Annals of Physical and Rehabilitation Medicine 62 (2019) 220–224

Available online at ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com

Original article

Botulinum toxin type A or selective neurotomy for treating focal spastic muscle overactivity?

Thierry Deltombe [a,*], Thierry Lejeune [b,c], Thierry Gustin [d]

[a] Department of Physical Medicine and Rehabilitation, CHU UCL Namur site Godinne, B-5530 Yvoir, Belgium
[b] Université catholique de Louvain, Secteur des Sciences de la Santé, Institut de Recherche Expérimentale et Clinique, NMSK, avenue Mounier 53, B-1200 Brussels, Belgium
[c] Cliniques universitaires Saint-Luc, service de médecine physique et réadaptation, avenue Hippocrate 10, B-1200 Brussels, Belgium
[d] Department of Neurosurgery, CHU UCL Namur site Godinne, B-5530 Yvoir, Belgium

ARTICLE INFO

Article history:
Received 16 January 2018
Accepted 27 July 2018

Keywords:
Hemiplegia
Muscle spasticity
Motor nerve block
Neurotomy
Equinovarus foot

ABSTRACT

Objective: To discuss the effectiveness, indications, limitations and side effects of botulinum toxin type A and selective neurotomy for treating focal spastic muscle overactivity to help clinicians choose the most appropriate treatment.
Methods: Expert opinion based on scientific evidence and personal experience.
Results: Botulinum toxin type A can decrease muscle tone in different types of spastic muscle overactivity, which allows for treating a large variety of spastic patterns with several etiologies. The toxin effect is sometimes insufficient to improve functional outcome and is transient, thereby requiring repeated injections. Selective neurotomy is a permanent surgical treatment of the reflex component of the spastic muscle overactivity (spasticity) that is effective for spastic equinovarus foot. The neurotomy provides a greater and more constant reduction in spasticity. However, the long-lasting effect on the non-reflex muscle overactivity, especially dystonia, is doubted. The effectiveness, clinical indications, advantages, side effects and limitations of both techniques are discussed.
Conclusion: Botulinum toxin type A has the highest level of evidence and the largest range of indications. However, the botulinum toxin effect is reversible and seems less effective, which supports a permanent surgical treatment such as selective neurotomy, especially for the spastic foot. Further research is needed to compare the effect of botulinum toxin type A and selective neurotomy for the different types of spastic muscle overactivity and clinical patterns.

1. Introduction

Spasticity frequently affects patients with neurological disorders and participates in motor impairment that emerges following a lesion to central motor pathways. It can be detected in 25% of the patients as early as 1 week post-stroke [1]. The prevalence of post-stroke spasticity in the chronic phase ranges from 17% to 43% [2]. Increased muscle tone, severe arm paresis, hemihypoesthesia and low Barthel index score are predictors of severe post-stroke spasticity [2,3]. The association of muscle weakness (paresis), muscle contracture and spasticity are called the deforming spastic paresis syndrome [4].

As a component of upper motor neuron syndrome, spasticity was first defined by Lance et al. as a motor disorder characterized by a velocity-dependant increase in the tonic stretch reflex (muscle tone) with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex. A recent proposal suggested renaming spasticity to muscle overactivity or spastic muscle overactivity (SMO), including spasticity (an increase in the velocity-dependent response to muscle stretch measured at rest, e.g., triceps surae), spastic dystonia (tonic muscle activity at rest sensitive to stretch, e.g., finger flexors), associated reactions (abnormal contraction distant from the muscles involved in voluntary effort, e.g., dynamic elbow flexum) and spastic co-contraction (inappropriate antagonistic contraction sensitive to stretch during voluntary agonist command, e.g., triceps surae contraction during activation of the tibialis anterior) [5]. SMO does not include dystonia defined as an involuntary muscle contraction, which in contrast to spastic

* Corresponding author.
E-mail address: thierry.deltombe@uclouvain.be (T. Deltombe).

https://doi.org/10.1016/j.rehab.2018.07.008
1877-0657/

dystonia is not sensitive to stretch. However, the term "spasticity" is still largely used, even in the literature, to define all these forms of SMO, which adds to the confusion. Indeed, the treatment strategy should probably differ according to these different types of SMO.

