results than a conventional treatment alone, in the spasticity reduction (in the dorsiflexor muscle) and an effective rehabilitation of the gait, greater stability, and confidence.

Finally, Intiso, Santamato, & Di Rienzo, (2017) performed a detailed analysis between the bibliography from MEDLINE/PubMed, the Cochrane Central Register of Controlled Trials, CINAHL and EMBASE data base, from January 1966 to January 2016, about the spasticity

treatment with botulinum toxin type A plus electrical stimulation. They were 9 studies among which 7 reported positive impacts in the decrease of the spasticity; these are: Hesse et al. (1995), Hesse et al. (1998), Frasson et al. (2005), Baricich et al. (2008), Picelli et al. (2011), Santamato et al. (2013) and Weber et al. (2010).

All previous works support the use of electrical stimulation, but in general explain that it is necessary more clinical studies. One example of positive results happened with NMES application but not in the decreasing of spasticity was the research of Kamper, Yasukawa, Barrett, & Gaebler-Spira (2006) which analyzed the NMES effects on upper limb impairment and in this case with cerebral palsy. They also found that although this treatment improved the wrist extensor muscles strength and increased the wrist extension range of motion, the spasticity measure did not decrease during the NMES therapeutic program.

In addition, Thomaz, Cipriano Jr, Formiga, Fachin-Martins, Cipriano, Martins, & Cahalin, (2019) claimed that the electrical stimulation therapy is an effective technique to enhance muscle volume, but it had no effects in reducing spasticity, thus producing more pain and fatigue. It demonstrates the importance of more investigations about these types of therapies like FES to a better comprehension of the effectiveness of these in the reduction and rehabilitation of spasticity.

The use of FES in patients with spinal cord injury produces changes in the spastic muscle tone; this was analyzed by Krause, Szecsi, & Straube (2008). As shown in Figure 14, the swinging is different before and after a session of FES.

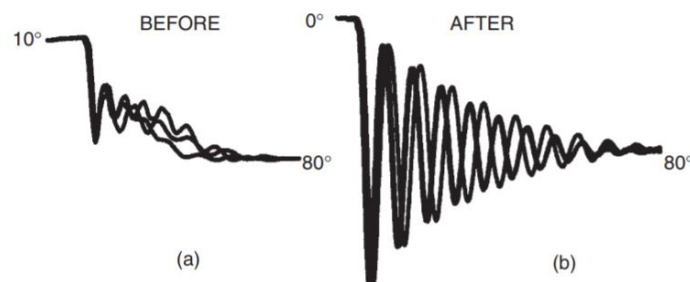


Figure 14: Graph of three records about of swing phases of a patient. (Source: Krause, Szecsi, & Straube, 2008)

In this work, the authors found that, after each intervention, the active session with FES or the passive movement session, the spastic muscle tone increase was diminishing. Also, it resulted in a limited degree of reduced spastic muscle tone. This means that there were changes in the spasticity as side effect of using electrical stimulation.

5.2.Wave shape

With regard to the type of wave to use in electrical stimulation, in the case of Sahin, Ugurlu, & Albayrak (2012) analysis, they used a neuromuscular electrical stimulation delivered to the wrist extensor muscles with frequency of 100 Hz, with a 0.1 ms pulse and a 9 s rest, for 15 minutes to provide the maximum muscular contraction. In addition, the treatment was valued through the modified Ashworth scale that proved to be a good indicator. In the work of Yıldızgören, Nakipoğlu Yüzer, Ekiz, & Özgirgin, (2014), the NMES consisted in a pulse of 300 ms, 30 Hz, an on-time of 12 seconds, and 10-25 mA. In the case of the FES treatment applied by Sabut, Sikdar, Kumar, & Mahadevappa (2011) consisted in a current with 0.28 ms pulses, at 35 Hz in the constant mode within the subject's tolerance level. Finally, Takeda, Tanino, & Miyasaka (2017) explain in their review that FES, TES and NMES devices are used for muscle strengthening and rehabilitation of strokes. The electrical stimulation produced by this type of devices seeks to artificially perform muscle contraction as a method of recovering the paralysis of these muscles. Due to the great diversity of devices and methods of use, these depend on the type of spasticity and patient to be treated. Among the side effects of these devices there is the muscle torque, fatigue and pain induced by this electro stimulators, which depend on the amount of energy and wave parameters used. The article shows that stimulation with sinusoidal waves produces greater muscular strength and less pain, in comparison with rectangular waves. It is also mentioned reports in his studies that monophasic and biphasic waves are more advantageous than burst waves and argue that the number of cycles per second can be influenced by muscle fatigue.

