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Stretching exercises in managing spasticity: effectiveness, risks, and adjunct therapies

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Abstract

Spasticity is a component of upper motor neuron disorders and can be seen in neurological conditions like stroke and multiple sclerosis. Although the incidence rate of spasticity is unknown, it can put pressure on the health condition of those with spasticity, and there is no absolute effective way to control it. In the past, stretching exercises were an accessible tool for physical therapists to manage and control spasticity, but opinions on the optimal dose, after-effect, and mechanism of effects were controversial. Therefore, this article tries to provide an overview of the effectiveness and risks of stretching exercises. Furthermore, there are several adjunct therapies, such as brain stimulation and botulinum injection, that can increase the effectiveness of a simple stretch by increasing cortical excitability and reducing muscle tone and their role is evaluated in this regard. The results of this study propose that several prospective and case studies have demonstrated the benefits of stretching to control spasticity, but it seems that other methods such as casting can be more effective than a simple stretch. Therefore, it is better to use stretching in combination with other therapeutic regimes to increase its effectivity.

Key Words: muscle spasticity, brain, stretch, botulinum toxin, noninvasive brain stimulation.

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Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) accompanied by increased tendon twitching as a result of hyperexcitability of the stretch reflex. This phenomenon is part of the spectrum of upper motor neuron syndrome, which can manifest as either intermittent or sustained involuntary muscle activation.[1,2]

Spasticity is typically seen following neurological conditions such as stroke, Multiple Sclerosis (MS), cerebral palsy, and brain injury. Although it affects the health status of these people, the exact incidence of spasticity is unknown,[3] but it is estimated that 38% of those with stroke (after 12 months),[4] 60-90% of those with MS,[5] and 17% of those with traumatic brain injury (one in six people)[6] experience it. Spasticity may primarily affect the elbow joint followed by the wrist and ankle joint.[7]

In addition to being influenced by velocity, spasticity is also influenced by length for instance, a short quadriceps muscle has greater spasticity than a long quadriceps muscle. Moreover, when muscles are in a long position, spasticity is greater in the flexors of the upper extremities and the ankle extensors.[1,2]

The results of spasticity could be both negative and positive. It allows the sufferers to transfer and move independently as the weak spastic limb allows positive weight bearing.[3] As it can result in better function, standing and walking, it can consequently protect the limb against deep vein thrombosis.[8]

However, it does not mean that spasticity can always result in better function. The restrictions in movement and Range Of Motion (ROM), abnormal limb posture and motor control, dysregulation of voluntary movements, increased resistance of muscle following a passive stretch, pain, ankylosis, tendon retraction, and muscle weakness are known to be the negative effects following spasticity.[3] These restrictions can affect the quality of activities in daily living for the patient and decrease the success rate of rehabilitation.[7,8]

Stretching, mechanism and effect

Aim and mechanism of stretching

Stretching aims primarily to augment the viscoelastic components of the Muscle Tendon Unit (MTU), in an attempt to decrease the likelihood of sustaining muscle tendon injuries. Despite its universal application in the sphere of rehabilitation, the mechanisms through which it acts remain elusive. As far as our current knowledge goes, stretching increases the extensibility of soft tissues by eliciting viscous deformation and structural adaptation in muscles, tendons, dermal tissues, and neural tissues. This is particularly beneficial when combined with other interventions such as orthosis, serial casting, surgical procedures, or pharmacological treatment. These can provide joint stability, and reduce the risk of contracture and deformity.[9–12]

In general, elastic structures revert to their initial length once the applied stretch is released; however, the situation for muscle is divergent owing to its viscous properties; consequently, this structure elongates more gradually. Should a muscle be subjected to prolonged stretching or allowed inadequate recovery time before a second or new stretch, the MTU will not revert to its original length. This can increase the number of sarcomeres as well.[9,10,13–15]

The establishment of concrete evidence to determine the efficacy of stretching is challenged by methodological restrictions.