In association with other impairments (weakness, sensory loss, muscle contracture, cognitive deficits etc.), SMO frequently worsens the patient's condition by affecting passive function (hygiene and increased carer burden) and active function (reduced mobility and inability to use limbs in function). SMO participates in pathological patterns such as elbow flexum, spastic hand with prehension difficulty and/or hygiene problems, adducted hip, stiff knee gait, spastic foot or claw toes. It affects patient's participation and quality of life (reduced social interaction and poor self-esteem) [6]. A large consensus supports an adequate SMO treatment strategy as possibly helping patients [7]. Such a strategy includes physical therapy, orthosis, oral medications, chemical neurolysis with phenol-alcohol or botulinum toxin type A (BoNT-A) injections, selective neurotomy (SN) and intra-thecal baclofen [8].

When SMO is focal or diffuse but only focally disabling, a focal treatment is recommended. Physical therapy with stretching is the first-line treatment allowing for only transient spasticity reduction, but the ability of stretching to prevent contracture is questioned [9,10]. A self-rehabilitation program, casting and splinting, electrical stimulation (transcutaneous electrical nerve stimulation [TENS] and neuromuscular electrical stimulation [NMES]) and extra-corporeal shock-wave therapy are also proposed [11].

This paper discusses the effectiveness, indications, limitations and side effects of BoNT-A and SN for treating focal spastic muscle overactivity to help clinicians choose the most appropriate treatment.

2. Botulinum toxin type A

BoNT-A is a neurotoxin produced by *Clostridium botulinum* that blocks the release of acetylcholine from the pre-synaptic nerve endings, thereby dose-dependently inducing muscle chemodenervation. Three BoNT-A toxins are marketed in Europe: abobotulinum toxin (Dysport®), incobotulinum toxin (Xeomin®) and onabotulinum toxin (Botox®).

BoNT-A used to treat spasticity has the highest level of evidence. Several randomized, double blind, placebo-controlled studies have demonstrated that BoNT-A injections can reduce muscle tone, increase passive and active range of motion and reduce carer burden [12,13]. In contrast, the effect on gait velocity and hand active grasp activity is more equivocal [14]. Evidence is high for the impairment domain of the International Classification of Functioning, Disability and Health (ICF) but weak or absent for the activity domain. BoNT-A can be injected during outpatient practice in every accessible muscle, under electromyography, electrostimulation and/or ultrasound guidance [15]. BoNT-A has a reversible effect because drug removal and sprouting reinnervation resolve muscle weakness, which requires repeated injections every 3 to 4 months that affect treatment costs. Furthermore, reimbursement disparities between countries frequently limit patient accessibility to BoNT-A treatment. These drawbacks support the need for a long-lasting treatment of spastic overactivity such as SN.

3. Selective neurotomy

First described by Stoffel in 1911, SN is a surgical procedure consisting of a partial section of the motor nerve branches innervating the spastic muscles. At that time, motor and sensory fibers were indistinctly cut, thus inducing neuropathic pain. Because of the adverse effects and the advent of chemical nerve blocks, the technique was nearly abandoned. In the 1990s, French and Belgian neurosurgical teams reintroduced the technique by using intraoperative electrical stimulation and an operating microscope [16,17]. SN is performed under general anaesthesia without muscle relaxant drugs to allow for intraoperative electrical stimulation. The nerve is dissected and the motor nerve branches are identified by intraoperative electrical stimulation. The selected motor nerve branches innervating the spastic muscle over a 5-mm length are partially sectioned under the microscope. The extent of the nerve section (from 50% to 100%) is determined according to the degree of spasticity and type of muscle [17]. Patients are allowed to move or walk the day after the surgery and no immobilization or casting is necessary. Frequently, SN is associated with tendon surgery (lengthening, transfer), which requires 6-week cast immobilization.