In an experiment conducted by Malhotra, Rosewilliam, Hermens, Roffe, Jones, & Pandyan (2012), ninety patients of ages between 32 to 98 years old undergone electrical stimulation treatment to prevent the development of the pain, and prevent contractures in the spastic muscles. The parameters used in this treatment were configured for the production of

slow movements that keep the patient in a comfortable position, among them the burst used was 300 μ s, for 15 s “on” and 15 s “off”, the frequency was 40 Hz. The intensity was adjusted to obtain a maximum extension in wrists and finger without producing fatigue or pain. Of the ninety patients, 19 patients showed improvements, while the rest did not show any improvement.

Also, for NMES, Stein, Gassen Fritsch, Robinson, Sbruzzi, and Della M ea Plentz (2015) used two shape waves in order to produce the electrical stimulation. One wave had pulse duration from 0.1 to 0.4 ms and frequencies from 18 to 50 Hz, whereas the second had frequencies in the range of 80-100 Hz and pulse duration of 0.1-0.3 ms.

FES wave shapes use a train of biphasic square-wave pulses of 20 to 40 Hz in frequency, amplitudes from 0 to 120 mA, and pulses ranging from 0 to 300 μ s. This type of wave shape is used because it induces charge transfer into the tissue and then immediately induces charge transfer out of the tissue. This pattern of charge transfer prevents galvanic processes that can cause tissue damage. (Lynch, & Popovic, 2008).

Intiso, Santamato, & Di Rienzo, (2017) considered wave parameters were intensity (in mA), frequency (in Hz), and type of wave (shape and phases). The parameter’s values, coupled with the concentration of BTXA (botulinum toxin type A), were: intensity from 50 to 90 mA or 100 mA, frequency of 20-25 Hz, and the type of wave was continuous train of 3 s and each train of 0.2 ms, rectangular wave and biphasic balanced current. The duration of treatment was of 30 minutes, 2, 5 or 6 times a day, and for 4, 12 weeks. The concentration of BTXA was from 500 IU to 4000 IU. They reported a significant reduction of the spasticity, muscle tone reduction, the score of the modified Ashworth scale, the upper limb spasticity and an improved hand function.

Regarding to the shape of the wave, a square shape can be obtained from a circuit of electrical stimulation with adjustable parameters of frequency (from 1 to 100 Hz with 1 Hz increments), duty cycle from 1% to 99% (with 1% increments), and current intensity of up to 1 mA with increments of 50 μ A. In this vein, the physicians can use it for a big amount of medical applications according to the needs. This type of circuit was proposed by Hern andez, & Thierry, (2014) and has a micro-controller. Other circuit to produce an electrical stimulation was proposed by Romo (2009) mainly consist of one IC556 in oscillator mode,

plus a transformer that works with alternating signal and that increases the voltage of 556's output. The output of this circuit is a biphasic square deformed wave with from 20 to 100 V, a maximum current of 10 mA, and 500 Hz of frequency.

6. Discussion

The use of electrical stimulation treatments is controversial in the medical field not only by the variability of result among research groups, but also for the lack of detailed information of the protocols and reproducibility of the findings. There is still a gap of knowledge about the cause-effect relationship between the application of protocols of electrical stimulation and the therapeutic outcomes that requires further research.

According the articles investigated the combination of therapies to the spasticity rehabilitation in the better way to conduce a treatment, due to the aims of each one. The combination and election of the therapies is performed according to the type, localization and severity grade of spasticity. This investigation shows that the combination of an electrical stimulation therapy with physical therapy and/or pharmacological therapy (Botulinum Toxin A) is the most effective for muscle rehabilitation.

The combination of NMES or FES with physical therapies demonstrate a have effectiveness as treatment, this combination is not used simultaneously, but successively. The NMES current is used alternately, it means that during the stimulation time in a session it's going to alternate between on and off. Once finished this electrical stimulation treatment, start the physical therapy where are realized passive movements by the patient and in the case of chronic spasticity its movement of any physical therapy need to be assisted. Likewise, a combination of Botulinum Toxin A with the electrical stimulation (FES or NMES) demonstrates a good effectiveness in the treatment of spasticity and even more when jointly consist with physical therapy.

The combination with BTXA, physiotherapy, and electrical combination rather than the use of only two or one of the treatments alone, has more durability thus reducing the side effects and allowing a better functionality of the muscle. Its combination shows a greater

stability and pain reduction during the treatment. The application of any type of combination of treatments requires a protocol for its application in the clinical trials.

The working hypothesis of this work is that the combination of electrical with physical and pharmacological therapies improves rehabilitation. A caveat of this hypothesis is the scarce information regarding this combination. If this hypothesis is correct it may enhance the effectiveness of the rehabilitation by reducing the side effects. It is recommendable to conduct more investigation on the reduction of spasticity reduction using different combinations of three therapies and not only of two or one. It is also important to investigate the deep causes of the spasticity pathology to find the way the electrical rehabilitation have to be applied to resolve the problem.

