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Chapter

Neurosurgical Spasticity Treatment: From Lesion to Neuromodulation Procedures

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Abstract

Spasticity is one of the most important and residual signs after pyramidal and para-pyramidal catastrophic lesions after diverse neurological insults, including stroke, brain and spine trauma and post-radiation; infection and immunological diseases affecting nervous system, between others. Spasticity is normally a compensatory motor mechanism that could ameliorate the patients´ disability. Nevertheless, disastrous spasticity is described when the extremities force is diminished in the affected limbs, or when is impossible to wake o to take objects, maximum when hand or foot deformity is exposed. The objective of this chapter is centered in the neurosurgical treatment of spasticity, including brain lesions with specific targets and the spine with its different modalities. This review shows not only the basic aspects in these techniques, but also the option of infusion pumps and deep brain stimulation. To close, a proposal is stablished to determinate the possible path to treat the spasticity in the future.

Keywords: spasticity, neuroablation, neuromodulation, lesioning neurosurgery, motor disorders

1. Introduction

Spasticity is a motor disorder characterized by muscular hypertonia with resistance to passive movement, in a context of hyperexcitability of the stretch reflex. This disorder is a component of the upper motor neuron syndrome, an alteration where the inhibitory influence of supraspinal structures is lost. Spasticity is an important clinical problem frequently found in neurological patients. This condition is estimated to occur in 17–39% of patients with stroke [1], 37–78% of patients with multiple sclerosis [2], 65–78% of patients with spinal cord injury [3], and 90% of patients with cerebral palsy [4]. Spasticity could be very disabling for patients and its management requires several specialists and different therapeutic options.

If acceptable control of spasticity is not achieved with pharmacological treatment, physical therapy and rehabilitation, surgical procedures are the next treatment option. The goal of surgery in these patients is to decrease the excess muscle tone and rebalance agonist and antagonist muscle groups to improve function

and limit deformities. To promote a better restoration of function in patients with spasticity, it is important to know the functional alterations of this disorder. Furthermore, in order to measure the effects on the pathology after some treatment, it is necessary to use classifications and scales. In the following sections, the first topic to be addressed is the neurophysiological alterations of spasticity and then it will be mentioned the scales with which the degree of affection is classified. Later, it is going to be described the neurosurgical procedures, both ablative and neuromodulatory, available to compensate for the physiological alterations of this disorder.

2. Spasticity: pathophysiology and classification

2.1 Pathophysiology of spasticity

Muscle tone depends on the intrinsic elasticity/stiffness of the muscle, and this in turn is modulated by the nervous system. In physiological conditions, the neural circuits in the spinal cord mediate muscle stretch reflexes. These local circuits also provide a mechanism to adjust muscle tone from supraspinal structures according to physiological requirements. Due to this configuration, lesions in these supraspinal structures or in the descending motor pathways are frequently associated with alterations in muscle tone. These alterations may involve an abnormal increase or decrease in tone. The most common form of hypertonia is spasticity, which is characterized by a velocity-dependent resistance to passive movement of a joint and its associated musculature. A slowly applied stretch in a patient with spasticity causes little resistance, but as the speed of the stretch increases, the stretch resistance also increases progressively. Thus, spasticity is primarily a phasic phenomenon. An active reflex contraction that occurs only during a rapid stretch; when the muscle is held in an elongated position, the reflex contraction decreases. However, in some cases hypertonia also has a tonic component, for example if reflex contractions persist even after the muscle is no longer stretched [5].

In the last years, the pathophysiology of spasticity has been increasingly understood. Stretch reflex hyperactivity was long thought to be caused by overactive gamma motor neurons. However, although gamma motor neurons may be overactive in some cases, changes in the background activity of alpha motor neurons and interneurons are probably more important. Particularly relevant seem to be modifications in the intrinsic properties of motor neurons that generate a sustained firing in response to a brief excitatory input. Another mechanism that produces spasticity is the strong facilitation of synaptic transmission in the sensory fiber of the monosynaptic reflex pathway. In fact, this provides a mechanism for treating this disorder. Currently, a relatively common therapeutic procedure favors presynaptic inhibition in the *Ia* fiber terminals by the intrathecal infusion of baclofen, a GABA-B receptor agonist, that blocks neurotransmitter release [5, 6].

2.2 Classification of spasticity

Classifying the severity and distribution of spasticity in each patient is an essential element to achieve effective treatment and to observe its response over time. The severity of this disorder can range from a focal problem with mild muscle stiffness to a severe and painful diffuse spasticity.

Based on its location, spasticity can be classified into Focal, Regional, and Generalized. In the first case, a single muscle group or part of the body is affected, in regional spasticity, adjacent muscle groups are affected in a region of the body. In the patients with generalized spasticity, all or almost all areas of the body are affected [6].

3. Spasticity assessment

The degree of affection, its evolution over time, and the response to the therapeutic options can be evaluated with the use of some scales. There are several widely used scales: Ashworth Scale (AS), Modified Ashworth Scale (MAS) and Gross Motor Function Classification System (GMFCS). They are formed of different points to construct ordinal scale that assess muscle tone. A higher score indicates a more intense spasticity [7].

Moreover, to comprehend correctly the information said before it is necessary to understand the meaning of two important concepts: spasticity and spasms. Spasticity is "hypertonia that is associated with one or both of the following signs: resistance to passive movement that increases with speed of stretch, or when the resistance to externally imposed movement rises rapidly above a threshold speed or joint angle" [8]. On the other hand, spasms can be defined as "episodes of involuntary motor contractions that occur following a lesion of the ascending motor pathway" [9]. It is important to mention that spasticity is detrimental to human health due to the miscommunication between the brain and the muscles, as a consequence those patients have a diminished quality of life. Spasms can be presented in long periods of time that is why problems in the musculoskeletal system can appear, affecting mobility and tone of posture.

Bryan Ashworth, in 1964, created a classification in which patients with multiple sclerosis could be graded in order of their clinical manifestations, starting to objectivize the knowledge of the pathology mentioned before [10]. Between the decades of 1960's to late 1980's the scale was used by doctors all over the world to help get a more accurate diagnose of spasticity, contributing to the progress of science in the field of neurology.