This refined technique allows for selectively cutting the motor nerve fascicles, including α and γ motor fibers and Ia, Ib and II proprioceptive fibers. Proximally, at a distance depending on the nerve, proprioceptive fibers leave the motor fascicles and join the sensitive fascicles containing the type III and IV fibers. The neurotomy should be performed distally relative to these crossing points. For each nerve, based on anatomical data, the surgeon knows where the neurotomy needs to be done to avoid sensory deficits. Because Ia, Ib and II fibers are not dissociable in the motor nerve fascicle, SN always involves sectioning these 3 types of fibers.

Thus, SN implies a sectioning of the α motor fibers mediating voluntary muscle contraction and the Ia fibers mediating the myotatic reflex [18,19]. The α motor fiber sectioning leads to a transient muscle weakness. The amount of α motor fibers lost after SN as measured by the motor unit number estimation technique is proportional to the extent of the nerve sectioning [19]. However, a sprouting process explains a recovery in triceps surae muscle strength associated with a return of the Mmax amplitude to baseline values after 8 to 12 months. Furthermore, this transient muscle weakness has no significant harmful effect for the patient. SN also implies a sectioning of the Ia fibers mediating the monosynaptic stretch reflex arc, which leads to spasticity suppression and clonus disappearance associated with an Hmax/Mmax ratio reduction [18,19]. Such a reduction is permanent because Ia fibers sprouting at the level of the muscle spindle is ineffective. These electrophysiological studies suggest that SN is effective for reflex spastic overactivity (spasticity and spastic component of spastic dystonia) but not for non-reflex overactivity (the dystonic part of the spastic dystonia, associated reactions and dystonia). Indeed, the non-reflex overactivity is immediately reduced after SN due to muscle weakness but recurs after 1 year via the collateral sprouting of α motor fibers. Finally, the effect of SN on spastic muscular co-contractions (related more to function) is unknown.

Tibial nerve SN for spastic foot management is the most frequently reported indication. In a systematic review based on case series, Bollens et al. found that tibial SN could be a permanent and efficient treatment to reduce spasticity in the spastic equinovarus foot [20]. Such a reduction is associated with a patient-reported improvement in standing and walking abilities during daily living activities [21]. Furthermore, diagnostic nerve block with anaesthetics helps predict the effect of tibial SN on spasticity reduction and gait improvement [22]. Only a few uncontrolled studies with a limited number of patients assessed SN of the musculo-cutaneous nerve (for elbow flexum), median and ulnar nerves (for spastic hand), obturator nerve (for adducted hip) and rectus femoris nerve (for stiff knee gait) [23–25].

4. Botulinum toxin type A or selective neurotomy?

In clinical practice, when SMO requires focal treatment, we frequently have to choose between BoNT-A or SN. To decide the optimal treatment, we have to know the respective effects, indications, advantages, side effects and limitations of each technique. Until now, only 2 studies compared the effect of BoNT-A injections and tibial SN for the spastic equinovarus foot after stroke. In an open-label study of 34 hemiplegic patients, SN performed on the motor nerve branches of the tibial nerve was more effective than BoNT-A injections (300 U onabotulinum toxin type A) on triceps surae spasticity, passive and active ankle range of motion, foot position and gait velocity [26]. Bollens et al. performed the only single-blind randomized controlled trial (RCT) demonstrating that SN performed on the soleus, tibialis posterior and flexor hallucis longus motor nerve branches induced a higher reduction in ankle stiffness and a comparable improvement in ankle kinematics during gait than BoNT-A injections in the same muscles. However, the 2 treatment groups did not differ in activity, participation and quality of life [27]. These studies confirmed our clinical experience that SN induces a greater reduction in spasticity than BoNT-A.