In order to think in a protocol of clinical trials, we must begin with a written informed consent that gives an agreement to participation and also permits a publication of the results. Once the corresponding permits have been obtained, the pertinent protocol for the study is carried out. In the case of this work is necessary a protocol to Spasticity reduction, for this is necessary to determine: subjects, electrical stimulation sessions (parameters and time), and qualitative and quantitative evaluation.

For the sample election is necessary to find subjects between 20 to 70 years old, who present injuries in the central nervous system with problems of spasticity. The subjects must not present other health problems like uncompensated cardiovascular diseases, acute infections, tumors, seizures, pacemakers or metal implants, due to the side effects that can produce the electrical stimulation exposure to these. These subjects need to sign an agreement to participation where they accept the treatment to be used.

In the case of the electrical stimulation is difficult to choose only one way to apply the stimulation, due to the great variety of required parameters (wave shape, amplitude, frequency, pulse, cycles, tension, current, etc.) for its configuration. The configuration is going to depend on the spastic site and the evolution and time of pathology progression.

Another problem with the study of spasticity reduction is the lack of information about the treatment, due to the clinical trials realized and presented in different articles are not complete, it means that the incomplete information is presented in each one of these documents about the process and protocol of the treatment. These do not give the values for

the pre-treatment characteristics with all the problems, another article presents the electrical variables but it is not specific and not show how or why it is used, the majority of articles not explain how the variables may change with the progress of the treatment like the amplitude, frequency, pulse duration or waveform used in the process (see Table 7).

Table 7

NMES protocols, Std (standard protocol), SP (short pulse direction protocol), LF (Low frequency protocol) and LA (low amplitude protocol). (Source: Gorgey, Black, Elder & Dudley, 2009)

Protocol	Amplitude (mA)	Frequency (Hz)	Pulse Duration (μ s)	Torque (Nm)	Activated CSA (cm ²)	Torque per Active Area (Nm/cm ²)
Std	74 \pm 18	100	450	166 \pm 41	30 \pm 7	5.7 \pm 1.2
SP	76 \pm 16	100	150 [†]	78 \pm 40 [‡]	18 \pm 10 [‡]	4.3 \pm 1.3 [‡]
LF	72 \pm 18	25 [†]	450	137 \pm 30 [‡]	36 \pm 8	3.9 \pm 0.9 [‡]
LA	56 \pm 13 [†]	100	450	109 \pm 35 [‡]	22 \pm 12 [‡]	5.8 \pm 2.1

The problem not only occurs with the NMES but also with FES, in the case of FES there is not a big amount of information about this treatment, it is for this that it is more difficult to understand when, how, and when to use it.

Each one of these protocol are going to depend to the level of spasticity present by the patient and how the treatment is applied, and as the treatment shows or does not improvements, in the case of not showing improvements, a new full assessment of the spasticity must be made (apart from the tests before and after the treatment) it will be done to proceed with the choice of a new configuration of the parameters of electro stimulation, more efficient than previously used.

The lack of information not only in the parameters but also the utilization of these protocols, show that is not possible realize an analysis between articles because the protocols used in each of one differs in parameters and therapies combinations, for this reason there are diverse opinions of the usage of electrostimulation, due to some articles perform studies with just FES or NMES, while others conduct studies on combinations with physical therapies and/or drugs.

The presented articles also were based on different types of spasticity (localization and severity) and age range of those concerned, and that it is not the same to deal with an adult with spasticity than a child with spasticity, the same would apply for the place and type of spasticity, therefore the use of a single protocol as a standard protocol would not be the most feasible as treatment but could even increase this spasticity, therefore for the choice of a protocol for a specific patient would depend on the variables of spasticity present and whether this treatment is correct or not for the patient.

Finally, and not less important it is important to know the spasticity changes like pain, muscle tone and clonus increasing/reduction as side effect of the electrostimulation when it is applied, it means that there are changes in the evolution of spasticity during the treatment. The positive changes are also called improvement rate, this rate is not constant for this reason is necessary evaluate the spasticity periodically. Like this rate is not constant, the parameters need to be change according to these variables whenever it is necessary.

7. Summary and recommendations

Spasticity is a very disabling disease, caused by the damage in the SCI, which must be treated at the time of injury to reduce its effects to the maximum. Spasticity presents many aggravating factors so it can be difficult to find the right treatment because it can vary, that is, improve or worsen if the appropriate treatment is not applied. To this end, multiple evaluation tools have been developed to differentiate both the type of spasticity and the area of this and thus be able to choose the correct combination of treatments to be used for recovery.