Table 1 shows the classic Ashworth Scale that consist in five different types of categories that describe clinical manifestations of spasticity [11].

Furthermore, in 1987 Richard Bohannon's group added to the scale a category "1+" for a more accurate classification regarding clinical manifestations in patients with spasticity. Since then, the scientific community has been using this scale of measure to add a more specific diagnose in patients that present rigid upper limb due to its extension [12]. It is important to mention that both scales, Ashworth and Modified Ashworth, are useful in the detection of spasticity and can be helpful to objectivize the manifestations of spasticity.

In this image, it can be seen the modifications that Richard Bohannon added to the Ashworth Scale (**Table 2**) [13].

Richard Penn has published several articles concerning spasticity, and also is attributed as the creator of a scale that measures the frequency of spasms "Penn Spasm Frequency Scale (PSFS)".

Table 3 shows the different categories of spasms over time to identify characteristics according to the clinical manifestations [14].

Scale	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release.
2	Marked increase in muscle tone throughout most of the range of motion, but affected part(s) easily move.
3	Considerable increase in muscle tone; passive movement difficult.
4	Affected part(s) rigid in flexion or extension.

Table 1.
Ashworth scale.

Scale	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release.
1+	Slight increase in muscle tone, manifested as a catch, followed through the remainder of the range of motion.
2	Marked increase in muscle tone throughout most of the range of motion, but affected part(s) easily move.
3	Considerable increase in muscle tone; passive movement difficult.
4	Affected part(s) rigid in flexion or extension.

Table 2.
Modified Ashworth scale (MAS).

Scale	Description
0	None
1	Lack of spontaneous spasms; vigorous sensory and motor stimulation outcome in spasms.
2	Occasional spontaneous spasms occurring es than once per hour.
3	Greater than 1, but less than 10 spontaneous spasms per hour.
4	Greater than 10 spontaneous spasms per hour

Table 3.
Penn spasm frequency score (PSFS).

4. Treatment

The objective of the different pharmacological and surgical treatment options is to compensate the excitation/inhibition imbalance that occurs in the motoneurons of the ventral horn, the common final pathway for motor control. When possible, the underlying cause (e.g., tumor, abscess) that could be generated this imbalance should be eliminated. It is important to say that spasticity does not always require specific treatment. In many cases, spasticity can be helpful in maintaining balance and compensate for loss of motor power. Thus, spasticity should be treated when excess muscle tone leads to further functional disability, impaired locomotion, causes deformities, or induces chronic pain [5].

The different treatment options can be classified according to the location (focal vs. general) and duration (temporary vs. permanent) of their therapeutic effect. The choice of a treatment is made according to the severity and extent of spasticity and is adjusted according to the response and evolution of each patient. In general, surgical treatment is considered a second-line option, for patients with non-satisfactory response with drugs and physical therapy. Neurosurgical treatment options are divided into neuroablative and neuromodulatory procedures. The latter allow chemical or electrical regulation in the functioning of the neural circuits involved in spasticity. Importantly, such neuromodulation is characteristically adjustable and reversible. Ablative procedures are fixed and non-reversible. However, they still constitute a viable option for many patients, especially in those circumstances in which the use of neuromodulation equipment is not available. **Figure 1** shows a general algorithm for the treatment of a patient with spasticity from its initial assessment to management with lesioning procedures. This algorithm is only a general guide, and in each patient the treatment should be individualized in the context of a multidisciplinary management.

