

Module 3: Surgical Management of Spasticity

Raphaël Gross¹, Monica Verduzco-Gutierrez², Nathalie Draulans³, Máximo Zimerman⁴, Gerard E. Francisco^{5,6}, Thierry Deltombe⁷

¹Nantes Université, CHU Nantes, Department of Physical and Rehabilitation Medicine, Movement - Interactions - Performance, MIP, UR 4334, F-44000 Nantes, France, ²Department of Physical Medicine and Rehabilitation, Joe R. and Teresa Lozano Long School of Medicine at UT Health San Antonio, San Antonio, Texas, USA, ³Department of Rehabilitation, Libra Rehabilitation and Audiology, Eindhoven, The Netherlands, ⁴Institute of Cognitive Neuroscience, ALCLA, Favaloro University Buenos Aires, Argentina, ⁵Department of Physical Medicine and Rehabilitation, McGovern Medical School, University of Texas Health Science Center, ⁶Department of Physical Medicine and Rehabilitation, UTHealth Houston McGovern Medical School and TIRR Memorial Hermann Hospital, Houston, Texas, USA, ⁷Department of Physical Medicine and Rehabilitation, Université catholique de Louvain, Centre Hospitalier Universitaire de Namur, Godinne Site, Avenue Docteur G Therasse, Yvoir, Belgium

Abstract

This module outlines the history of the development of surgical interventions for treating spasticity and discusses when surgical intervention is most appropriate for managing spasticity. A range of surgical techniques are considered; intrathecal baclofen, neurotomy, and muscle or tendon lengthening and transfer procedures. The implications and limitations of the surgical techniques are considered. The need for a multidisciplinary team to deliver optimal surgical treatment is also considered.

Keywords: Spasticity- surgical management - indications - intrathecal treatment -multidisciplinary team

LEARNING OBJECTIVES

On completion of this module, the learner will be able to:

1. Evaluate the need for surgical management options as part of the treatment paradigm for patients with spasticity
2. List the indications for surgical management options including intrathecal baclofen (ITB), neurotomy, and muscle/tendon lengthening and transfer procedures
3. Describe the implications and limitations of the surgical techniques
4. Organize a multidisciplinary team to effectively treat patients who need surgical management for spasticity.

BACKGROUND AND HISTORY

Deforming spastic paresis is a condition in which muscle imbalance across joints leads to abnormal positioning and tightness. Muscle over-activity (spasticity, spastic dystonia) and muscle contracture of the agonists coexist with paresis of the antagonists, as the paretic component and the spastic component of the upper motor neuron syndrome are not equally distributed to the muscles (see Module 1 in this Supplement). This drives abnormal postures and deformation of the body segments. Well-known examples of such troublesome situations are the spastic equinus (or equinovarus) foot at the lower limb and the clenched fist at the upper limb. Unlike spasticity, which involves muscles, contractures can involve muscles as well as other soft tissues. The term “contracture”

refers to shortening and stiffening of muscles, tendons, and other soft tissues, which lead to joint deformities. In extreme cases, the joint capsule can be involved (arthrogenic contracture). Affected limbs typically demonstrate abnormal posture that affects both passive and active functions.

The initial treatment of spasticity includes conservative management, physical therapy and splinting, oral medications (e.g., baclofen and dantrolene), and injectable neurolytic medications (e.g., botulinum toxin [BoNT] and phenol). These techniques have been discussed in detail in Paper 2 in this Supplement. Patient education and goal setting are covered in Curriculum 1 in this Supplement.

Physicians and other members of teams who care for patients with disabling spasticity are well aware of the fact that medical treatment, either general (using oral medications) or focal (using chemodenervating agents, such as BoNT or phenol), might be insufficient to obtain satisfying results in terms of passive or active function and to prevent muscle contractures and joint deformities. In addition, adverse events of general treatments, gamma aminobutyric acid-ergic (GABAergic) agents and calcium-blocking drugs, and risks associated with repeated injections of BoNT or phenol drive medico-surgical teams to consider treatments that provide a permanent reduction of spasticity. In patients with severe, disabling, spasticity, surgical treatment can be considered.

Surgical procedures for the treatment of spasticity have been developed for almost 150 years. The first-known report was made by Lorenz (1854–1946), an Austrian surgeon who, in 1887, described the section of the obturator nerve to treat spastic hip adduction.^[1] In 1912, Stoffel reported on tibial

Access this article online

Quick Response Code:



Website:
www.jisprpm.org

DOI:
10.4103/2349-7904.347809

How to cite this article: Gross R, Verduzco-Gutierrez M, Draulans N, Zimerman M, Francisco GE, Deltombe T. Module 3: Surgical management of spasticity. *J Int Soc Phys Rehabil Med* 2022;5:S38-49.



Figure 1: Technique for implantation of the intrathecal catheter and pump. ©Medtronic

nerve neurotomy to treat spastic equinus and median nerve neurotomy for pronation of the forearm and clenched fist.^[2] The neurotomy technique spread in Europe and North America, and the first descriptions of surgeries combining neurotomies with muscle lengthening procedures were made shortly after the World War II in the US.^[3] Since then, technical improvements were developed by French neurosurgeons such as Gros and Sindou and Mertens to enable selective neurotomies at the nerve fascicle level to be performed. Gros introduced peroperative nerve stimulation to identify the different fascicles among a nerve.^[4] Sindou and Mertens used microscopes to perform precise dissections of the nerve fascicles, together with bipolar nerve stimulation.^[5] At the same time, in the 1970s/80s, surgical neurotomies in the upper limb were developed, for the treatment of spastic elbow flexion^[6] or wrist/finger flexion.^[7] There has been a considerable number of descriptions since, about how selective neurotomies can be performed to treat spastic muscle over-activity in muscle groups in the upper and lower limbs, and about their association with orthopedic procedures targeting muscle, tendons, joints, and bones.^[8]

Conceptually, selective peripheral neurotomies aim at reducing muscle over-activity of one or several muscles. A first inherent limit is therefore that it does not allow lengthening of a muscle which is already shortened (contracture). Other techniques, aiming at muscle and/or tendon lengthening, have therefore been described. While it is likely that sections of the tendons were already performed in antiquity, the first complete descriptions were made in the early 19th century, in patients with club-foot.^[9,10] Many techniques have been described since, to move forward from the simple tendon resection to adjustable tendon lengthening and intramuscular lengthening procedures.^[8] The second inherent limit of peripheral neurotomy is its inadequacy when overall spasticity is to be treated, even if several neurotomies can be performed during a single-event surgery. Hence, parallel to peripheral neurotomy, surgical procedures targeting the spinal roots in case of more diffuse spasticity have emerged. Otfried Foerster, a German neurologist and neurosurgeon, was the first to introduce dorsal root sections (rhizotomy) in human patients, to treat lower limb spasticity. Based on experiments by the famous neurophysiologist Sherrington who, in 1898, showed that

muscle over-activity could be reduced by sectioning the dorsal roots of the spinal cord in an animal model of decerebration. Foerster performed in 1908 the first dorsal root resection at the lumbar and sacral levels (from root L1 to root S2) in men.^[11] The follow-up of these patients showed that a systematic resection of the dorsal roots L1 to S2 could result in sensory loss as well as functional loss in some patients who were able to walk despite (or thanks to) their spasticity. Foerster also reported that there was variability in the innervation level of the lower limb muscles by the lumbar and sacral roots.^[12] This lead followers to improve the selectivity of the resection by performing a mapping of the innervation, using peroperative stimulation of the dorsal roots and observation of the stimulated activation of the muscles.^[13] Further developments of the selective dorsal rhizotomy consisted in the application of this technique to the cervical roots to treat the spasticity in the upper limb^[14] and in the use of chemical intrathecal radicotomies^[15] or percutaneous thermocoagulation of the dorsal roots.^[16] In addition, the French team lead by Marc Sindou developed a technique known as DREZotomy, where the spinal afferents from the dorsal root are cut not at the dorsal root level but in the dorsal root entry zone (DREZ) of the spinal cord. The topographical segregation between the reflex and nociceptive fibers (lateral part of the DREZ) and the big sensory fibers that must be spared (medial part) is the anatomic rationale for this surgery, initially developed for chronic refractory pain, and rapidly adapted to focal spasticity of the limbs.