Table 1 shows the advantages and disadvantages of BoNT-A and SN. BoNT-A is the treatment for focal spasticity with the highest level of evidence (level A). BoNT-A effects are confirmed by several placebo RCTs, whereas the effect of SN is supported by only 1 RCT (level C). BoNT-A is available worldwide, with a network of trained physicians making it accessible to patients in outpatient practice. In contrast, SN requires surgical skill and an interdisciplinary approach, which explains why it is only performed by a few teams in a few countries [8]. A preoperative diagnostic nerve block (DNB) with anaesthetics is also needed as an assessment tool. Such DNB helps in differentiating the respective responsibility of SMO, contracture and weakness in a spastic deformity and identify an excessive activity of the antagonistic muscles that may cause an inverted pattern after SMO treatment. Of note, the DNB predicts the functional improvement obtained with SN for the spastic equinovarus foot [22]. DNB can also be useful before BoNT-A because it helps identify the muscles to inject. We also recommend the use of BoNT-A as a long-lasting block when the DNB is effective, which confirms that SMO is the main problem and supports an increase in BoNT-A dose (or performing SN) if the first BoNT-A injection is ineffective. The reduction in spasticity and gait improvement is usually greater after DNB and SN than after BoNT-A alone. The BoNT-A effect is sometimes insufficient. It is confirmed by the fact that many patients and physicians estimate that they can benefit from injection intervals shorter than 3 months and higher doses than those permitted by the country directives [28]. In contrast, patients rarely report that SN effect is insufficient, which confirms our clinical experience that SN induces a greater reduction in spasticity than does BoNT-A. Even if higher BoNT-A doses can be used without additional side effects, some patients with multiple spastic patterns require a BoNT-A dose greater than the recommended maximal dose per session, so BoNT-A and SN are complementary (i.e., SN to treat a spastic foot and BoNT-A for other patterns) [29]. Furthermore, repeated BoNT-A injections do not result in an ineffective response to nerve stimulation of previously injected muscles during later SN.

Table 1
Advantages and disadvantages of botulinum toxin type A (BoNT-A) and selective neurotomy (SN).

	BoNT-A	SN
Level of evidence	A	C
Accessibility for patients	++	±
SMO reduction	+	++
Muscle selection	++	±
Reversibility	++	–
Long lasting	–	++
Side effects — risk	±	+
Cost	+	++

SMO: spastic muscle overactivity; – absent; ± slight; + good; ++: excellent.

BoNT-A can be injected in nearly every muscle with SMO even the less accessible ones (i.e., flexor digitorum profundis), which allows for treating every spastic pattern both in the upper and lower limbs. Even if SN can be theorically performed on every accessible motor nerve, it is mainly performed on the tibial nerve (for equinovarus foot), musculo-cutaneous nerve (for elbow flexum), obturator nerve (for adducted hip) and femoral nerve (for stiff knee gait).

BoNT-A is a reversible treatment that has to be renewed every 3 to 4 months, with consequent practical (medical consultation) and financial issues, which supports the need for a permanent treatment such as SN. Reversibility is an advantage when treating a functional spastic pattern (i.e., hand with prehension function) because SMO reduction may be associated with weakness in the same muscles. If BoNT-A worsens patient function, the patient will recover when the BoNT-A effect disappears. However, the SN effect is permanent, without any possibility to recover the pre-operative status.

The spasticity reduction observed after SN is immediate and stable over time, but patients receiving BoNT-A have to wait several days or weeks before experiencing its effects and 1 to 2 months for the maximum effect. Such spasticity variations may interfere with the rehabilitation program and the goal to be achieved.

Publications support the long-lasting effect of SN on spasticity [17,30], but muscle denervation related to α motor fiber sectioning may induce muscle contracture. This situation could explain the deformity recurrence described by some authors in case reports [31]. However, recent data also support that BoNT-A induces muscle structural and mechanical changes related to neurogenic atrophy [32]. This observation highlights the need for a prolonged muscle stretching program after SN and BoNT-A [8].

BoNT-A side effects are usually non-serious and range from 2% to 9%, including pain at the injection site, dysphagia, muscle weakness and fatigue [12,29]. The incidence of adverse events seems to be higher with oral medications (tizanidine) than with BoNT-A [33]. SN side effects include wound infection, delayed wound healing and sensory disturbances when sensory fibers are sectioned or excessively manipulated into the nerve trunk. Rousseaux et al. reported pain or dysesthesia at the plantar sole in 13 of 34 patients, in the first 2–6 weeks after SN when tibialis posterior and flexor digitorum longus nerve were sectioned [26]. In a series of 30 patients, Deltombe et al. did not report additional sensory deficits, but nerve branches to the flexor digitorum longus muscle were spared [17]. Sensory deficits could be related to the surgical technique. Ideally, neurotomy should be performed as close as possible to the muscle and at least at the level of the motor branches after they have left the nerve trunk. In some cases, neurotomy can be done inside the nerve trunk without causing sensory deficits or neuropathic pain (e.g., soleus inferior nerve or flexor hallucis longus nerve into the tibial nerve trunk). This technique allows for small skin incisions. In other cases, sectioning of the motor fascicles in the nerve trunk will result in sensory deficits and neurotomy must be performed close to the muscle (e.g., median and ulnar nerves, flexor digitorum longus nerve). This hyperselective neurotomy requires larger skin incisions and is therefore more aggressive.