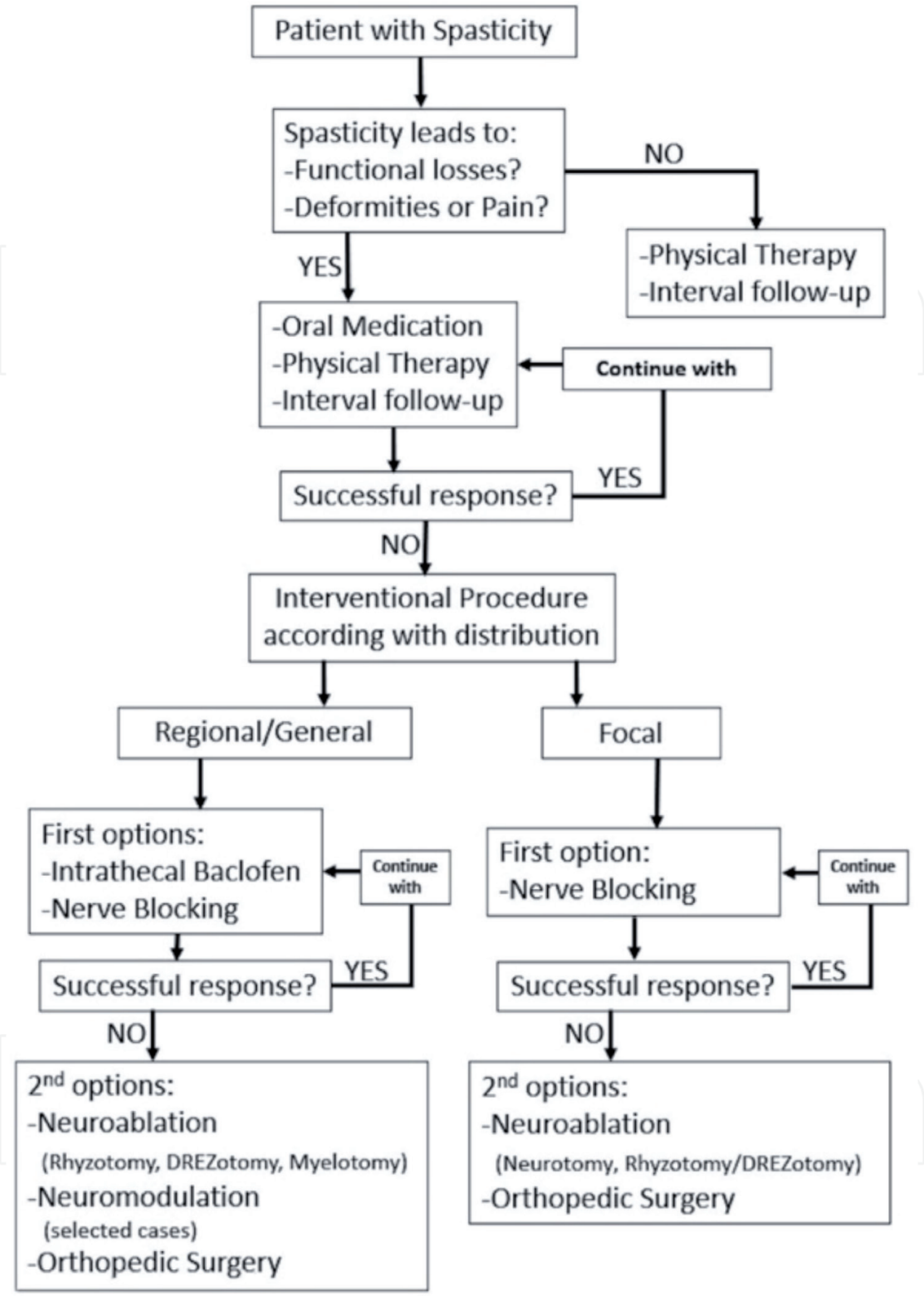


Figure 1.
General purpose algorithm for the treatment of spastic patients.

4.1 Ablative treatment

This treatment modality implies the realization of some injury in certain levels of the nervous system that participate in the motor function in order to counteract spasticity. These lesions include selective neurotomies, rhizotomies, DREZotomies,

myelotomies, and supramedular lesions. These procedures should be performed in such a way as to reduce excess muscle tone, but preserving residual sensorimotor functions and useful muscle tone. In cases of refractory spasticity, including paraplegic, hemiplegic or tetraplegic patients, the evolution and severity of the spasticity may require neuroablative management. In these cases, the injury procedure must be selective, and will be chosen considering the location of the spasticity (**Figure 1**). The characteristics of the most important and useful injuries will be reviewed below.

4.1.1 Selective neurotomy

Peripheral neurotomy was first introduced by Lorenz in 1887 for hip spasticity and by Stoffel in 1912 for spasticity in the foot [15]. This procedure consists of selectively identifying and injuring one motor nerve bundle that supply the spastic muscles. The goal of selective neurotomy is to inhibit the segmental reflex arc and thus limit the level of muscle spasticity. Selective neurotomy is indicated in cases of localized spasticity in a single or a few muscle groups, both in cases of focal or multifocal spasticity. The target nerves are selected according to the spastic region affected. Examples include lower subscapular nerve injury for spastic shoulder, median nerve injury for pronation spasticity of the upper limb, ulnar neurotomy for spastic wrist flexion with ulnar deviation, obturator nerve lesion in case of hip adduction spasticity, sciatic nerve for knee flexion spasticity and tibial neurotomy for equinus or equinovarus spastic foot [16].

The evaluation prior to neurotomy should include nerve blocks with a reversible agent such as botulinum toxin. These blocks allow us to observe a therapeutic effect previous to neurotomy and to evaluate its usefulness and acceptance. The injury must be performed with intraoperative electrophysiological monitoring of the nerve to be injured and must include 50–80% of the spastic muscle fibers to expect an effective result [5]. The most relevant long-term complications of the neurotomies are allodynia and neuropathic pain. To reduce the probability of the appearance of these adverse effects, it is important to identify and try to avoid sensory fibers during the procedure [17].

4.1.2 Selective dorsal rhizotomy

Sir Charles Sherrington, in 1898, showed that stiffness could be abolished by dorsal rhizotomy in a feline model with midbrain transection [18]. With this background, Otfrid Foerester, in 1913, reported the first dorsal rhizotomy for the management of lower limb spasticity in patients with cerebral palsy [19]. Rhizotomy consists of the selective section of the dorsal roots of the spinal nerve at a specific medullary level. It is thought that its effect is due to the reduction of the afferent information of the *Ia* fibers of the spastic muscle, which would produce a decrease in the excitatory input, and an increase in the inhibitory activity of the interneurons, to the alpha motoneurons [18, 19]. Dorsal rhizotomy is indicated in patients with diffuse or regional spasticity, in patients with spasticity in one or two limbs. There is no consensus on the precise selection criteria, but it is most frequently performed in paraplegic patients with spasticity in the lower extremities, however, it has also been performed successfully in the cervical region in patients with spasticity in the upper extremities [20, 21].

The procedure for the lower extremities is usually performed on the posterior roots of L1-S2 levels, exposed with a laminectomy or laminoplasty. For spasticity of the upper extremities, rhizotomy has been described from C1-C3 levels, not sectioning dorsal root of C4 to avoid affecting the diagrammatic function [20, 21]. Similar to neurotomies, it is important to perform intraoperative electrophysiological mapping to identify the roots that contribute to spasticity. In cases of pediatric

cerebral palsy, it has been observed that the patients with the greatest improvement are those between 4 and 7 years old and have a preoperative gross motor function measure test (GMFM-88) between 65–85% [22]. The most important side effects of this procedure are impaired sensation, sphincter dysfunction, cerebrospinal fluid fistula, and chronic low back pain [17]. Techniques with more limited and selective lesions have been tried to improve the results and reduce the probability of the appearance of side effects [7].