Eventually, the treatment of spasticity by intrathecal infusions of baclofen emerged at the end of the 20th century. Baclofen is known as an agonist of the GABA-B receptors. Its use as an oral drug is hindered by a low risk–benefit ratio, due to cognitive adverse effects, as well as to its low penetration in the cerebrospinal fluid when administered orally. Penn and Kroin were the first to inject baclofen directly in the subarachnoid space by lumbar puncture, to depress spinal reflexes at the lumbar spine (where the density of GABA-B receptors is particularly high).^[17] The industrial development of implantable, programmable pumps and catheters allowed ITB therapy to become an adjustable and reversible treatment of spasticity in the 1980–1990s (see specific section below).^[18]

SURGICAL MANAGEMENT

Surgery cannot stand alone in the treatment of spasticity. There needs to be medico-surgical collaborative approach and team of allied specialists. Ideally, rehabilitation physicians will lead the team (including neurosurgeons, orthopedic surgeons, and plastic surgeons, depending on available competencies) and be supported by physiotherapists, occupational therapists, and nursing teams.

The overall principle of surgical treatment is to improve muscle balance by lengthening and/or reducing the activity of muscles that are agonists to the deformity and, if possible, improve the antagonists' mechanical action. This implies the need for surgery for contractures (lengthening procedures) and

for motor over-activity (peripheral neurotomy and selective dorsal rhizotomy being the most common). The release of the short and/or spastic agonists allows potential antagonist muscle activity to be expressed, and specific procedures (tendon transfers) aim at improving the biomechanical action of the antagonists. Joint mobility and stability must be considered and can be affected as side effects of a surgical release. Therefore, stabilization procedures targeting the joints and bones are sometime needed.^[8]

Neuro-orthopedic surgery will only be as successful as the preoperative assessment is accurate. The two first key points are that (i) there must be differentiation between deformities that are useful for function and those that are detrimental and that (ii) it is of utmost importance to understand whether muscle hypoextensibility is due to hypertonicity or a fixed contracture. Motor nerve blocks are key to the unraveling of muscle over-activity and contracture in a given muscle or muscle group. Therefore, before surgery, the functional benefit of a spasticity reduction must be assessed by performing a motor nerve block with anesthetics to mimic. This technique has been shown to predict improvement provided by the neurotomy in cases of equinovarus foot.^[19]

The third key point in the surgical management of muscle over-activity is the careful attention to the extent of troublesome spasticity in the patient. If it is focal, then localized treatments, such as peripheral neurotomy are indicated. By contrast, diffuse spasticity affecting the entire limb, or even several limbs, requires surgery able to reduce spastic muscle over-activity at multiple levels by one single procedure, such as selective dorsal rhizotomy or ITB therapy through an implantable pump (see below for details about the different techniques).

MECHANISMS, INDICATIONS, BENEFITS, AND POTENTIAL RISKS OF INTRATHECAL BACLOFEN THERAPY

Oral baclofen, given for generalized spasticity, has a poor blood–brain barrier penetration. In ITB therapy, baclofen is given directly in the cerebrospinal fluid surrounding the spinal cord, by using an intrathecal catheter and an implantable device consisting of a reservoir and a pump system. Intrathecal administration results in more effect on spasticity with lower serum levels of baclofen and therefore less systemic side effects.

Indication

ITB should be considered in patients with diffuse and significantly refractory spasticity or patients who are experiencing unacceptable side effects or an inadequate response to oral medication or focal treatments. It is appropriate for both adults and children, those with progressive and nonprogressive disease and ambulatory and nonambulatory patients.^[20–23] However, ITB therapy must always be undertaken with realistic goals of treatment in mind. Suggested goals for ITB therapy are given in Table 1.^[24]

There are no contraindications for patients with ventricular shunts for hydrocephalus, those experiencing seizures, or those on antiplatelet therapy (unless lumbar puncture is required).

Table 1: Goal setting for intrathecal baclofen therapy

Examples of goals potentially achieved with intrathecal baclofen	
Active goals	Passive goals
Improved mobility: speed, safety, and quality in the home and community	Improved positioning
Increased ability and independence for ADLs: dressing, eating, hygiene	Improved wheelchair tolerance
Decreased time for ADLs	Prevention of complications such as contractures
	Decreased caregiver burden and time
	Improved quality of sleep
Reduced stretching time during home exercise	Deduced spasticity-related pain
	Discontinuation of oral antispasmodics

Taken from^[24] ADLs: Activities of daily living

There is generally no relevant effect of ITB therapy on the unaffected limb in stroke.^[25,26] A significant effect of ITB versus conventional medical management (oral medication and physiotherapy) has been demonstrated for both upper and lower limbs in stroke.^[22]

Working with ITB requires a dedicated team. Before implantation of the ITB pump and catheter, patients and carers should be well informed about expectations and risks. A trial with bolus or continuous administration of baclofen intrathecally through an external catheter can help in the decision-making process and the realistic goal setting.^[27,28] Regular follow-up for titrating the dosage and refill is necessary (usually every 6 months for Synchronomed™ pumps and 60 days for Prometra® pumps). If the pump is not refilled, there is a risk of acute withdrawal. Malfunctioning of the baclofen pump and/or incorrect dosing are concerns considered in Curriculum 4 in this Supplement.

Technical procedure

There are basically two types of ITB pumps; those with adjustable flow rate (e.g., Synchronomed II™ pump, Medtronic Inc; Prometra II®, Cardiva or Flowonix) and those without adjustable flow rate where changing the concentration in the drug reservoir is necessary to adapt the daily dosage. The technical procedure of implantation is similar. The Prometra® II pump, which can be programmed remotely using a separate hand-held device, was more recently been introduced. Compared to Synchronomed II™, Prometra II® has a larger reservoir, more programming options, and longer battery life (>10 years). Unlike the motor-driven Synchronomed II™, Prometra II broadly disperses medication into the intrathecal space through its pressure-driven, valve-gated delivery system.

A lumbar incision is used for intrathecal catheter insertion. A pocket is created for the pump using a paraumbilical incision. Subcutaneous tunneling from the pump pocket to the lumbar incision is necessary for connecting the pump with the intrathecal catheter [Figure 1].

In patients with spinal cord injuries, the catheter tip is often positioned at the lesion level. In hemiplegic patients, the tip is often positioned midthoracic.

In ambulatory patients, a trial should be considered. The starting daily dose is usually twice the effective trialing bolus. If no trial was done in nonambulatory patients, starting at 100 mcg/day should be considered.

Adverse effects can include postlumbar puncture headache or meningitis.^[27]

Follow-up and maintenance

After pump implantation, patients must be followed up by the multidisciplinary team. It is necessary to evaluate the rehabilitation plan and assess whether any additional therapy is required. Knowledge of different programming modes for programmable pumps is essential, and the team should have experience in troubleshooting. Pumps are fitted with alarm systems in case of malfunction, and it is important that the patient is made aware of the alarms and how to deal with them.

Refills involve emptying the drug reservoir by aspiration and puncturing the membrane of the baclofen reservoir with Port-a-Cath[®] needle. Consequently, the reservoir is refilled with usually 20cc or 40cc of intrathecal baclofen (e.g., 1000 mcg/mL or 2000 mcg/mL), depending on the capacity of the reservoir.