Antithrombotic medications such as anticoagulants and antiplatelets are frequently administered after ischemic stroke, atrial fibrillation and deep vein thrombosis. Even if theoretically

implying increased risk of bleeding and compartment syndrome, antithrombotics do not seem to be an absolute contraindication to intra-muscular BoNT-A injections. A recent study found no report of compartment syndrome or bleeding episodes in series of patients injected while under antithrombotics [34]. In our practice, we inject patients who are under antithrombotics after informed consent and under ultrasound guidance to avoid vessel damage. Less experienced injectors should take the risk of bleeding into account in patients under anticoagulant medications especially if they do not use ultrasound guidance. In contrast, anticoagulant therapy should be stopped (frequently by a transient shift to low-molecular-weight heparin) before performing a diagnostic nerve block and SN. Similarly, SN requires general anaesthesia without curarisation. A local anesthesia is not possible because it will limit the motor nerve identification with electrical stimulation during the procedure. Therefore, in patients with high thrombotic risk who cannot stop their anticoagulant therapy, even transiently, and/or who cannot benefit from general anaesthesia, BoNT-A is preferred especially if performed by an experienced injector under ultrasound guidance. Finally, no study has compared the relative cost of BoNT-A and SN. The cost probably differs depending on the country. In Belgium, the cost for SN is equivalent to 6 consecutive BoNT-A injection sessions.

Table 2 summarizes the effect of BoNT-A and SN on the different types of spastic muscle overactivity. BoNT-A reduces spasticity, as measured by the modified Ashworth score and the Tardieu scale [12], clonus [35] and dystonia. BoNT-A is also effective to treat patterns characterized by spastic dystonia (i.e., hand) [12,13], but the effect on associated reactions (i.e., elbow flexum) is unclear. Vinti et al. showed that a BoNT injection reduces the spastic co-contraction of the non-injected antagonist muscles probably by increasing reciprocal inhibition [36]. Reducing spastic co-contraction is a concern in our practice because co-contraction is probably linked more to function than spasticity. SN is effective to restrain spasticity and clonus. Most studies have shown clonus disappearance after SN [20]. Indeed, SN destroys the Ia fibers included in the motor nerve branch, which leads to permanent clonus disappearance because Ia fibers are not able to sprout at the level of the muscle spindle. This phenomenon is supported by a permanent reduction in Hmax/Mmax ratio after SN [18,19]. In contrast, SN has never been studied specifically in spastic dystonia, associated reactions, spastic co-contraction and dystonia. We question the long-lasting effect of SN on the non-reflex component of spastic overactivity, especially for spastic dystonia with muscle activation at rest and dystonia. In such patterns, SN acts for several months because the α motor fibes sectioning causes a muscle weakness that will recover with sprouting after 8 to 12 months. The dystonic part of the spastic dystonia pattern is suspected to reappear in relation to the α motor sprouting, which explains the deformity recurrence. This phenomenon probably explains why SN provides more long-lasting results with spasticity (i.e., spastic foot) than spastic dystonia (i.e., spastic hand) and spastic motor overflow (i.e., elbow flexum). Furthermore, in our opinion, dystonia is a contraindication to SN because of the rapid recurrence of the deformities but also an unpredictable poor outcome with a worse deforming pattern than initially. A complete clinical assessment is required to determine the type of spastic muscle overactivity before proposing SN. A minimum follow-up of 2 years is recommended after SN.