4.1.3 DREZotomy

This procedure was initially performed by Sindou, in 1972, for the surgical treatment of pain. He observed that this technique also produced important hypotonia in the muscles corresponding to the severed medullary segment, and suggested its application in cases of spasticity [23, 24]. This surgery is similar to rhizotomy, but the injury here is made in the spinal cord, at the Dorsal Root Entry Zone (DREZ). The underlying mechanism in this case, is a disruption of *Ia* afferent inputs to the dorsal horn and a disruption of local circuits that contribute to muscle tone [6]. This procedure is indicated in cases of severe regional spasticity, especially those associated with pain and poor or no regional function, such as in paraplegic or hemiplegic patients with painful hyperspasticity or severe spasms [25, 26]. The surgery, which requires an intradural approach and adequate visualization of the posterior surface of the spinal cord, consists of 3-mm deep incision at the dorsolateral sulcus, down to the dorsal horn, following its axis. When spasticity is associated with focal dystonia, DREZotomy should be more deeply down to the base of the ventral horn [5]. DREZotomy can be performed at the C5-C8 medullary levels (at 35° angle) for the management of spasticity in the upper extremities or at L1-S2 levels (at 45° angle) for the lower extremity affection. The most important complications of this technique are damage to the pyramidal pathway with loss of strength and severe hypotonia, so it should be considered in patients with severe refractory spasticity who have little residual function in the limb.

4.1.4 Longitudinal myelotomy

Bischof originally described the longitudinal myelotomy in 1951, and it was subsequently performed more selectively by Pourpre in 1960, and by Laitinen & Singounas in 1971 [8, 27, 28]. This procedure consists of a frontal separation between the ventral and dorsal horns at the level of the lumbosacral enlargement. The goal of surgery is to interrupt the spinal reflex arc by severing the connection between the posterior and anterior horns of the spinal cord. Through a T9-L1 laminectomy or laminoplasty, the procedure is performed at the T11-S2 medullary levels. Once the spinal cord is exposed, a posterior longitudinal sagittal incision is made deep to the central canal prior to performing a transverse cut using a stylet with a right-angled extremity, to separate the ventral and dorsal horns [7]. This surgery has been used in the treatment of patients with paraplegia, especially in cases with triple flexion and loss of sphincter function [6].

4.1.5 Other procedures

Some stereotaxic procedures in the thalamus and cerebellum have been performed for the treatment of spasticity in selected cases. These lesioning procedures include ventrolateral thalamotomy, pulvinarotomy, dentatotomy, and lesion of nucleus fastigii [29]. Due to the complexity, risks and lack of better effectiveness compared to the surgical options for spinal cord and peripheral nerve, these procedures were abandoned [6].

On the other hand, orthopedic surgery is another lesioning option. Neurosurgical procedures are the first choice for the management of spasticity and dystonia, but orthopedic surgery is a complementary surgical option for cases in which spasticity persists after neurosurgical treatment. Orthopedic surgery can reduce spasticity by releasing or lengthening tendons in the affected region. Orthopedic procedures may be indicated primarily when contractures and ankyloses are predominant or like the last option when the deformity is so strong [6].

4.2 Neuromodulation treatment

The classic procedures to treat spasticity are focused to perform lesions or ablative brain, spinal or nerve surgeries, but in recent times it exists the opportunity of neuromodulation. This term involved the use of chemical or electric pulses to increase or decrease neuron threshold with the main goal to diminish or abolish neurological or/and psychiatry symptoms. Neuromodulation is widespread to treat neuropathic and phantom pain, movement disorders abnormalities like Parkinson's disease, essential tremor and dystonia; partial and generalized epilepsy; obsessive–compulsive disorder, depression or anorexia; motor problems of neurogenic bladder, and neurologic deafness and blindness.

In this sense, neuromodulation in spasticity is concerned mainly to the use of infusion pumps, treatment acknowledged all over the world. Nevertheless, it exists the possibility to utilize electric stimulation in two targets: spinal cord and cerebellar sites to ameliorate the stiffness limb, but is not spread like infusion pumps.

4.2.1 Infusion pumps in spasticity

The next lines are dedicated to resume the infusion pump in the treatment of spasticity. It should be said that there is no cure to spasticity, but different methods and treatments can be useful. Infusion pumps are generally described as: "Complicated electromechanical systems that are used to deliver anesthetic drugs with moderate precision" [30]. Regarding neurosciences, different drugs are used in order to help patients with spasticity and other similar illnesses, mainly baclofen. Since 1984, the usage of infusion pumps with baclofen was proposed by Richard Penn due to prior scientific evidence that this chemical could work as an analogue of "γ-aminobutyric acid" (GABA) in its B receptor, how it is mentioned [31].

Humans can be treated effectively with intrathecal baclofen to decrease drastically the symptoms, and the burden that this illness means. Baclofen is a GABAergic drug that is transmitted intrathecally by infusion to the subarachnoid space. Although baclofen can be taken orally, the quickness and effectiveness of the chemical is decreased in comparison with the utilization of pumps to treat patients with spasticity. When taken orally, baclofen can have as a maximum dosage approximately 360 mg per day, but when infused intrathecally the dosage reduces to approximately 250–500 μg per day. Although, Baclofen is a very effective chemical it also has some repercussions like muscle weakness and slowness in walking speed due to the effects that induces, but they can be controlled if the dose is adequate for the patient (**Table 4**) [32].

Overall, the experience of scientists and neurosurgeons using intrathecal baclofen can be described as a positive one, but most important good results have been shown for patients with spasticity. In **Table 4**, it can be seen how several authors have been using this technique to improve the development of the pathology. The former table contains twelve different categories in which all the authors present the results obtained by the research. In the "year" column it can be seen when the paper was published, and in the "number of patients" the amount of people that took participation in the research. In general, the "follow-up" period