In the event of contracture, an elongated muscle will neither maintain nor increase sarcomeres if the length of the tendon or the elasticity of the connective tissue remains constant, although the number of sarcomeres can be preserved. Although immobilization can conserve muscle length, it can result in the loss of sarcomeres of its antagonist, the accumulation of connective tissue, and as a result a decrease in elasticity. Connective tissue does not appear to accumulate in the presence of contractile activity.[3,9,15]

Efficacy of stretching

Long-term low-force stretching induces plastic deformation in connective tissue, an effect that is amplified when the connective tissue is heated before stretching and cooled after stretching ceases. Enhancing ROM is the main objective of stretching, but there are additional alterations to functional parameters such as maximal isometric torque, muscle-tendon stiffness, Passive Resistive Torque (PRT), and structural parameters, including muscle and tendon stiffness, fascicle length, and pennation angle that can occur after stretching.[3,9,15,16]

In the context of chronic stretching, two plausible explanations can impact the MTU. First, the sensory theory, suggests that the tolerance to stretch could potentially increase, implying increased passive tension after intervention, with no alteration in tension for a given length. Second, mechanical theory, indicates a reduction in joint resistance, such as a decrease in passive joint torque at a particular angle, possibly as a result of the mechanical properties or geometry of the MTUs.[17]

Based on previous research, an increase in ROM without any alteration in joint resistance was observed as a short-term effect (2-8 weeks) after stretching. Consequently, the chronic impact of stretching may also be associated with increased tolerance to stretching.[18] Furthermore, regardless of some basic factors, such as duration, evidence supports the idea that passive forces decrease after an acute stretching exercise, regardless of the stretching method.[16]

There are some contradictory results in changes in muscle and tendon stiffness. Some studies have reported a decrease in muscle stiffness without any change in tendon stiffness.[19,20] In contrast, other studies have found a decrease in tendon stiffness, with muscle stiffness remaining unchanged.[21,22] These discrepancies may be attributed to differences in the length and frequency of the stretching regimens implemented in different studies. One understanding could be static stretching for 60 to 120 seconds affects muscle tissues, while continuous static stretching for more than 10 minutes can affect tendon tissues as well.[16]

In a recent systematic review, it was shown that acute stretching had a significant moderate effect on MTU stiffness while there was no significant change for long-term stretching. The average duration of acute static stretching was reported as 248.6 seconds (approximately 4 minutes) in this study and the duration range for long-term stretching was 10-50 minutes per week for 2.9-12 weeks.[23]

In terms of time, it is shown that muscle stiffness can be reduced after 5.8 weeks [24] however, these data are consistent with a healthy population, and to the best of our knowledge, we couldn't find the relevant information consistent with spasticity.

Principles for effective stretching

In the context of cases of Cerebral Palsy (CP), reports suggest that muscles exhibit greater stiffness than tendons. Despite possible muscle and fascicle elongation during stretching, the degree of stretch could not sufficiently alter anticipated properties, such as fascicle length or reduced connective tissue.[25] The results regarding the type of stretch for these cases are still contradictory. Kruse *et al.* reported MTU elongation after acute static stretch by 14.6% and a decreased MTU elongation after PNF stretching by 8.8%.[26] This finding is in contrast to healthy studies that reported decreased muscle stiffness following both types of stretch.[19,27,28] In contrast to the acute effects of stretching, Fosdahl *et al.* reported non-significant data for 16-week stretching and resistive training.[29]

Although we lack information on the response of spastic tendons to stretching, current evidence indicates that in healthy individuals, stretching does not provide adequate stimulus to modify tendon properties and effect elongation compared to resistance training.[25,30,31] A recent study on MS patients also suggests that there is no priority for daily stretching exercises as the first line of spasticity management.[32]

Theis *et al.* demonstrated a reduction in joint stiffness by 32% and muscle stiffness by 12% over a 6-week passive stretching regimen, although they did not document any changes in mechanical properties such as tendon length or resting fascicle length. However, this does not exclude potential alterations in other tissues, such as microfilaments

or titin. They also noted an increase of 23% and 13% in muscle and fascicle stress, respectively.[25]