Treatment can be tailored to the individual's requirement when using programmable pumps. For example, it is possible to give a higher dose at night for comfort or in the morning for ease of care.

Dose-dependent side effects must be a consideration, for example, urinary retention, constipation, drooling, sexual dysfunction, respiratory depression, altered mental status (in overdosing), and loss of trunk balance or ability to walk, if these were due to spastic co-contraction.^[22,29]

Every 5–8 years, the pump has to be replaced due to end of life of the battery. If the catheter is still functioning well, it is not replaced.

Costs

ITB therapy is an expensive treatment. However, when the consequent reduction of other spasticity treatments and spasticity-related hospital readmissions, are taken into account it is considered to be cost-effective.^[30-33]

Administration of intrathecal phenol by lumbar puncture is a much cheaper way of reducing spasticity, but it also causes problems of incontinence and may provoke major neuropathic pain.

COMPETENCY ASSESSMENT 1

The answers to these questions can be found at the end of the module before the references.

Are the following statements True or False?

1. Regular refill of the pump reservoir is necessary to avoid potentially dangerous acute withdrawal symptoms that can occur when the drug reservoir is empty
2. ITB therapy cannot be used in hemiplegic patients because it weakens the nonaffected side
3. ITB therapy cannot be combined with BoNT injections because the working mechanism is similar

4. An ITB trial can be useful in helping to identify underlying voluntary strength
5. Sexual dysfunction with disappearance of reflex erection in spinal cord injury patients is a potential side effect of ITB therapy
6. Due to its working mechanism, ITB therapy can resolve fixed contractures in patients with severe spasticity.

MECHANISMS, INDICATIONS, BENEFITS, AND POTENTIAL RISKS OF SELECTIVE NEUROTOMY

Surgical technique

Selective neurotomy (SN) is a surgical procedure consisting in a partial and selective section of a motor nerve innervating a spastic muscle.^[5,34,35] SN is indicated where there is disabling focal spasticity of a muscle innervated by a motor nerve branch that is accessible for surgery and the condition requires permanent treatment.

The SN is ideally performed at the level of a motor nerve branch after it leaves a nerve trunk – for example – soleus, tibialis posterior, and rectus femoris motor nerve branch.

SN can also be performed on a nerve trunk, in which case nerve dissection is necessary to localize and isolate the motor nerve branch – for example – flexor hallucis longus nerve in the tibial nerve and biceps brachialis and brachialis nerves in the musculocutaneous nerve.^[36]

The procedure involves general anesthesia without curarization to preserve responses to perioperative nerve stimulation. The motor nerve branches are identified (ideally after nerve trunk dissection under a microscope) with intraoperative electrical stimulation inducing corresponding muscle contraction. The absence of contraction under nerve stimulation identified a sensory nerve branch that must be carefully spared to avoid sensory deficits and/or neuropathic pain. When the motor nerve branches to treat are identified, a partial section over 5–10 mm length is performed under microscope (e.g., the soleus nerve is 2 mm in diameter). The motor nerve branches to treat and the extent of the section (ranging from 50% to 100%) are determined according to the degree of spasticity and the preoperative assessment.

Postoperative care consists of 3 days hospitalization. Casting is not necessary, and the patient should be mobilized (including walking) as soon as possible allowing for scar pain. Rehabilitation should be promoted according to availability and the patient educated to perform a daily stretching program for 2 years. Clinical assessment (including goal attainment) is recommended after 2 months, 1 year, and 2 years. If necessary, SN can be performed in association with tendon surgery (e.g., tibial neurotomy and Achilles tendon lengthening in case of equinovarus foot).

Neurophysiological effects

The mechanism of action underlying neurotomy proposes that sectioning of the Ia, Ib, and II afferent fibers in the nerves, which mediate this myotatic or stretch response, will lead to

a permanent reduction in spasticity and clonus disappearance correlated to the Hmax/Mmax ratio reduction.^[37] Such reduction is permanent as Ia fibers sprouting at the level of the muscle spindle are ineffective, explaining the permanent effect on the reflex component of the muscle over-activity, particularly spasticity and clonus. SN also implies a section of the α motor fibers mediating voluntary muscle contraction that leads to a transient muscle weakness. The muscle weakness induced by such α motor fibers explains the reduction of nonreflex muscle over-activity (i.e., dystonia, spastic dystonia, and associated reactions) observed immediately after SN. However, a sprouting process appears, explaining a voluntary muscle strength as well as nonreflex muscle over-activity reappearance after 8–12 months. This suggests that SN is effective on the reflex spastic over-activity (spasticity and spastic component of spastic dystonia) but probably not on the nonreflex over-activity (the dystonic part of the spastic dystonia, associated reactions and dystonia). Indeed, the nonreflex over-activity is immediately reduced after SN in relation to muscle weakness but recurs after 1 year by means of α motor fibers collateral sprouting.^[37]

Preoperative assessment

Before considering an SN, making an appropriate determination of the different types of spastic muscle over-activity of the impact of the different muscle's spasticity on the deformity and the presence of a muscle shortening is mandatory. A diagnostic nerve block with anesthetics (see Curriculum 2 in this Supplement) by transiently reducing the spasticity of the muscle(s) innervated by the targeted nerve(s) may address these questions. Interestingly, the diagnostic nerve blocks can predict the decrease in spasticity and the improvement in gait kinematics observed after tibial SN in case of spastic equinovarus foot.^[19]

BoNT-A can also be used to predict improvement. It has the advantage of acting for several months, allowing patients time to evaluate the functional benefits in daily living activities, while the diagnostic nerve block only acts for a few hours. Therefore, BoNT-A is frequently performed as an initial treatment after an effective diagnostic nerve block to provide a useful indicator of potential benefits that may be achieved with neurotomy. While BoNT-A is a useful treatment in itself, it is estimated less effective than neurotomy in the case of equinovarus foot).^[38–40]

Indications for neurotomy by frequency are given in Table 2.

Therapeutic effects and indications

Studies of the therapeutic effects of selective neurotomy have mainly been conducted in the lower limb. The majority

of studies have targeted the tibial nerve in cases of spastic equinovarus foot.^[35] In a study compared with BoNT-A, tibial neurotomy induced a higher reduction in ankle stiffness. Both treatments induced a comparable improvement of ankle kinematics during gait, while activity, participation, and quality of life stayed unchanged.^[39]

Neurotomy of the rectus femoris in the case of stiff knee gait has been investigated in an observational study in stroke patients, assessed in the short term.^[41] Compared with preoperative values, there was a significant increase in maximal walking distance, gait speed, and stride length at 3 months. All kinematic parameters improved, and the average internal early swing phase knee extension moment decreased. The duration of the rectus femoris EMG burst decreased postoperatively.

Small case series on neurotomy of obturator, musculocutaneous, median, and ulnar nerves have been published.^[42,43] Recently, tibial cryoneurotomy was described as a novel and safe alternative to open surgery.^[44]

Side effects

Reported side effects remained extremely low. Delay in healing due to sweating and maceration, hematoma, and local infections is reported. For lower limb neurotomy, systematic administration of anticoagulant therapy associated with early return to walking and rehabilitation is recommended. The most severe complication is sensory deficit and neuropathic pain when sensory fibers are unexpectedly sectioned.^[38] Some sensory fibers are closely related to motor fibers (i.e., tibial nerve: sensory fibers to the sole of the foot and motor nerve branches to the flexor digitorum longus) allowing surgical treatment of such muscle spasticity by means of tendon lengthening instead of SN.

COMPETENCY ASSESSMENT 2

The answers to these questions can be found at the end of this module before the references.