Number	Author	Year	No. patients	Follow-up "months"	% improvement	Ashworth PRE	Ashworth POP	Other Scale PRE	Other scale POP	Baclophen's Dose	Other Dose
1	R D Penn	1989	n = 20	19.2(10–33)	70%	4(1)	1.2(0.4)	NS	NS	100–150 μg/day	NS
2	P. G. Loubser	1991	n = 9	3–22	69.31%	3.78(1.34)	1.16(0.48)	Reflex: 3.57(1.05)	Reflex: 0.64(0.87)	535.8(269) μg/day	NS
3	R. Becker	1996	n = 18	13–54	48%	4.5	2.33	PSFS: 2.16	PSFS: 0.94	265 μg/day	NS
4	Alexei I. Korenkov	2002	n = 12	12	48%	4.2	2.2	NS	NS	180(65–280) μg/day	NS
5	Daniel Guillaume	2005	n = 138	12	51.24%	4.02(0.92)	1.96(0.78)	NS	NS	288 μg/d	NS
6	Giulia Stampacchia	2016	n = 14	12	86%	3.5	0.5	NS	NS	250.5(187.5) μg/day	NS
7	Tanja Kraus	2017	n = 13	60 (12–100)	55.26%	3.8	1.7	NS	NS	According patients' needs: pediatrics	NS
8	Mithra B Maneyapanda	2017	n = 42	36	NS	NS	NS	NS	Functional Independence: 13 (33%)	605.89(333.1) μg/day	NS
9	Elke Pucks-Faes	2018	n = 116	64.4(40.7)	50%	4	2	NS	NS	152.7(76.8) μg/day	NS
10	Pedro Videira Reis	2019	n = 155	96 (9–132)	75%	4(3–4)	1(1–2)	PSFS: 4(3–4)	PSFS: 1(0–1)	230(95 to 400) μg/day	NS

Table 4.
Comparison between authors according to baclofen treatment in this table it has been merged information from different articles that inform about intrathecal baclofen usage [14]; [33–41]. NS = not specified.

was variant, but the tendency as years pass is determined to give the patient a longer period between the follow-up in comparison with the older papers. It should be said, that when analyzing the Ashworth Scale comparing the preoperatory and the post-operatory, results show that using intrathecal baclofen can help reduce drastically the stiffness to a slight increased muscle tone in the majority of the patients.

Regarding the "dose", the difference between pediatric and adult treatment should be addressed to understand how to apply intrathecal pumps. Some factors in children may affect the treatment, that is why there should be a pediatrician involved in the process of treating infants in order to apply the correct dosage and time to realize the pump implantation, and do not interfere with their development. In adults, it can be said that covering a good dosage is easier because developed organisms can receive intrathecal treatments better with doses from 200–600 μg/day. Moreover, due to biological variability different patients need more baclofen if they are still showing symptoms of spasticity, and less in the case of having problems with movement and also muscle weakness; that is why the doctor should be ready to evaluate when to adjust the dose of the drug according to patients' requirements.

Authors with long number of participants tend to have similar results, that is why it is important to have a correct number of participants and dose to generate a more accurate investigation. The dose of those investigations with more than a hundred participants express that the amount of baclofen needed to help a patient can approximately be 220 μg/day as a good reference to start the drug's usage. Although, none of the authors used another drug in their papers it should be said that baclofen is an effective chemical that can help patients with spasticity infused intrathecally. Also, it is important that doctors have to analyze correctly the dose for a better performance of the pump in each individual, and avoid toxicity problems (**Figures 2–6**).

In the following years, neurosurgeons and their teams have been adding information to the methods and the correct usage of this technique, gathering approximately 35 years of experience in the field of infusion pumps and the usage of baclofen in order to treat spasticity.

4.2.2 Spinal cord stimulation

The first person who used SCS in the treatment of an illness was Shealy when he was neurosurgery resident in the 60's decade. In the 50's, the original idea was emerged after the use of battery connected to cardiac electrodes located in the animal's atrium and modified the myocardial electricity in the treatment of arrhythmias. After an experimental period, the use of a voltaic pile and heart electrodes brings the first pacemaker in man, and stablished one of the most important medical knowledge about implants in the human being until now.

Shealy thought this principle of pacemakers, first in dogs and after in humans, could be used in neurologic patients. He took the first patients with uncontrolled pain cancer, and he implanted in the spinal cord, a system similar to the cardiac patients. The results are the amelioration of pain. Indeed, in this moment Shealy was opened the door to Neuromodulation at the neurologic patients [42, 43].

With respect to spasticity, based in the experience of Shealy with SCS for pain treatment in 1973, Cook and Wenstein reported one Multiple Sclerosis (MS) patient with pain alleviation but with the fortuitous finding that also limb spasticity amelioration was presented [44]. In 1976, Illis in UK introduced percutaneous electrodes in two MS patients and peridural space was stimulated and improved spasticity [45]. In 1979, Richardson and cols wrote an article with six spastic patients, in which spasticity was measure by a scale similar to Ashworth, with 36.11% of improvement, and also for first time the article described SCS