Table 3 summarizes the different spastic patterns treatments reported with BoNT-A and SN. SN has been mainly reported at the tibial nerve as a treatment for the spastic equinovarus foot [20,27]. As already noted, SN at the flexor digitorum longus nerve with claw toes involves risk of neuropathic pain, so alternative treatments such as BoNT-A injections or tendon lengthening must be proposed. Some publications reported neurotomy at the level of the rectus femoris nerve [23] in case of stiff knee gait, the median and ulnar nerves [24] in case of the spastic hand for passive and active function and the musculo-cutaneous nerve [25] in case of elbow-flexor spastic dystonia and associated reactions. However, these studies included only a small number of patients, generally with a short follow-up. In contrast, BoNT-A has been shown to be effective for the equinus foot and upper limb [12,13,37].

BoNT-A has been studied in stroke, traumatic brain injury and cerebral palsy, with level A evidence, and in multiple sclerosis with level B evidence [38]. In contrast, SN has been mainly reported as a treatment of spastic overactivity after stroke and traumatic brain injury. However, in our opinion, the type of SMO and spastic patterns is more important than the neurological disorder. Only anecdotic reports have mentioned other aetiologies such as multiple sclerosis and cerebral palsy (CP). Whether SN can be proposed to children with CP is debated. The particularity of the CP in the growing phase is that the spastic muscle (in itself at risk of contracture) must grow with bones to avoid deformity of joints and bones. The risk of SN, by inducing a massive muscle denervation, is in blocking and/or limiting the muscle growth and increasing the deformity in the long term. Berard postulated that muscle fiber reinnervation leading to extensive motor units explains the deformity recurrence after SN in hemiplegic children requiring orthopaedic surgery [39]. Because the long-lasting risk of contracture after SN in children with CP is unknown, we do not recommend SN and prefer BoNT-A injections.

In conclusion, BoNT-A is the recommended treatment for focal SMO, with the highest level of evidence. It can be injected with high safety in many muscles and has been found effective in nearly all types of SMO, many spastic patterns and almost all central nervous system diseases. However, the BoNT-A effect is transient, fluctuating and sometimes insufficient or limited by the recommended maximal dose per session, requiring repeated injections every 3 to 4 months. These drawbacks support the need for a permanent and more effective treatment such as SN. SN is mainly proposed at the level of the tibial motor nerve branches (except branches to the flexor digitorum longus) for spasticity and clonus responsible for an equinovarus deformity after stroke and traumatic brain injury. It can also be performed on the musculo-cutaneous, femoral and obturator nerves. Further research is needed to assess the efficacy (RCT) and long-lasting effect of SN in different types of SMO, spastic patterns and aetiologies. Randomized studies comparing the effect of BoNT-A and SN are also needed.

Table 2
Effectiveness of BoNT-A and SN for types of spastic muscle overactivity.

	BoNT-A	SN
Spasticity	++	++
Clonus	+	++
Spastic dystonia	+	?
Associated reactions	±	?
Spastic co-contraction	+	?
Dystonia	++	–

?: unknown; – : absent; ± : slight; + : good; ++: excellent.

Table 3
Effectiveness of BoNT-A and SN for different spastic patterns.

	BoNT-A	SN
Stiff knee gait	+	+
Equinovarus foot	++	++
Claw toes	++	?
Elbow flexum	+	+
Hand	++	+

?: unknown; – : absent; ± : slight; + : good; ++: excellent.

Disclosure of interest

T. Deltombe served as a consultant for Allergan, Ipsen and Merz and received research grant support from Ipsen. T. Lejeune served as a consultant for Merz and received research grant support from Ipsen. T. Gustin served as a consultant for Ipsen.