- The selective neurotomy surgical procedure requires (1 good answer)
 - A local anesthesia
 - Dissection of a nerve trunk in every case
 - A perioperative electrical stimulation to identify the motor nerve branch.
- The selective neurotomy induces (1 good answer)
 - A transient reduction in spasticity
 - A permanent clonus disappearance correlated to Hmax/Mmax reduction
 - A permanent reduction in nonreflex muscle over-activity.
- Neurotomy of the femoral nerve motor branch to the rectus femoris has been found to: (1 good answer)
 - Decrease gait speed
 - Increase falls
 - Increase stride length

Table 2: Indications for selective neurotomy

Condition	Nerve
Equinovarus foot	Tibial nerve and motor branches
Flexed elbow	Musculocutaneous nerve
Adducted hip	Obturator motor branches
Stiff knee gait	Rectus femoris motor branch
Internal shoulder rotation	Pectoralis major motor branch
Wrist-hand	Median-ulnar nerves

Other neurosurgical interventions for spasticity

Selective dorsal rhizotomy

Selective dorsal rhizotomy (SDR) is a well-studied therapy for lower extremity spasticity in children with cerebral palsy (CP) with good selective motor skills, minimal contractures, and good underlying strength.^[45-47]

The surgical technique involves single or multilevel laminotomies exposing L2–S2 nerve roots. During SDR, excitatory input from the dorsal roots is attenuated by sectioning (25%–70%) of individual rootlets. Theoretically, this selective sectioning results in improving the balance of the excitatory and inhibitory influences on the alpha motor neurons. EMG-monitoring is used to identify rootlets innervating more clinically abnormal muscle groups.

Some degree of lower limb weakness can be unmasked postoperatively by reducing the spasticity, making intensive physical therapy necessary. Patients must have the cognitive and social capacity for such an invasive intervention and rehabilitation. Long-term complications in children are infrequent and concern sensory dysfunction, bladder and bowel dysfunction, and back pain.^[48]

The role of this operation in the treatment of other spasticity causes in adults is less well defined as this procedure has not been systematically studied in contexts outside of CP. Multiple sclerosis and traumatic spinal cord injury are the most commonly reported non-CP diagnoses in patients who have undergone SDR.^[49] Although reported results are described satisfactory, no standardized outcome data are available outside of the CP population. Therefore, SDR, as an irreversible neuroablative procedure, should only be performed with great caution and in centers with an experienced multidisciplinary team.

Ventral rhizotomy

Ventral rhizotomies have been performed in individuals with complete spinal cord injury, but the consequent muscle atrophy increases the risk of pressure ulcers.^[50]

Microsurgical DREZotomy

Surgery in the DREZ was developed to treat some types of topographically limited pain. Because of its inhibitory side effects on muscular tone, the method is very rarely performed in patients with severe focalized spasticity.^[51,52]

Microsurgical DREZotomy (MDT) requires a strong knowledge of spinal cord anatomy. With bipolar coagulation forceps, the small myelinated fibers (considered nociceptive) and the large myelinated myotatic fibers are interrupted, while sparing the large myelinated fibers (considered sensory primary afferents).^[51]

MDT can theoretically be considered in hemiplegic patients for the treatment of the paralyzed upper limb when it is affected with severe and sometimes painful hyperspasticity. It could also be considered in severely disabled paraplegic patients, but intrathecal baclofen remains first choice.^[51]

MECHANISMS AND INDICATIONS FOR TENDON LENGTHENING AND TRANSFER

Shortening of muscles is a frequent occurrence subsequent to neurological damage. Among stroke patients, muscle shortening is often seen accompanied by increased muscle stiffness.^[53]

The “deforming spastic paresis” syndrome defines the association between spasticity, weakness, and muscle shortening.^[54] Early shortening correlates with poor recovery after stroke^[55] and physical therapy or orthosis is only poorly effective at prevention or treatment of this effect.^[56] Thus, surgical muscle and/or tendon lengthening may be indicated for restoring joint mobility.^[57] The indications for muscle lengthening are summarized in Figure 2.

The indications include muscle shortening that causes active limitation (gait and prehension); passive limitation (comfort, hygiene, dressing, aesthetic considerations, etc.); or pain (excessive foot pressure, claw toes, etc.).

Muscle shortening must be confirmed and differentiated from muscle over-activity by clinical examination and diagnostic nerve block with anesthetics (See Curriculum 1 in this Supplement). It should be resistant to physical therapy and orthoses, and there should be no osteo-articular limitation, for example due to heterotopic ossifications.

Muscle and tendon lengthening/release procedures

The surgical techniques commonly used are:

- Intramuscular lengthening, which allows a maximum of 0–20 mm of lengthening
- Tendon lengthening (section in Z shape + suture), which may be performed to the extent that is required. Postoperative immobilization is necessary
- Tenotomy or tenectomy is a total release of the tendon^[58]
- Proximal, gross muscle release (e.g., Page-Scaglietti surgery of the forearm which is a proximal release of the pronator teres and wrist/finger flexors)^[59]
- Tendon release procedures, such as described by Braun at the forearm (flexor digitorum superficialis to profundus tendon transfer)^[60]
- Whatever the technique, lengthening and release procedures allow for an adjustable gain in passive range of motion, contrary to transverse tenotomy/tenectomy. The actual gain that can be expected depends on the following rule: for each millimeter of lengthening, the joint can be moved 1 more degree. The exact lengthening is often decided during the procedure, depending on the improvement in passive range of motion obtained (see image below).

An alternative to typical surgical procedures described above is the percutaneous tenotomy using a large, sharp needle, through a simple skin puncture, performed under local anesthesia. It is used in case of troublesome deformities of a joint with no

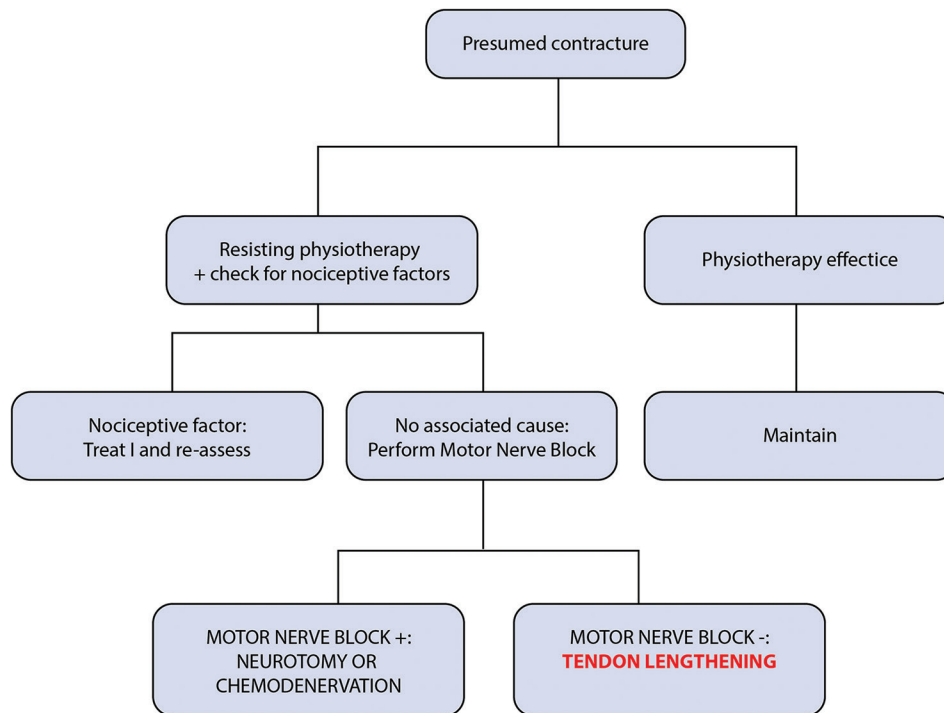


Figure 2: Indications for muscle and tendon lengthening

active function, if general anesthesia is contraindicated.^[58,61,62] It does not allow a precise correction but a transverse section of the tendon. It can be performed by nonsurgeons but requires former training on cadavers to allow for a good anatomical targeting.^[63]

Tendon transfer procedures

The aim of tendon transfers is to compensate for the paresis of some muscles relative to their over-active antagonists, to rebalance the forces at a joint. There are two main types of transfers: active or passive. In active transfers, the distal part of an active muscle is sectioned and implanted elsewhere so that its action at the joint level is more appropriate. Due to disordered motor control in spastic patients, the strength of the transferred muscle is hardly predictable, as is the integration of the transferred muscle into its new function. In passive transfers (also termed tenodesis), the tendon of a paretic muscle is fixed proximally to a bone or another tendon to increase its tension.