Nakamura *et al.* reported a 13% reduction in passive joint stiffness after a four-week stretching regime.[33] It is worth noting that muscle belly length differs from muscle fascicle length in pennate muscles, with a reduced muscle belly length suggesting muscle contracture.[34]

Some indicators point toward alterations in specific structural and mechanical properties, such as changes in fiber size and distribution, proliferation of extracellular matrix, and changes in stiffness in both spastic muscle cells and extracellular material in spastic muscle. However, barring muscle fascicle length in gastrocnemius muscle in individuals with spastic CP versus comparison individuals, there was a general consistency in the findings in all studies that indicated that muscle volume, cross-sectional area, thickness, and belly length tend to be reduced in individuals with spastic CP.[34]

In a recent meta-analysis, Gomez-Cuaresma *et al.* posited that passive stretching required less time than other stretching methods and did not require external assistance from physical therapists. Although there are improvements in some variables, not all returned significant results to demonstrate their effectiveness.[9] Pradhan and Bansal also noted the necessity of multiple repetitions to manage spasticity and proposed 2.5-3 hours between these repetitions to prevent pain.[35]

Regarding neural and mechanical properties, Gomez-Cuaresma *et al.* suggested that the passive static stretching program decreased the Hmax/Mmax ratio in healthy individuals and those with spasticity.[9] However, functional exercise-based stretching programs are reported to increase the Hmax/Mmax ratio and reduce the latency of the H-reflex, with these changes lasting at least 2 months.[9]

Other reports indicated that 90 seconds of static stretching with five repetitions reduced muscle resistance through passive stiffness, peak torque, and stress relaxation. The reduction in unit muscle tension resistance reverted to baseline in 1 hour, except for stress relaxation. Shorter hold times, less than 60 seconds, and fewer repetitions did not produce such an effect; therefore, it can be inferred that changes in the viscoelastic properties of the muscle-tendon unit depend on the duration rather than the repetitions of the stretches. This is supported by evidence that even ten 45-second stretches per day for 3 weeks or four 45-second stretches twice daily, 7 days per week for 13 weeks, did not alter the mechanical or viscoelastic properties of the muscle.[36]

Regarding the type of stretch, static stretching of the Achilles tendon for six weeks significantly altered the passive resistance torque, while the ballistic stretching technique resulted in a significant decrease in tendon stiffness.[37] Furthermore, there is robust evidence that stretching for less than 7 months in cases with or without neurological conditions does not produce clinically significant outcomes in joint mobility.[38] Salazar *et al.* also elaborated that having static stretching and positioning in the rehabilitation program is better than no therapy for the wrist flexors.[39]

Thus, it can be concluded that different stretching techniques can yield varying results. Moreover, depending on the therapeutic goals, the appropriate stretching technique must be carefully selected as a valuable tool in the treatment process.

What are the potential risks of stretching?

Blisters, discomfort, skin abrasion, and bruising are potential risks associated with stretching.[15,40] Moreover, overloading and hyperextension can result in conditions such as tendinitis and tendinopathy.[41,42] At the molecular level, tendon failure may occur due to the stretching of collagen, the primary component of tendons. An elongation greater than 8% can lead to collagen fiber rupture and microscopic tendon failure. An elongation between 4% and 8% can cause rupture of collagen cross-links and sliding of collagen fibers past one another. Up to 4% elongation generally does not result in tissue damage, implying that it is well tolerated by tendon structures. Tension overload can manifest in muscles, musculotendinous junctions, or at the point where tendons attach to bones.[41]

Furthermore, there are three primary risk factors associated with muscle strain. These include advanced age, a history of previous injury, and muscle weakness relative to the contralateral side or the antagonist.[43] Given our understanding, in neurological conditions, one or a combination of these factors may be present. Therefore, the potential for muscle strain related to stretching must be carefully considered.