References

[1] Wissel J, Schelosky L, Scott J, Christe W, Faiss J, Mueller J. Early development of spasticity following stroke: a prospective, observational trial. J Neurol 2010;257:1067–72.
[2] Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. Neurology 2013;80:S13–9.
[3] Opheim A, Danielsson A, Alt Murphy M, Persson H, Sunnerhagen K. Early prediction of long-term upper limb spasticity after stroke: part of the SALGOT study. Neurology 2015;85:873–80.
[4] Gracies JM. Coefficients of impairment in deforming spastic paresis. Ann Phys Rehabil Med 2015;58:173–8.
[5] Yelnik A, Simon O, Paratte B, Gracies JM. How to clinically assess and treat muscle overactivity in spastic paresis. J Rehabil Med 2010;42:801–7.
[6] Winstein C, Stein J, Arena R, Bates B, Cherney L, Cramer S, et al. Guidelines for adult stroke rehabilitation and recovery. Stroke 2016;47:98–169.
[7] Wissel J, Ward A, Erztgaard P, Bensmail D, Hecht M, Lejeune T, et al. European consensus table on the use of botulinum toxine type A in adult spasticity. J Rehab Med 2009;41:13–25.
[8] Deltombe T, Wautier D, De Cloedt P, Fostier M, Gustin T. Assessment and treatment of spastic equinovarus foot after stroke: guidance from the Mont-Godinne interdisciplinary team. J Rehabil Med 2017;49:461–8.
[9] Bovend'Eerdt T, Newman M, Barker K, Dawes H, Minelli C, Wade D. The effects of stretching in spasticity: a systematic review. Arch Phys Med Rehabil 2008;89:1395–406.
[10] Katalinic O, Harvey L, Herbert R. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: a systematic review. Phys Ther 2011;91:11–24.
[11] Khan F, Amatya B, Bensmail D, Yelnik A. Non-pharmacological interventions for spasticity in adults: an overview of systematic reviews. Ann Phys Rehabil Med 2017 [In press, available online 16 october 2017].
[12] Gracies JM, Brashear A, Jech R, McAllister P, Banach M, Valkovic. et al. Safety and efficacy of abobotulinumtoxin A for hemiparesis in adults with upper limb spasticity after stroke and traumatic brain injury: ad double–blind randomized controlled trial. Lancet Neurol 2015;14:992–1001.
[13] Foley N, Pereira S, Salter K, Fernandez M, Speechley M, Sequeira K, et al. Treatment with botulinum toxin improves upper-extremity function post stroke: a systematic review and meta-analysis. Arch Phys med rehabil 2013;94:977–89.
[14] Foley N, Murie-Fernandez M, Speechley M, Salter K, Sequeira K, Teasell R. Does the treatment of spastic equinovarus deformity following stroke with botulinum toxin increase gait velocity? A systematic review and meta-analysis. Eur J Neurol 2010;17:1419–27.
[15] Grigoriu A, Dinomais M, Remy-Neris O, Brochard S. Impact of injection-guiding techniques on the effectiveness of botulinum toxin for the treatment of focal spasticity and dystonia: a systematic review. Arch Phys Med Rehabil 2015;96:2067–78.
[16] Decq P, Filipetti P, Cubillos A, Slavov V, Lefaucheur JP, Nguyen JP. Soleus neurotomy for treatment of the spastic equinus foot. Neurosurgery 2000;47:1154–61.
[17] Deltombe T, Gustin T. Selective tibial neurotomy in the treatment of spastic equinovarus foot in hemiplegic patients: a 2-year longitudinal follow-up of 30 cases. Arch Phys Med Rehab 2010;91:1025–30.
[18] Feve A, Decq P, Filipetti P, Verroust J, Harf A, N'Guyen JP, et al. Physiological effects of selective neurotomy on lower limb spasticity. J Neurol Neurosurg Psychiatry 1997;63:575–8.
[19] Deltombe T, Jamart J, Hanson P, Gustin T, Soleus H. reflex and motor unit number estimation after tibial nerve block and neurotomy in patients with spastic equinus foot. Neurophysiol Clin 2008;38:227–33.
[20] Bollens B, Deltombe T, Detrembleur C, Gustin T, Stoquart G, Lejeune T. Effects of selective tibial nerve neurotomy as a treatment for adults presenting with spastic equinovarus foot: a systematic review. J Rehabil Med 2011;43:277–82.