A wide variety of tendon transfers has been described in the literature [Figures 3-7].^[8] The most common active transfers, aiming at activating a function in patients with spastic paresis, target the foot and ankle in cases of excessive varus/supination, which is related to imbalance between an overactive tibialis anterior and weak long toe extensors and peronei. The SPLATT (SPLit tibialis Anterior Tendon Transfer) procedure,^[64] the tibialis posterior transfer on the dorsal part of the mid-foot or on peroneus brevis,^[65] and the tenodesis of the tibialis anterior on the peroneus brevis (Bardot's procedure)^[66] are the most well-known techniques.

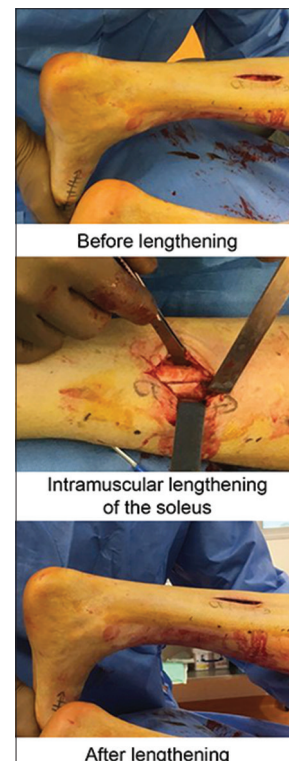


Figure 3: Surgical intramuscular lengthening of the soleus using a Z-lengthening procedure at the emergence of the soleus aponeurosis. This technique allowed a gain of approximately 10° of ankle dorsiflexion in the patient. Courtesy of Dr. G. Gadbled, Nantes University Hospital

Active transfers have also been described at the knee joint, to improve the stiff-knee gait pattern.^[67]

Table 3: Surgical techniques in muscle lengthening and transfers

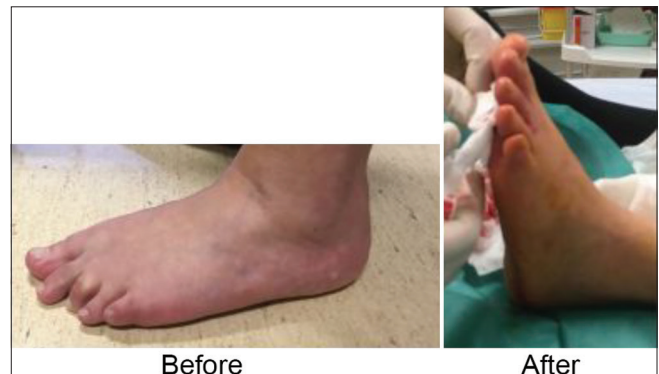
Condition	Involved muscle	Techniques
Equinus	Triceps surae	Intramuscular lengthening of soleus and/or gastrocnemii Achilles tendon lengthening
Varus related to muscle shortening	Tibialis posterior	Intramuscular lengthening and/or tenotomy of the tibialis posterior
Varus related to imbalance between tibialis anterior evertors	Tibialis anterior	SPLATT Tibialis posterior transfer on peroneus Tenodesis of the tibialis anterior on the peroneus brevis
Stiff knee gait	Rectus femoris	Distal release of rectus femoris (tenotomy) Distal rectal femoris transfers
Flexed elbow	Brachialis Biceps Brachioradialis	Tenotomy Tendon lengthening Proximal release
Flexed wrist	Wrist flexors	Tenotomy Intramuscular lengthening
Pronated forearm	Pronator teres	Intramuscular lengthening Proximal release
Clenched fist	Pronator quadratus Finger flexors Wrist/finger flexors and pronator teres Flexor digitorum superficialis Flexor digitorum profundus	Surgical release Tenotomy Intramuscular lengthening Proximal release (Page-Scaglietti procedure) Flexor digitorum superficialis on profundus tendon transfer (Braun's procedure) Tenotomy Intramuscular lengthening
Thumb-in-palm deformity	Flexor pollicis longus	Intramuscular lengthening of the FPL (flexion of the interphalangeal joint) Release of the intrinsic thumb muscles from the metacarpals (adducted thumb)

SPLATT: SPLit tibialis Anterior Tendon Transfer

**Figure 4:** Patient in a minimally responsive state and severe equinus before and after needle tenotomy of the Achilles tendon. Courtesy of Matthieu Gahier, MD and Raphaël Gross, MD, PhD

Active transfers are much less common at the upper limb in adult spastic patients. Tenodesis however can be used to correct a deformity in nonfunctional wrist/hands, such as the shortening tenodesis of the extensor carpi radialis brevis in case of a drop hand.^[8]

A summary of the involved muscles and the appropriate surgical techniques is given in Table 3.

**Figure 5:** Before/after needle tenotomy of the third long toe flexor in a patient with troublesome claw toe caused by spastic paresis of the lower limb due to a brain tumor. Courtesy of Matthieu Gahier, MD and Raphaël Gross, MD, PhD

Benefits and risks of muscle/tendon lengthening or transfers

Surgical intervention can help overcome the problems of shortened muscles by increasing muscle/tendon length. Consequently, there is decreased resistance to passive motion, making passive movements easier, but decreased voluntary muscle strength (e.g., Achilles tendon lengthening decrease triceps surae muscle strength). Surgery can result in restitution of the physiological range of motion so that it can produce an



Figure 6: Elderly patient with a clenched fist due to stroke. Pictures before and after tenotomy of the flexor digitorum superficialis and profundus. Courtesy of Matthieu Gahier, MD and Raphaël Gross, MD, PhD

improvement in passive function of the limb and sometimes improvement in active function, for example, as is seen with the Page-Scaglietti intervention. However, the decreased force of the muscle can be either positive or detrimental, and unfortunately, this is not predictable. Unlike for peripheral neurotomy, there is no predictive test for muscle/tendon lengthening procedures. The improvement will depend on the extent of the lengthening and the skill and experience of the surgeon. Besides, most surgical procedures require a period of postoperative immobilization which will impact on the patient's independence. The need for postoperative immobilization and its duration is variable and depends much on the surgeon's habit. In tendon lengthening with sutures, and in large release procedures, immobilization is the rule, lasting from 3 to 8 weeks. In tendon transfers, a firm immobilization is also needed, usually for 6–8 weeks, to protect the tendon suture. On the other hand, intramuscular tendon lengthening without postoperative immobilization is possible.

The risks of surgical intervention include bleeding; nerve injury with sensory loss and neuropathic pain; infection; skin breakdown; tendon rupture; delayed tendon healing; complex regional pain syndrome; overlengthening which can lead to over-correction (for example from pes equinus to pes talus) and a loss of passive or active function; and decompensation of the shortening of the toe flexors when an equinus foot is corrected (giving rise to claw toes to a variable extent).