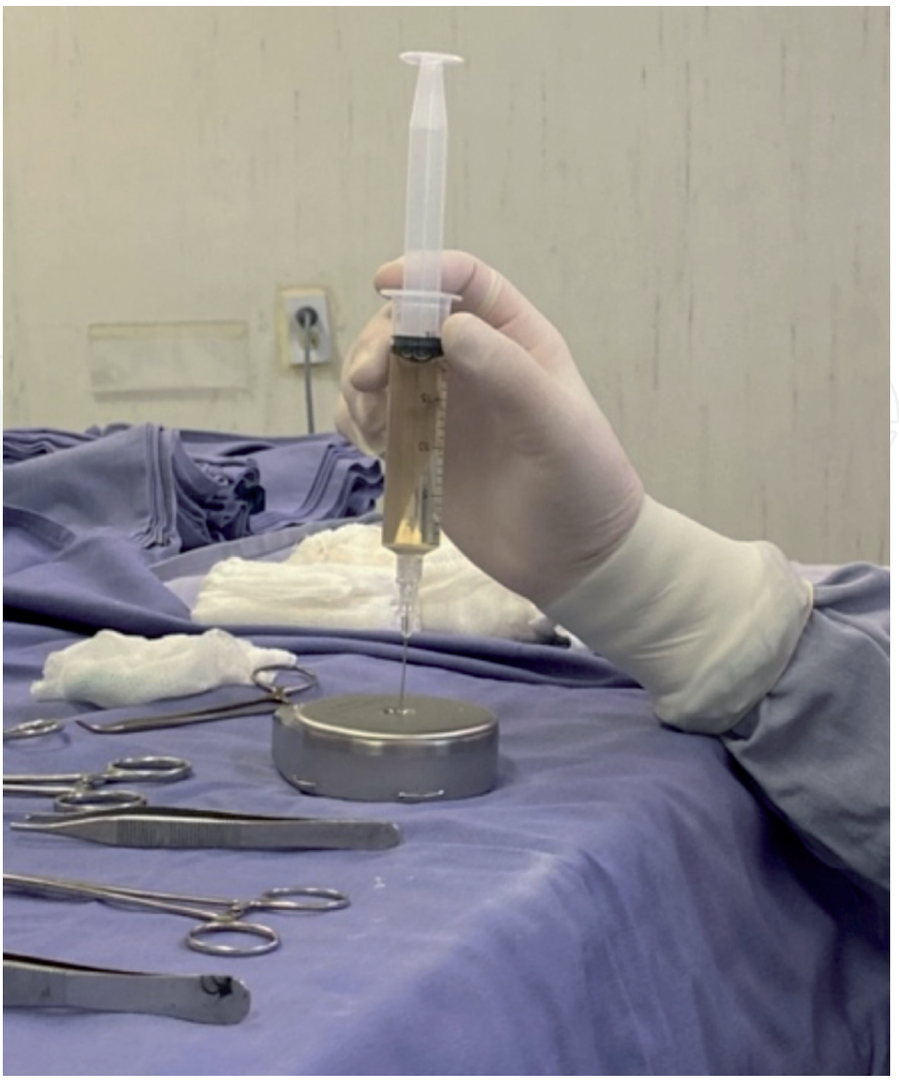


Figure 2.
It is shown how baclofen should be injected into the pump's reserve.

electric parameters of stimulation (Voltage from 0.5 to 2 V, frequency 33 to 75 HZ, and pulse width of 100 to 200 μsec) [46]. In 1980, Read and cols reported 16 MS patients: 9 had spasticity amelioration, 3 without change and 2 had worst evolution with increase in spasticity; the total alleviation was 56.25% in the group [47]. In the same year, Siegfried showed his experience with originally 26 patients using test stimulation before definitive implantation, consisting in percutaneous electrodes and depending if the test was positive, they implanted a definitive one. Only 11 patients were operated and followed 3 years with amelioration of the spasticity. Best results depending of spinal and partial damage more than cerebral site of spasticity. They also used electrophysiological measures like H-reflex to contrast the amelioration [48]. Also, in this year, Dimitrijevic showed the results of 11 patients with spasticity that improved 56.56% after SCS, mainly clonus and EMG patterns. In 1985, Barolat-Romana reported 6 spasticity patients with spasms and the immediate alleviation after SCS [49]. In 1986, Dimitrijevic and cols studied 59 patients in which spasticity was reduced in 63% of the group. They found spasticity was controlled better if the electrode was located below the spinal lesion more than above, also patients had better results with partial lesion that with complete ones [50]. In 1986, the same group with Campos (like the first author) described 8 patients with minimal function of posterior columns, 90% responded to the

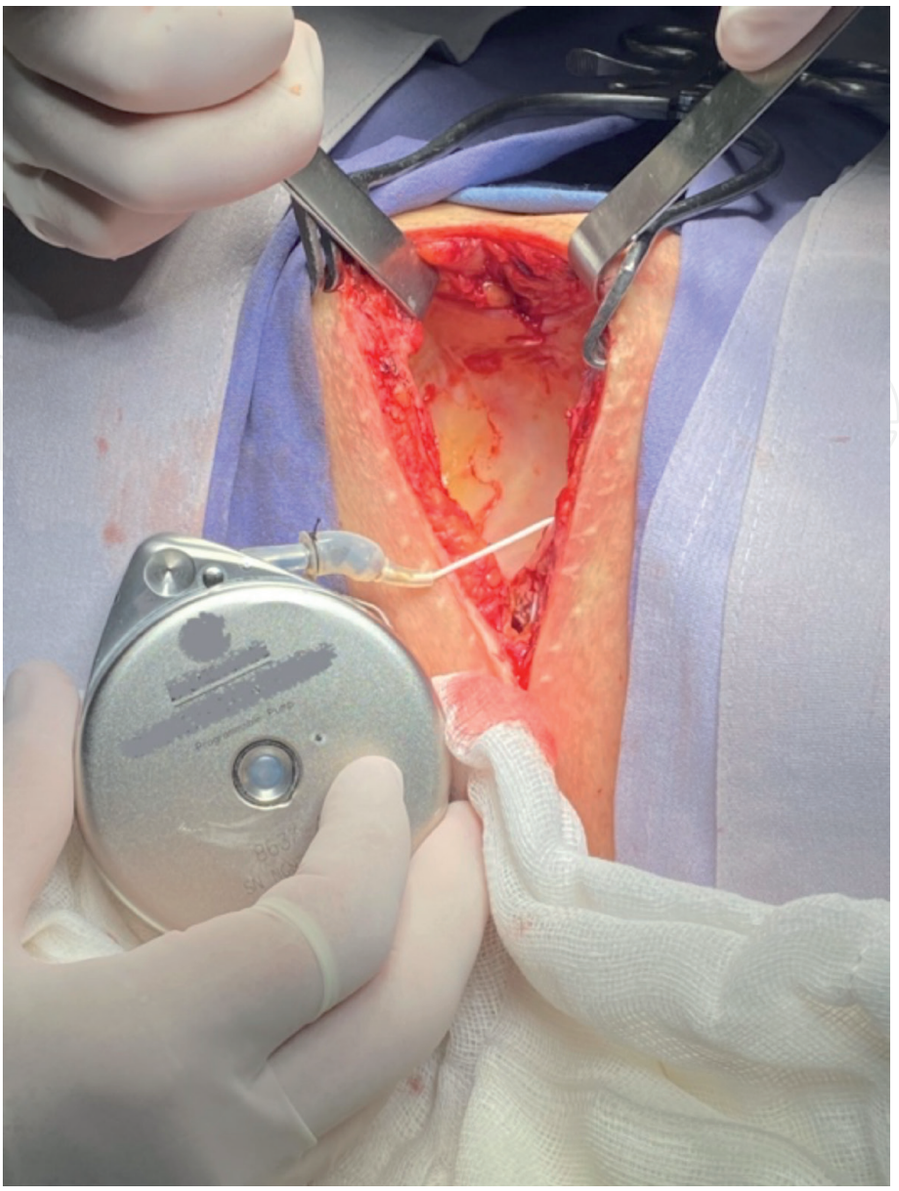


Figure 3.
It is demonstrated how the pump should be implanted, so as the stitch used in order to seal the neck of the gadget with the catheter.