[21] Le Bocq C, Rousseaux M, Buisset N, Daveluy W, Blond S, Allart E. Effects of tibial nerve neurotomy on posture and gait in stroke patients: a focus on patient-perceived benefits in daily life. J Neurol Sci 2016;15:158–63.
[22] Deltombe T, Bleyenheuft C, Gustin T. Predictive value of Diagnostic Tibial Motor Nerve Branches Block with Anaesthetics before Selective Tibial Neurotomy in Hemiplegic Patients with Spastic Equinovarus Foot. Ann Phys Rehabil Med 2015;58:54–9.
[23] Gross R, Robertson J, Leboeuf F, Hamel O, Brochard S, Perrouin-Verbe B. Neurotomy of the rectus femoris nerve. Short-term effectiveness for spastic stiff knee gait: clinical assessment and quantitative gait analysis. Gait Posture 2017;52:251–7.
[24] Maarawi J, Mertens P, Luaute J, Vial C, Chardonnet N, Cosson M, et al. Long-term functional results of selective neurotomy for the treatment of spastic upper limb: prospective study in 31 patients. J Neurosurg 2006;104:215–25.
[25] Shin D, Jung Y, Hong J, Kim M, Kim S. Selective musculocutaneous neurotomy for spastic elbow. J Korean Neurosurg Soc 2010;48:236–9.
[26] Rousseaux M, Buisset N, Daveluy W, Kozlowsli O, Blond S. Comparison of botulinum toxin injection and neurotomy in patients with distal lower limb spasticity. Eur J Neurol 2008;15:506–11.
[27] Bollens B, Gustin T, Stoquart G, Detrembleur C, Lejeune T, Deltombe T. A randomized controlled trial of selective neurotomy versus botulinum toxin for spastic equinovarus foot after stroke. Neurorehabil & Neural Repair 2013;27:695–703.
[28] Bensmail D, Hanschmann A, Wissel J. Satisfaction with botulinum toxin treatment in post-stroke spasticity: results from two cross-sectional surveys (patients and physicians). J Med Econ 2014;17:618–25.
[29] Wissel J, Bensmail D, Ferreira J, Molteni F, Satkunam L, Moraleda S, et al. Safety and efficacy of incobotulinumtoxinA doses up to 800U in limb spasticity: the TOWER study. Neurology 2017;88:1321–8.
[30] Rousseaux M, Buisset N, Daveluy W, kozlowski O, Blond S. Long term effect of tibial nerve neurotomy in stroke patients with lower limb spasticity. J Neurol Sci 2009;278:71–6.
[31] Collado H, Bensoussan L, Viton JM, Milhe De Bovis V, Delarque A. Does fascicular neurotomy have a long lasting effects? J Rehabil Med 2006;38:212–7.
[32] Mathevon L, Michel F, Decavel P, Fernandez B, Paratte B, Calmels P. Muscle structure and stiffness assessment after botulinum toxin type A injection. A systematic review. Ann Phys Rehabil Med 2015;58:343–50.
[33] Simpson D, Gracies JM, Yablon S, Barbano R, Brashear A. Botulinum neurotoxin versus tizanidine in upper limb spasticity: a placebo-controlled study. J Neurol Neurosurg Psychiatry 2009;80:280–5.
[34] Phadke C, Thanikachalam V, Ismail F, Boulias C. Patterns of botulinum toxin treatment for spasticity and bleeding complications in patients with thrombotic risk. Toxicon 2017;138:188–90.
[35] Thanikachalam V, Phadke C, Ismail F, Boulias C. Effect of botulinum toxin on clonus: a systematic review. Arch Phys Med Rehabil 2017;98:381–90.
[36] Vinti M, Costantino F, Bayle N, Simpson D, Weisz D, Gracies JM. Spastic cocontraction in hemiparesis: effects of botulinum toxin. Muscle Nerve 2012;46:926–31.
[37] Kaji R, Osako Y, Suyama K, Maeda T, Uechi Y, Iwaski. et al. Botulinum toxin type A in post-stroke lower limb spasticity: a multicenter, double blind, placebo-controlled trial. J Neurol 2010;257:1330–7.
[38] Safarpour Y, Mousavi T, Jabbari B. Botulinum toxin treatment in multiple sclerosis – a review. Curr Treat Options Neurol 2017;17:19–33.
[39] Berard C, Sindou M, Berard J, Carrier H. Selective neurotomy of the tibial nerve in the spastic hemiplegic child: an explanation of the reccurence. J Pediatr Orthop 1998;7:66–70.