In conclusion, tendon lengthening or transfer is the most effective way of correcting muscle shortening or imbalance of muscle groups. The problems due to the shortened muscle must be clearly identified and placed within the context of any concomitant issues. Different surgical techniques can produce

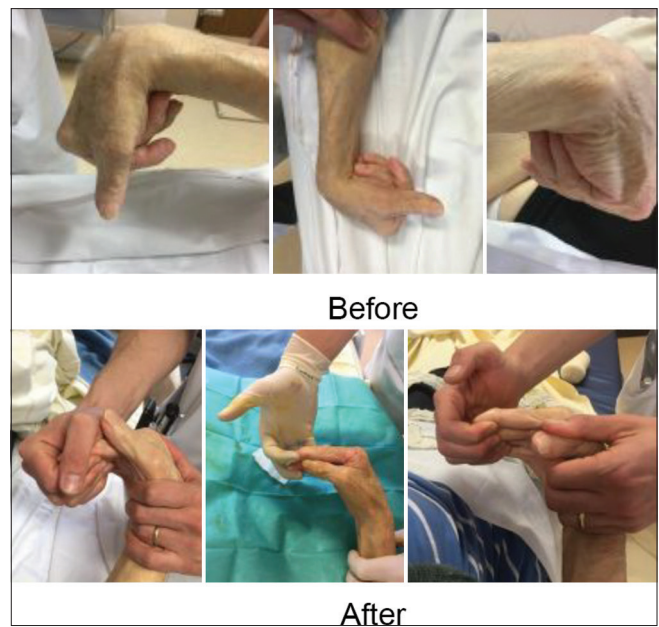


Figure 7: Patient with clenched fist due to shortening of interossei following a stroke. Pictures of before and after needle tenotomy of the intrinsic muscles of the second, third, and fourth intermetacarpal spaces. Courtesy of Matthieu Gahier, MD and Raphaël Gross, MD, PhD

different ranges of muscle lengthening. A drawback is that there is no predictive tool that will allow assessment of the expected improvement and a variable period of postoperative immobilization is required. Surgical muscle/tendon lengthening may be combined with other interventions, such as BoNT-A or neurotomy, which may improve walking capacities and achieve personal goals over single interventions alone.^[68]

MECHANISMS, INDICATIONS, BENEFITS, AND POTENTIAL RISKS OF ASSOCIATED BONE PROCEDURES

Associated bone and joint procedures may accompany tendon and muscle procedures. The use of bone procedures has decreased with improvement in early care. Further, soft tissue surgery (involving tendons and muscles, see above) has proved successful in spasticity and contracture treatment, especially when performed early and deformities are mild; it is more easily performed than bone procedures and provides more function and mobility.^[69]

Another drawback of bone surgery is that it may also require prolonged immobilization and prevent weightbearing. Patients with vascular disorders such as arteriopathy (e.g., after ischemic stroke) and smokers are at increased risk of bone healing and sepsis.

^[8] However, bone surgery should not be considered a last resort and should be included in a treatment program where appropriate.

Triple arthrodesis

The most common bony procedure is triple arthrodesis where the talocalcaneonavicular, subtalar, and calcaneocuboid joints are fused. This technique is used to correct severely pronated or supinated feet [Figure 8].

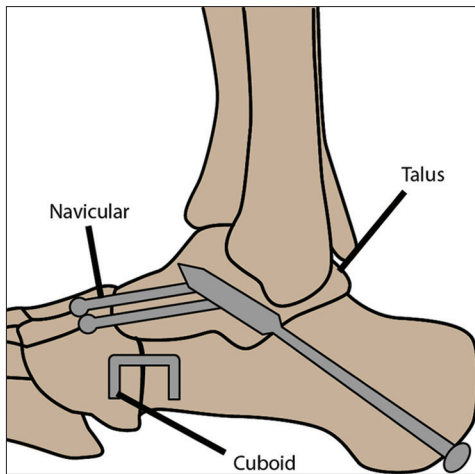


Figure 8: Fusion of the talocalcaneonavicular, subtalar, and calcaneocuboid joints

Long-term walking with an uncorrected triceps surae contracture is a frequent cause of foot pronation, and loss of function in the tibialis posterior can also cause pronation.^[8]

Resection of carpal bones and/or arthrodesis of the wrist

Other than heterotopic ossification (HO) resection, bony procedures are less common in the upper limb. However, CP, the most common cause of disability in children, is often accompanied by severe flexion contractures of the wrist and fingers and adduction of the thumb. Hand function is severely affected, maceration may occur in joint creases, and tight finger deformities may occur.^[70] When the deformities are not yet fixed, there are a number of treatment options (physiotherapy, BoNT-A, tendon transfers, or soft tissue lengthening); however, when deformities are fixed, resection of the proximal row of the carpal bones and wrist arthrodesis can be a solution.^[70] Lengthening of the wrist and extrinsic finger flexors may also be performed. The wrist is immobilized until bony fusion is achieved.^[8]

Failures

The success of neuro-orthopedic procedures is very dependent on the preoperative examination. The two main complications are overcorrection, with release of the previously masked dystonia of the antagonist muscle, and the recurrence of the deformity due to transferring a muscle that is too weak or after failure of sutures.^[8]

Combined surgery (with botulinum toxin A as complementary treatment)

In most cases of spasticity, it is strongly recommended to begin with preventative techniques. When prevention fails, then the orthopedic options discussed above aid in management of spasticity. BoNT can be used in conjunction with surgical options. Where BoNT aids in reducing spasticity, it cannot change the extracellular matrix, viscosity, or elasticity. BoNT can either be used as a clinical test to differentiate between spasticity and true contracture or to stimulate the effects of reducing muscle activity in a functional muscle.

With surgery, BoNT is best used preoperatively. It can aid in pain management as the surgical procedure can increase spasticity postoperatively. More importantly, preoperative injections can reduce the tension and promote lengthening of a muscle that is being transferred or lengthened.

COMPETENCY ASSESSMENT 3

The answers to these questions can be found at the end of this module before the references. Each question has one answer.

- Which technique among the followings is adequate for the treatment of severe foot supination during the swing phase of gait in stroke patients?
 - Split anterior tendon transfer procedure
 - Achilles tendon tenotomy
 - Tibialis anterior tenotomy.
- What kind of surgical treatment do you suggest for a patient without severe comorbidities who is able to walk and presents a fixed equinus following stroke?
 - Percutaneous needle tenotomy of the Achilles tendon
 - BoNT-A injections into the soleus
 - Intramuscular tendon lengthening of the triceps surae muscles.
- A known adverse event of neuro-orthopaedic surgery, such as muscle/tendon lengthening, is:
 - Hypercalcemia
 - Depressed alkaline phosphatase
 - Clonus
 - Overlengthening.

COMPETENCY ASSESSMENT ANSWERS

Competency Assessment 1

Are the following statements True or False?

- Regular refill of the pump reservoir is necessary to avoid potentially dangerous acute withdrawal symptoms that can occur when the drug reservoir is empty. (T)
- ITB therapy cannot be used in hemiplegic patients because it weakens the non-affected side. (F)
- ITB therapy cannot be combined with BoNT injections because the working mechanism is similar. (F)
- An ITB trial can be useful in helping to identify underlying voluntary strength. (T)
- Sexual dysfunction with disappearance of reflex erection in spinal cord injury patients is a potential side effect of ITB therapy. (T)
- Due to its working mechanism ITB therapy can resolve fixed contractures in patients with severe spasticity. (F)

Competency Assessment 2

- The selective neurotomy surgical procedure requires (1 good answer)
 - A local anesthesia
 - Dissection of a nerve trunk in every case
 - A perioperative electrical stimulation to identify the motor nerve branch.