epidural stimulation [51]. In 1988, Barolat made a clinical trial expanded the initial experience to 16 myelopathic subjects with amelioration of spasm and clonus, inclusive from one year of follow-up [52]. In 1993, the same group was amplified the experience of 509 plates implanted in patients suffered pain and spasticity: 350 in the whole group, 227 for pain, 105 for motor disturbances (spasms/spasticity following spinal cord or cranial trauma, multiple sclerosis, cerebral palsy, spasmodic torticollis and other motor problems) and 18 patients with both condition: pain and spasticity. From these, 3.4% had infection, 1.1% with electrode migration and less of 1%, breakage [53]. In the decade of 1990's it existed poorly advanced in this area. In 2000, Pinter and Dimitrijevic discovered that severe spasticity in paraplegic patients could improve with the electrode's position upper to the spinal lesion, with frequency of 50–100 Hz, 2–7 Volts and 210 μsec and adapted depended of the case [54]. In 2015, Dekopov and Russian team evaluated two groups of spasticity patients: Cerebral palsy and spinal cord lesion. SCS ameliorated Ashworth scale (58.8%) in cerebral palsy group, but it did not for the spinal cord lesion [55].

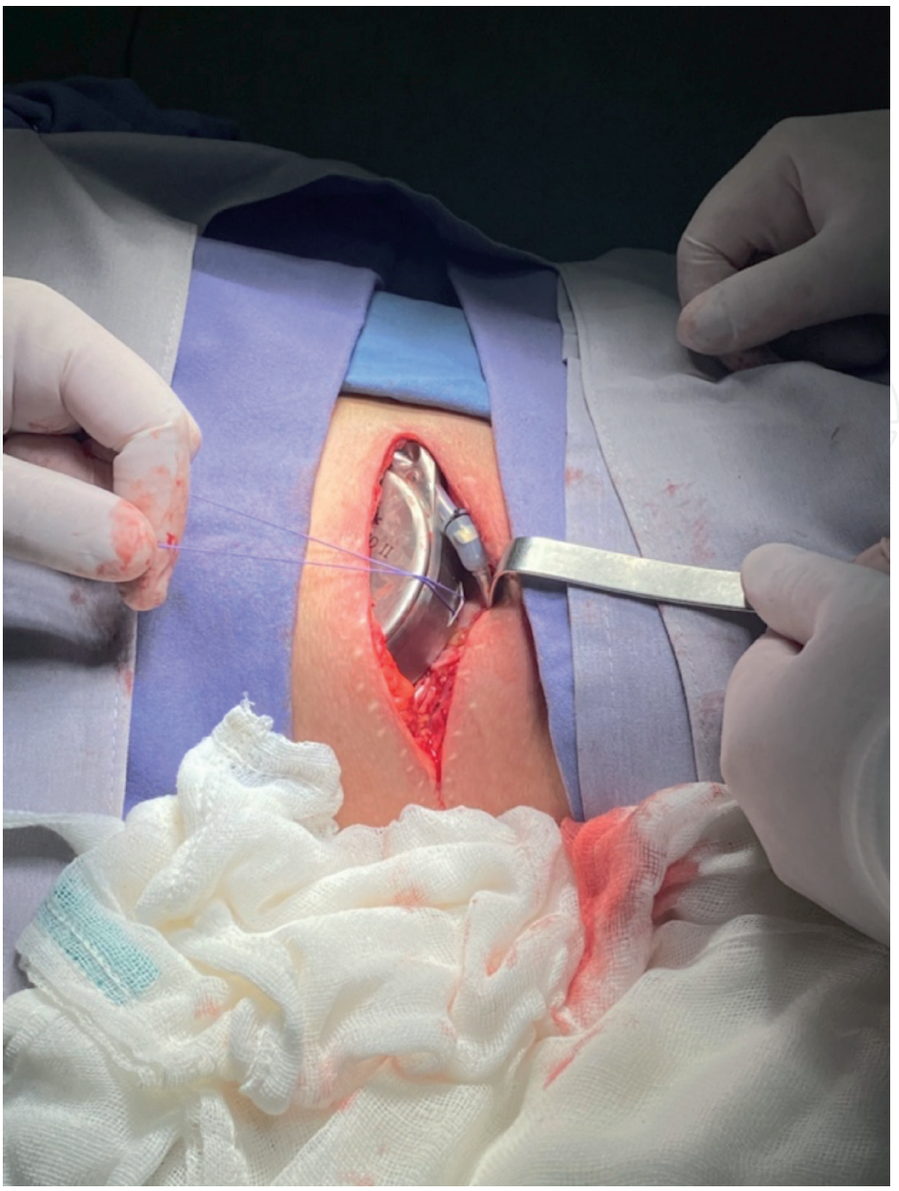


Figure 4.
In the implantation process is important to attach the pump handles to the tissue surrounding the equipment in order to stabilize the device and prevent future complications.

4.2.3 Cerebellar stimulation

The understanding of cerebellar human stimulation began in the experiments developed in rats, cats, dogs and monkeys. It was based in these antecedent that Cooper and cols in 1973 located electrodes on the anterior and posterior surface of cerebellum to treat not only spasticity, but seizures also [56–58]. After these reports, Cooper and his group contribute significantly to this neuromodulation area with different types of articles including implantation technique, clinic evolution, surgical complications, neurophysiological changes, psychological reactions, between other issues [59–64]. Other groups started to perform CS. In 1977, Manrique and cols implanted 4 patients with good results to diminished spasticity [65], Penn found in some patients diminished spasticity and, in other, no changes [66, 67]. Cooper's work was continued by Davis [68–73] spreading the experience in this field. In 2003 and 2007, respectively, Galanda & Horvath proposed new insights about CS [74, 75]. In the last decade there has been no progress in this area.