Supplementary treatments and techniques

Various treatments and techniques can enhance the effects and minimize the risk of muscle, tendon, or skin injury from stretching exercises, such as electrical stimulation[44] taping and casting,[45,46] acupuncture[47], and orthopedic surgeries such as selective dorsal rhizotomy[48] which are too invasive to enhance stretching. Various pharmacological options exist to control spasticity, including tizanidine, diazepam, clonidine, dantrolene, and baclofen[49–51] although these may also present potential side effects[51,52]. However, alternatives such as botulinum injections which are cost-effective[53], and non-invasive brain stimulation (NIBS) with fewer adverse effects compared to pharmacological options[54] appear to be better solutions for spasticity management.

Botulinum injection for sponginess control

The use of Botulinum Toxin (BTX) for the treatment of focal spasticity is effective and safe. It is suggested that by blocking the release of acetylcholine at the neuromuscular junction, causing temporary muscle paralysis, BTX can reduce muscle spasticity for 3-4 months. However, factors such as inadequate drug dosage, accurate muscle selection for injection, injection technique, and an unclear treatment objective can affect the outcomes of BTX injection and compromise its efficacy.[55–57]

In the lower extremity, spasticity typically affects the knee extensors, causing a stiff knee, while the plantar flexors of the ankle result in an abnormal presentation of the equinovarus foot. Although many muscles contribute to the spastic equinus foot, the gastrocnemius and soleus appear

to play a significant role, thus being targeted by BTX.[57] For the upper limb, BTX is the recommended first-line treatment for post-stroke survivors, although weakened and spastic wrist and finger flexor muscles can limit hand mobility for stroke survivors, causing some difficulties in motor function after reduced spasticity achieved by BTX injection.[58,59] According to review articles, BTX has been demonstrated as a safe and effective treatment for reducing spasticity in both the upper and lower extremities, particularly at higher doses, and also in improving muscle tone after a stroke or traumatic brain injury.[57,60] However, Farag *et al.* reported that there is insufficient evidence to support the use of BTX as an additional treatment to alleviate upper limb spasticity symptoms in children with cerebral palsy.[61]

Stretching is one of the most popular adjunctive treatments after BTX injection in both academic and non-academic (87.7% and 98.2% respectively) settings while the effectivity showed to be very low[62] and to our understanding, it is not yet clear that the stretching after injection is immediate or a home program for a longer time. The available strong data for the direct effectivity of stretch after BTX are scarce but according to Picelli *et al.*, if stretching is performed for less than 7 months, there would be no effect on joint mobility.[15] Carda *et al.*[63] compared the effects of casting, taping, and stretching before, 20 days after, and 90 days after BTX injection. Their results indicated that, except for the modified Ashworth scale, the stretching group did not change statistically in the 6-minute walk test, 10-meter walk test, passive ROM, and strength of the dorsal flexors and functional categories. Another study also mentioned that taping and electrical stimulation have higher effects compared to stretching after toxin injection.[64] Santamato *et al.*[65] used daily stretching combined with passive mobilization and palmar splint after injection for 10 days and compared this method with taping after injection, but no significant changes were reported for the stretch group in any of the studies. However, Giovannelli *et al.*[66] reported positive effects of physiotherapy after BTX injection. They did not use stretching exercises solely but had it in their daily 40 minutes of physical therapy routine, which is not a specific result to cite as the effect of stretching after injection in the management of spasticity.

The general idea behind stretching and increasing the passive length of the muscle could be the fact that adopting muscle elongation regimes whether it is manual stretch, serial casting, and/or posturing would be more attainable and easier to perform after the injection.[67] Easier stretch or changes in stretch reflex following BTX injection can be explained by denervating the extrafusal muscle fibers which are innervated by alpha motor neurons and denervating the intrafusal muscle fibers which affect the sensitivity of the muscle spindles to changes in the length of the muscle.[68] Therefore, it is possible to have more effects for simple stretching even immediately after the injection. However, there is no evidence to support the claim for immediate effects of stretching right after the injection of BTX. One reason could be the fact that the toxin works on extrafusal neuromuscular junctions more quickly than the intrafusal fibers meaning that the activity of the muscle spindles would be mediated later than extrafusal neuromuscular junctions.[68] Another possible reason could also relate to risks for muscle and joint injuries as it is shown that the toxin can cause muscle atrophy and weakness and consecutively lead to joint degeneration and osteoarthritis.[69] If the muscle tone is reduced right after the toxin injection, it is possible that an invasive, immediate stretching can impose a higher injury risk to the muscle, tendon, and joint.