2. The selective neurotomy induces (1 good answer)
 - a. A transient reduction in spasticity
 - b. A permanent clonus disappearance correlated to Hmax/Mmax reduction**
 - c. A permanent reduction in nonreflex muscle over-activity.
3. Neurotomy of femoral nerve motor branch to the rectus femoris has been found to: (1 good answer)
 - a. Decrease gait speed
 - b. Increase falls
 - c. Increase stride length.**

Competency Assessment 3

1. Which technique among the followings is adequate for the treatment of severe foot supination during the swing phase of gait in stroke patients?
 - a. Split anterior tendon transfer procedure**
 - b. Achilles tendon tenotomy
 - c. Tibialis anterior tenotomy.
2. What kind of surgical treatment do you suggest for a patient without severe comorbidities who is able to walk and presents a fixed equinus following stroke
 - a. Percutaneous needle tenotomy of the Achilles tendon
 - b. BoNT-A injections into the soleus
 - c. Intramuscular tendon lengthening of the triceps surae muscles.**
3. A known adverse event of neuro-orthopaedic surgery, such as muscle/tendon lengthening, is:
 - a. Hypercalcemia
 - b. Depressed alkaline phosphatase
 - c. Clonus
 - d. Overlengthening**

REFERENCES

1. Lorenz A. Über chirurgische behandlung der angeborenen spastischen gliedstare. *Wien Klin Wochenschr* 1887;21:25-7.
2. Stoffel A. The treatment of spastic contractures. *Am J Orthop Surg* 1912;10:611-44.
3. Keats S. Combined adductor-gracilis tenotomy and selective obturator-nerve resection for the correction of adduction deformity of the hip in children with cerebral palsy. *J Bone Joint Surg Am* 1957;39-A:1087-90.
4. Gros C. In: Springer, editor. *Spasticity – Clinical Classification and Surgical Treatment*. Vienna: Adv. Tech. Stand. Neurosurg.; 1979. p. 55-97.
5. Sindou M, Mertens P. Selective neurotomy of the tibial nerve for treatment of the spastic foot. *Neurosurgery* 1988;23:738-44.
6. Garland DE, Thompson R, Waters RL. Musculocutaneous neurectomy for spastic elbow flexion in non-functional upper extremities in adults. *J Bone Joint Surg Am* 1980;62:108-12.
7. Brunelli G, Brunelli F. Partial selective denervation in spastic palsies (hyponeurotization). *Microsurgery* 1983;4:221-4.
8. Genêt F, Denormandie P, Keenan MA. Orthopaedic surgery for patients with central nervous system lesions: Concepts and techniques. *Ann Phys Rehabil Med* 2019;62:225-33.
9. Bouvier M. Mémoire sur la Section du Tendon d'Achille dans le Traitement des Pieds-Bots. *Me'm Académie R Me' decine*. Vol. VII. Mémoire de l'Académie Royale de Médecine; 1838.
10. Hernigou P, Gravina N, Potage D, Dubory A. History of club-foot treatment; part II: Tenotomy in the nineteenth century. *Int Orthop* 2017;41:2205-12.
11. Foerster O. Über eine neue operative methode der behandlung spastischer lähmungen mittels resektion hinterer rückenmarkswurzeln. *Z Orthop Chir* 1908;22:463-74.
12. Foerster O. On the indications and results of the excitation of posterior spinal nerve roots in men. *Surg Gynecol Obstet* 1913;16:463-74.
13. Gros C, Ouaknine G, Vlahovitch B, Frèrebeau P. Selective posterior radicotomy in the neurosurgical treatment of pyramidal hypertension. *Neurochirurgie* 1967;13:505-18.
14. Sindou M, Mifsud JJ, Boisson D, Goutelle A. Selective posterior rhizotomy in the dorsal root entry zone for treatment of hyperspasticity and pain in the hemiplegic upper limb. *Neurosurgery* 1986;18:587-95.
15. Guttmann, L. The treatment and rehabilitation of patients with injuries of the spinal cord. In Cope, Z. (ed.), *Medical History of the Second World War, Surgery*. HMSO, London, 1953;2:422-516.
16. Segnarbieux F, Frerebeau P. The different (open surgical, percutaneous thermal, and intrathecal chemical) rhizotomies for the treatment of spasticity. In: Sindou M, Abbott R, Kervel Y, editor. *Neurosurgery for Spasticity A Multidisciplinary Approach*. Vienna: Springer Verlag; 1991. p. 133-9.
17. Penn RD, Kroin JS. Intrathecal baclofen alleviates spinal cord spasticity. *Lancet* 1984;1:1078.
18. Penn R, Kroin J. Long-term intrathecal baclofen infusion for treatment of spasticity. *J Neurosurg* 1987;66:181-5.
19. Deltombe T, Bleyenheuft C, Gustin T. Comparison between tibial nerve block with anaesthetics and neurotomy in hemiplegic adults with spastic equinovarus foot. *Ann Phys Rehabil Med* 2015;58:54-9.
20. Taricco M, Adone R, Pagliacci C, Telaro E. Pharmacological interventions for spasticity following spinal cord injury. *Cochrane Database Syst Rev*. 2000;2000(2):CD001131. doi:10.1002/14651858.CD001131.
21. McIntyre A, Mays R, Mehta S, Janzen S, Townson A, Hsieh J, et al. Examining the effectiveness of intrathecal baclofen on spasticity in individuals with chronic spinal cord injury: A systematic review. *J Spinal Cord Med* 2014;37:11-8.
22. Creamer M, Cloud G, Kossmehl P, Yochelson M, Francisco GE, Ward AB, et al. Effect of intrathecal baclofen on pain and quality of life in poststroke spasticity. *Stroke* 2018;49:2129-37.
23. Middel B, Kuipers-Upmeyer H, Bouma J, Staal M, Oenema D, Postma T, et al. Effect of intrathecal baclofen delivered by an implanted programmable pump on health related quality of life in patients with severe spasticity. *J Neurol Neurosurg Psychiatry* 1997;63:204-9.
24. Saulino M, Ivanhoe CB, McGuire JR, Ridley B, Shilt JS, Boster AL. Best practices for intrathecal baclofen therapy: Patient selection. *Neuromodulation* 2016;19:607-15.
25. Meythaler JM, Guin-Renfroe S, Brunner RC, Hadley MN. Intrathecal baclofen for spastic hypertonia from stroke. *Stroke* 2001;32:2099-109.
26. Francisco GE, Boake C. Improvement in walking speed in poststroke spastic hemiplegia after intrathecal baclofen therapy: A preliminary study. *Arch Phys Med Rehabil* 2003;84:1194-9.
27. Boster AL, Adair RL, Gooch JL, Nelson ME, Toomer A, Urquidez J, et al. Best practices for intrathecal baclofen therapy: Dosing and long-term management. *Neuromodulation* 2016;19:623-31.
28. Bilsky GS, Saulino M, O'Dell MW. Does every patient require an intrathecal baclofen trial before pump placement? *PM R* 2016;8:802-7.
29. Borriani L, Bensmail D, Thiebaut JB, Hugeron C, Rech C, Jourdan C. Occurrence of adverse events in long-term intrathecal baclofen infusion: A 1-year follow-up study of 158 adults. *Arch Phys Med Rehabil* 2014;95:1032-8.
30. Sampson FC, Hayward A, Evans G, Morton R, Collett B. Functional benefits and cost/benefit analysis of continuous intrathecal baclofen infusion for the management of severe spasticity. *J Neurosurg* 2002;96:1052-7.
31. Bensmail D, Ecoffey C, Ventura M, Albert T; SOFMER French Society for Physical Medicine and Rehabilitation. Chronic neuropathic pain in patients with spinal cord injury. What is the efficacy of regional interventions? Sympathetic blocks, nerve blocks and intrathecal drugs. *Ann Phys Rehabil Med* 2009;52:142-8.
32. Slof J, Serrano D, Álvarez M, Álvarez López-Dóriga M, Marqués T, Benito J, et al. Cost-effectiveness model results of intrathecal baclofen therapy compared to conventional medical management in patients with non-focal disabling spasticity who are resistant or intolerant to oral