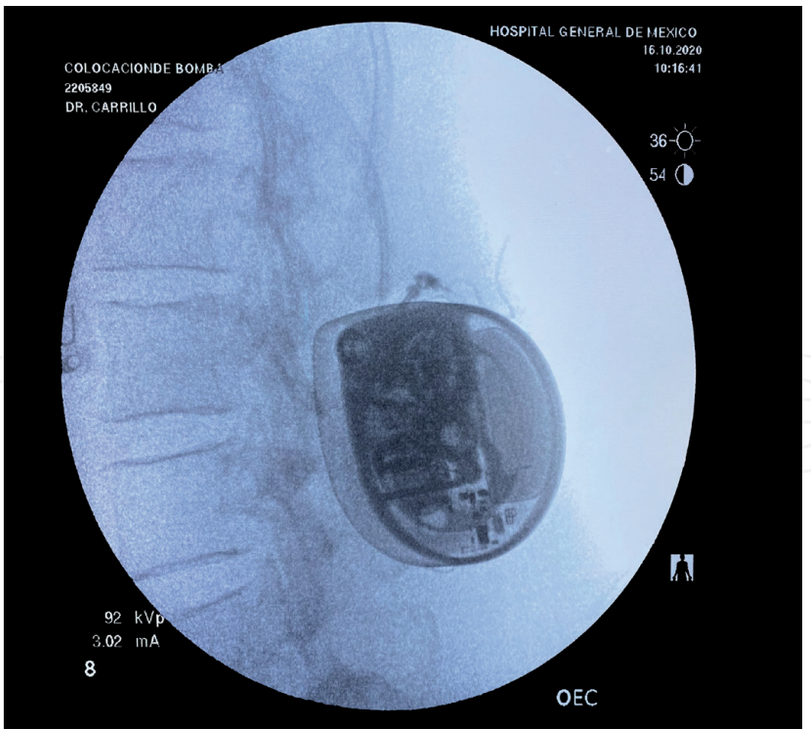


Figure 5.
Post-operatory picture of the pump immediately after the implantation surgery. X-ray image must be taken to verify the correct placement of the equipment.

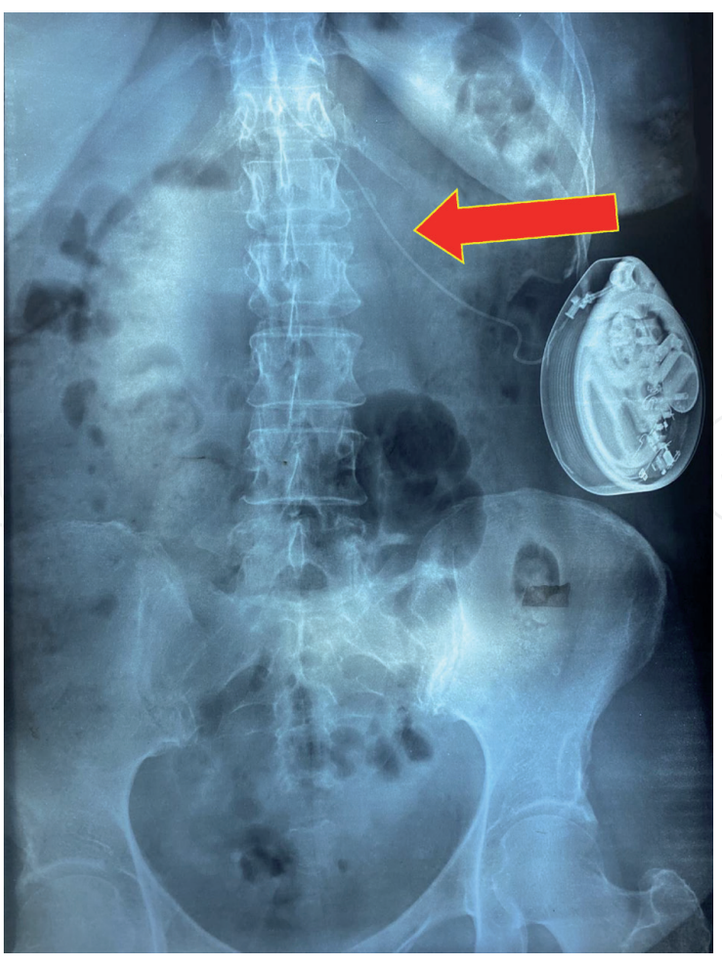


Figure 6.
X-ray image shows the infusion pump implanted in the lower part of the left hypochondria. The red arrow indicates the catheter connected to the infusion pump, and the tip goes to T-10 level into the subarachnoid space.

5. Post-surgery considerations

After any surgical procedure, physical therapy and recovery are very important. There is no consensus on the duration and specific type of rehabilitation that should be followed. There is great variability in the literature, but some authors recommend close follow-up to 3 months after hospital convalescence [16]. The physical therapy and rehabilitation protocols must be adapted to each patient, and so should be empathized that is crucial to promote functional improvement after surgical management. The results and their functional impact on patients can be seen in variable times, and the follow-up should be at least for 6 months. All management and follow-up of the patient with spasticity should be carried out, whenever possible, by a multidisciplinary team to promote the best results, as well as avoid possible problems [5].

6. Other techniques and targets

Recently, it exists other possibilities to treat spasticity. It is only mentioned to avoid extend this review. For one side, magnetic transcranial or spinal stimulation. These techniques mean the performance of a coil connected to an electric source to produce electromagnetic waves modifying the brain's plasticity: primary motor cortex or cerebellar cortex are the selected targets; or in an experimental manner in spinal circuitry.

On the other hand, electrical transcutaneous stimulation over the limb or spinal cord try to ameliorate also spasticity. It is remarkable that neurosurgical procedures in spasticity are the second line of treatment, when rehabilitation maneuvers were excluded. After the surgery has been performed the use of physical exercises is mandatory. To finish, it should be said that orthopedic procedures must be done when the extremities are deformed.

Conflict of interest

The authors declare no conflict of interest.

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