Recently a new method of stretching, known as Guided Self-rehabilitation Contract (GSC), has been introduced to perform after BTX injection. This method consists of two parts. The first part is a high-load self-stretch below the pain threshold to the targeted antagonist muscle and the second part. The patient should perform the stretching at least 10 minutes per day. After this, unassisted rapid movements characterized by maximal amplitude are performed in brief succession against the antagonist to gradually reduce antagonist co-contraction over time.[70] There is a report regarding the effectivity of this technique combined with BTX injection to reduce the adverse effects of spasticity deeply and more significantly and the result conveys that this could be a promising technique.[71] However, to our knowledge, it can directly increase the risk of soft tissue injuries for the patients. Moreover, there is no evidence to compare the effects of GSC with a simple conventional stretch program, therefore, Using GSC instead of conventional stretching programs might not be a priority.

Brain stimulation as a spasticity control approach

NIBS has recently emerged as a novel means of controlling spasticity, with Transcranial Magnetic Stimulation (TMS) and transcranial Direct Current Stimulation (tDCS) being the most common stimulation methods. Their effectiveness in spasticity control is potentially due to neuroplastic changes in the cerebral cortex resulting from the excitatory effect of stimulation in the stimulated area, which means that NIBS could modulate motor cortex excitability and decrease the excitability of motor neurons in the spinal cord. TMS and tDCS appeared to be the most prevalent brain stimulation conditions. The possible explanation behind their effectiveness in controlling spasticity is that neuroplastic changes occur in the cerebral cortex due to the excitatory effect of stimulation in the stimulated area.[72] The results of previous studies remain inconclusive. A systematic review and meta-analysis demonstrated that TMS and tDCS significantly reduced spasticity in patients, where low frequencies of TMS stimulated the unaffected hemisphere, and anodal tDCS stimulation proved more effective in this group of patients.

In tDCS studies, researchers used 0.7 mA- 2mA tDCS, with 0.7mA and 1.2mA showing significant effects but 2mA intensity did not reach the significance level in stroke patients. [72] Other studies suggest that the ipsilesional primary motor cortex (C3/C4 region based on 10/20 International Electroencephalogram Coordinate System) area is a reliable target for tDCS stimulation in various groups of patients, such as those with CP, stroke, and MS.[73–78] However, the optimal dose and positioning of NIBS remain

unknown but could serve as a possible supplementary treatment method to improve the effects of stretching exercises to effectively reduce spasticity. To our knowledge, tDCS is more practical than TMS as it does not require much space, is user-friendly, and the device itself is more affordable for both patients and clinicians.

Conclusions

In conclusion, there remain uncertainties regarding the mechanisms involved in the stretching of spastic muscles. Despite conflicting evidence regarding its effectiveness and possible injury risks, the straightforward and cost-effective nature of stretching may still be a valuable tool for physical therapists in their rehabilitation programs. It is recommended that clinicians favor the effects of acute static stretching more than long-time stretching and it can affect the muscle, tendon, and MTU stiffness. Moreover, NIBS or techniques such as BTX injection can optimize the effects derived from individual or multiple stretching exercises to improve function and decrease spasticity. However, further research is needed for a better understanding of the mechanisms underlying stretching and adjunctive therapies for stretching exercises for patients with spasticity.

List of abbreviations

MS, multiple sclerosis
MTU, Muscle Tendon Unit
ROM, range of motion
CP, cerebral palsy
NIBS, non-invasive brain stimulation
BTX, botulinum toxin
GSC, guided self-rehabilitation contract
TMS, transcranial magnetic stimulation
tDCS, transcranial direct current stimulation

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MGL, project design, supervision, writing (editing); MMJ, literature research; MMJ writing (first draft); PV, writing (editing).

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Conflict of interests

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