- therapy at the Institut Guttman. *Value Health* 2014;17:A399-400.
33. Saulino M, Guillemette S, Leier J, Hinnenenthal J. Medical cost impact of intrathecal baclofen therapy for severe spasticity. *Neuromodulation* 2015;18:141-9.
 34. Decq P. Peripheral neurotomies for the treatment of focal spasticity of the limbs. *Neurochirurgie* 2003;49:293-305.
 35. Bollens B, Deltombe T, Detrembleur C, Gustin T, Stoquart G, Lejeune TM. 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.
 36. Sindou M, Georgoulis G, Mertens P. Surgery in dorsal root entry zone. In: *Neurosurg. Spasticity*. Verlag: Springer Verlag; 2014. p. 141-56.
 37. 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.
 38. Rousseaux M, Buisset N, Daveluy W, Kozlowski O, Blond S. Comparison of botulinum toxin injection and neurotomy in patients with distal lower limb spasticity. *Eur J Neurol* 2008;15:506-11.
 39. 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.
 40. Deltombe T, Lejeune T, Gustin T. Botulinum toxin type A or selective neurotomy for treating focal spastic muscle overactivity? *Ann Phys Rehabil Med* 2019;62:220-4.
 41. 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.
 42. Shin DK, Jung YJ, Hong JC, Kim MS, Kim SH. Selective musculocutaneous neurotomy for spastic elbow. *J Korean Neurosurg Soc* 2010;48:236-9.
 43. Gras M, Leclercq C. Spasticity and hyperselective neurectomy in the upper limb. *Hand Surg Rehabil* 2017;36:391-401.
 44. Winston P, Mills PB, Reebye R, Vincent D. Cryoneurotomy as a percutaneous mini-invasive therapy for the treatment of the spastic limb: Case presentation, review of the literature, and proposed approach for use. *Arch Rehabil Res Clin Transl* 2019;1:100030.
 45. McLaughlin J, Bjornson K, Temkin N, Steinbok P, Wright V, Reiner A, *et al.* Selective dorsal rhizotomy: Meta-analysis of three randomized controlled trials. *Dev Med Child Neurol* 2002;44:17-25.
 46. Tedroff K, Hägglund G, Miller F. Long-term effects of selective dorsal rhizotomy in children with cerebral palsy: A systematic review. *Dev Med Child Neurol* 2020;62:554-62.
 47. Grunt S, Becher JG, Vermeulen RJ. Long-term outcome and adverse effects of selective dorsal rhizotomy in children with cerebral palsy: A systematic review. *Dev Med Child Neurol* 2011;53:490-8.
 48. Steinbok P. Selective dorsal rhizotomy for spastic cerebral palsy: A review. *Childs Nerv Syst* 2007;23:981-90.
 49. Gump WC, Mutchnick IS, Moriarty TM. Selective dorsal rhizotomy for spasticity not associated with cerebral palsy: Reconsideration of surgical inclusion criteria. *Neurosurg Focus* 2013;35:E6.
 50. Barolat G. Surgical management of spasticity and spasms in spinal cord injury: An overview. *J Am Paraplegia Soc* 1988;11:9-13.
 51. Sindou M. Microsurgical DREZotomy (MDT) for pain, spasticity, and hyperactive bladder: A 20-year experience. *Acta Neurochir (Wien)* 1995;137:1-5.
 52. Sindou M, Georgoulis G. Keyhole interlaminar dorsal rhizotomy for spastic diplegia in cerebral palsy. *Acta Neurochir (Wien)* 2015;157:1187-96.
 53. Le Sant G, Nordez A, Hug F, Andrade R, Lecharte T, McNair PJ, *et al.* Effects of stroke injury on the shear modulus of the lower leg muscle during passive dorsiflexion. *J Appl Physiol* (1985) 2019;126:11-22.
 54. Gracies JM, Bayle N, Vinti M, Alkandari S, Vu P, Loche CM, *et al.* Five-step clinical assessment in spastic paresis. *Eur J Phys Rehabil Med* 2010;46:411-21.
 55. de Gooijer-van de Groep KL, de Groot JH, van der Krogt H, de Vlugt E, Arendzen JH, Meskers CG. Early shortening of wrist flexor muscles coincides with poor recovery after stroke. *Neurorehabil Neural Repair* 2018;32:645-54.
 56. Katalinic OM, Harvey LA, Herbert RD. Effectiveness of stretch for the treatment and prevention of contractures in people with neurological conditions: A systematic review. *Phys Ther* 2011;91:11-24.
 57. 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 group. *J Rehabil Med* 2017;49:461-8.
 58. Corioan F, Jourdan C, Froger J, Anquetil C, Choquet O, Coulet B, *et al.* Percutaneous needle tenotomy for the treatment of muscle and tendon contractures in adults with brain damage: Results and complications. *Arch Phys Med Rehabil* 2017;98:915-22.
 59. Thévenin-Lemoine C, Denormandie P, Schnitzler A, Lautridou C, Allieu Y, Genêt F. Flexor origin slide for contracture of spastic. *J Bone Joint Surg Am* 2013;95:446-53.
 60. Facca S, Louis P, Isner ME, Gault D, Allieu Y, Liverneaux P, Braun's flexor tendons transfer in disabled hands by central nervous system lesions. *Orthop Traumatol Surg Res* 2010;96:656-61.
 61. Schnitzler A, Diebold A, Parratte B, Tliba L, Genêt F, Denormandie P. An alternative treatment for contractures of the elderly institutionalized persons: Microinvasive percutaneous needle tenotomy of the finger flexors. *Ann Phys Rehabil Med* 2016;59:83-6.
 62. Schnitzler A, Genêt F, Diebold A, Mailhan L, Jourdan C, Denormandie P. Lengthening of knee flexor muscles by percutaneous needle tenotomy: Description of the technique and preliminary results. *PLoS One* 2017;12:e0182062.
 63. Chesnel C, Genêt F, Almangour W, Denormandie P, Parratte B, Schnitzler A. Effectiveness and complications of percutaneous needle tenotomy with a large needle for muscle contractures: A cadaver study. *PLoS One* 2015;10:e0143495.
 64. Deltombe T, Decloedt P, Jamart J, Costa D, Leboul P, Gustin T. Split anterior tibialis tendon transfer (Splatt) and achilles tendon lengthening for the correction of the varus foot after stroke a prospective longitudinal study. *Int J Phys Med Rehabil* 2014;s5:006. [doi: 10.4172/2329-9096.s5-006].
 65. Sturbois-Nachef N, Allart E, Grauwin MY, Rousseaux M, Thévenon A, Fontaine C. Tibialis posterior transfer for foot drop due to central causes: Long-term hindfoot alignment. *Orthop Traumatol Surg Res* 2019;105:153-8.
 66. Bardot A, Delarque A, Curvale G, Peragut JC. Orthopedic surgical corrections of spastic disorders. In: *Neurosurg. Spasticity a Multidisc. Approach*, Vienna: Springer; 1991. p. 201-8.
 67. Namdari S, Pill SG, Makani A, Keenan MA. Rectus femoris to gracilis muscle transfer with fractional lengthening of the vastus muscles: A treatment for adults with stiff knee gait. *Phys Ther* 2010;90:261-8.
 68. Deltombe T, Gilliaux M, Peret F, Leeuwerck M, Wautier D, Hanson P, *et al.* Effect of the neuro-orthopedic surgery for spastic equinovarus foot after stroke: A prospective longitudinal study based on a goal-centered approach. *Eur J Phys Rehabil Med* 2018;54:853-9.
 69. Gatín L, Schnitzler A, Calé F, Genêt G, Denormandie P, Genêt F. Soft tissue surgery for adults with nonfunctional, spastic hands following central nervous system lesions: A retrospective study. *J Hand Surg Am* 2017;42:1035.e1-7.
 70. Donadio J, Upex P, Bachy M, Fitoussi F. Wrist arthrodesis in adolescents with cerebral palsy. *J Hand Surg Eur Vol* 2016;41:758-62.