Among the available treatments we have, the best one is the Botulinum Toxin A combined with physical therapy and electrical stimulation, which has shown great progress in the recovery of the spastic muscles to perform their activities normally. For this, the use of electrical stimulation-based therapies such as FES, NMES have been studied and compared both in their effectiveness and their protocols of use.

As a result of this study it was concluded that there cannot be a single treatment protocol for this type of electrical stimulation due to the changes that occur in spasticity during the

application of the same, with this in mind there has been to know the importance of the study of these types of treatments and the complete diffusion of the necessary information to reproduce these types of treatments.

Another important point that was deduced during the realization of this work is that due to the lack of information of these types of treatments it is not possible to conclude how efficient each of these can be without the supplementation with an additional treatment like the main characteristics of each one of them. This lack of information is the main difficulty present in this type of studies, so it is recommendable not only to look for basic information, but to deepen into the pathophysiology of this disease to have a better understanding of what should be treated and how electrical stimulation would help to the recovery of the patient, mainly in the upper limbs since the information on this is very limited.

8. Conclusion

Spasticity arises from the negative signs of the superior neuromotor injury, especially paresis and the reduction of selective motor control. Their treatments are very varied, among them is rehabilitation by electro stimulation, which is carried out mainly through the use of techniques such as FES (Functional Electrical Stimulation) and NMES (Neuromuscular Electrical Stimulation). These treatments when used alone do not show effectiveness, so that multiple combinations of treatment techniques have been developed such as the combination of drugs and electro-stimulation or physical therapy. The lack of information, data and studies on this electro stimulation as a treatment of spasticity causes a great debate about the effectiveness of this treatment, for this reason it is necessary to carry out more research on the subject, as well as clinical trials that allow us to better understand the effectiveness of this treatment.

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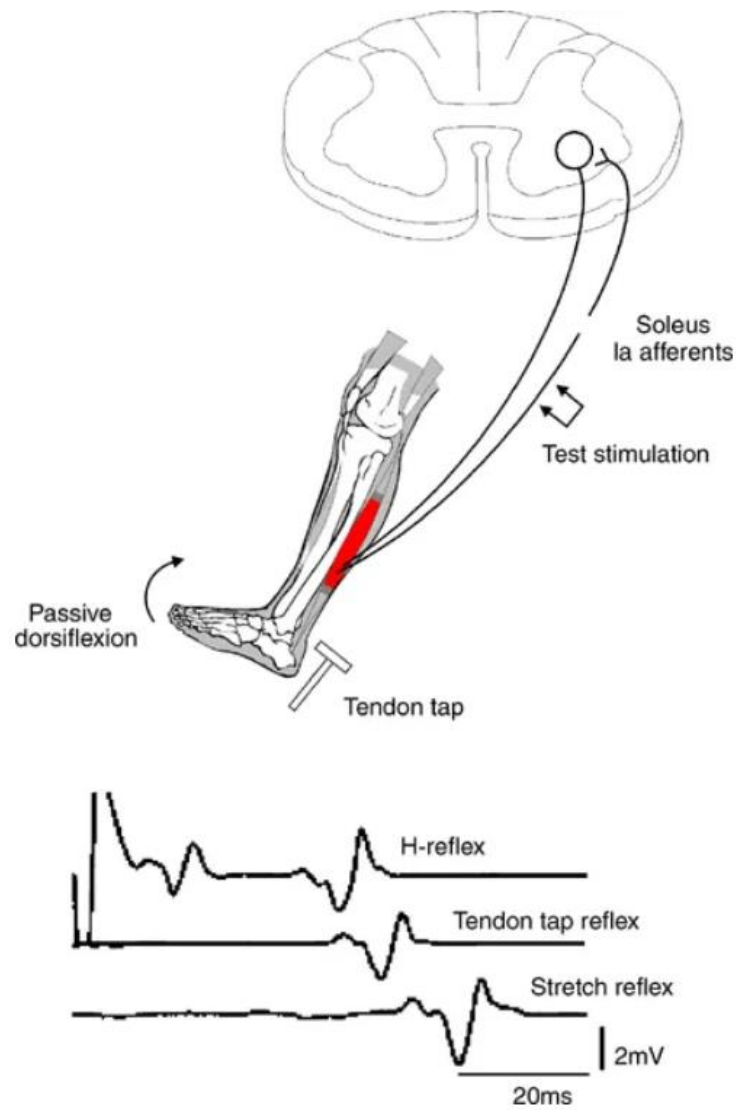
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APPENDIX

Appendix A:

Main responses of the EMG measurements of spasticity.



Taken from Biering-Sørensen, Nielsen, & Klinge, 2006