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SPECIAL REPORT

ASSESSMENT AND TREATMENT OF SPASTIC EQUINOVARUS FOOT AFTER STROKE: GUIDANCE FROM THE MONT-GODINNE INTERDISCIPLINARY GROUP

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Objective: **To present interdisciplinary practical guidance for the assessment and treatment of spastic equinovarus foot after stroke.**

Results: **Clinical examination and diagnostic nerve block with anaesthetics determine the relative role of the factors leading to spastic equinovarus foot after stroke: calf spasticity, triceps surae - Achilles tendon complex shortening and dorsiflexor muscles weakness and/or imbalance. Diagnostic nerve block is a mandatory step in determining the cause(s) of, and the most appropriate treatment(s) for, spastic equinovarus foot. Based on interdisciplinary discussion, and according to a patient-oriented goal approach, a medical and/or surgical treatment plan is proposed in association with a rehabilitation programme. Spasticity is treated with botulinum toxin or phenol–alcohol chemodenervation and neurotomy, shortening is treated by stretching and muscle-tendon lengthening, and weakness is treated by ankle-foot orthosis, functional electrical stimulation and tendon transfer. These treatments are frequently combined.**

Conclusion: **Based on 20 years of interdisciplinary expertise of management of the spastic foot, guidance was established to clarify a complex problem in order to help clinicians treat spastic equinovarus foot. This work should be the first step in a more global international consensus.**

Key words: stroke; muscle spasticity; foot; guidance.

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Stroke is the third most common cause of death and the primary cause of severe disability in industrialized countries. Following stroke, approximately 80% of patients regain walking function with decreased gait velocity and asymmetrical gait pattern (1). Spastic equinovarus foot (SEVF) is one of the most common disabling deformities observed among hemiplegic patients. SEVF is frequently associated with other kinematic gait abnormalities, such as stiff knee gait, genu recurvatum, and painful claw toes. SEVF deformity forces the patient to increase their hip and knee flexion in the swing phase. If they are unable to do this (e.g. if they have associated stiff knee gait), the patient will present a hip circumbduction in the swing phase. Correction of such equinus may therefore improve distal as well as proximal gait disturbances.

SEVF deformity has 4 main causes (2, 3). The first is spasticity of the calf muscles (soleus, gastrocnemius, tibialis posterior, flexor digitorum and flexor hallucis longus muscles), responsible for SEVF in the stance phase of gait and for painful toe curling with callosities on the pulp and dorsum of the toes. The peroneus longus and brevis muscles may also be spastic (often with clonus), but such spasticity is useful to limit the varus and stabilize the ankle. Secondly, the spastic muscles have a tendency to remain in a shortened position for prolonged periods, which, in turn, results in soft-tissue changes and contractures, leading to a fixed deformity (4). Thirdly, weakness of the ankle dorsiflexor muscles (tibialis anterior, extensor digitorum and hallucis muscles) as well as the peroneus longus and brevis muscles is responsible for drop-foot in the swing phase of gait. Such weakness is often emphasized by triceps spastic co-contraction and/or contracture. The weakness also affects the triceps surae muscles, leading to a lack of propulsion at the end of the stance phase of gait. Lastly, an imbalance between the tibialis anterior and the peroneus muscles leads to varus of the hind-foot in the swing phase, as peroneus activation must compensate for physiological varus positioning related to contraction of the tibialis anterior. In such a case, the foot will be placed in an unstable varus position during the swing phase and at the beginning of the stance phase.

The respective role of the main causes of SEVF (spasticity, shortening, weakness, and imbalance) varies from patient to patient, and therapeutic decisions are therefore challenging. Indeed, as emphasized by Fuller, the causes of SEVF are varied and complex, due to a variety of deforming forces, and thus a single procedure does not exist to correct all deformities (3). Hence there is a need for guidance and guidelines.

Treatments for SEVF described in the literature are multimodal and include rehabilitation, orthosis, botulinum toxin (BoNT-A) injections, alcohol and phenol nerve blocks, functional neurosurgery (selective

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neurotomy and intrathecal baclofen therapy) and orthopaedic surgery (tendon transfer, tendon lengthening and bone surgery) (5). SEVF rehabilitation programmes include strengthening of the tibialis anterior and peroneus muscles, electrical stimulation, stretching of the triceps surae to reduce spasticity and prevent contracture, and gait and balance training. Modern therapeutic approaches, such as task-oriented strategy and treadmill with bodyweight support, are promoted. Several publications support the effectiveness of these treatments in SEVF (6–8). However, only 3 studies have compared different treatment options (9–11). A systematic review of surgical correction in adult patients with stroke emphasized the need to compare treatments in order to generate evidence on which to base algorithms (8). In fact, no practical guidelines are available for use in daily practice. Evidence regarding choice of treatment is poor, thus therapeutic decision-making is based on professional personal preferences and beliefs rather than on scientific evidence. An interdisciplinary approach with a physical medicine and rehabilitation (PMR) specialist and rehabilitation team, neurosurgeon, and orthopaedic surgeon is therefore mandatory in order to optimize treatments.

The aim of this paper is to present and discuss the Mont-Godinne interdisciplinary guidance (Fig. 1), based on the existing literature and on 20 years of experience of an interdisciplinary medical and surgical approach to SEVF.

CLINICAL ASSESSMENT AND GOAL

Before making any therapeutic decision, a precise knowledge of the patient's medical history and a complete evaluation according to the World Health Organization's (WHO) International Classification of Functioning, Disability and Health (ICF) model is mandatory. Such assessment is necessary to determine the different causes and consequences of gait abnormalities, the related complaints of the patient, and to anticipate the effect of treatments.

Assessment of impairment level includes muscle strength testing (Medical Research Council MRC grade 0–5), spasticity testing (modified Ashworth scale and Tardieu scale), passive and active range of motion assessment (ROM) and gait kinematics (12). Assessment of activity and participation levels includes gait parameters (gait speed, step cadence and step length) obtained by means of a 10-m walk test and/or a 6-min walk test, pain scale (VAS scale), walking aids requirement, functional walking category (FWC) and functional ambulation categories (FAC). Of course, this list is not exhaustive. In our practice, we systematically recorded a video (front and profile)

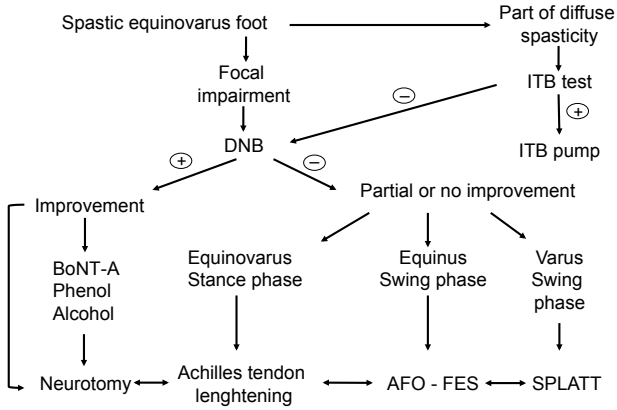

Fig. 1. Mont-Godinne Guidance Pathway.

of the gait kinematic including trunk, hip, knee and ankle view. Video is a simple, low-cost assessment tool, making therapeutic decision and follow-up easy and interactive with the patient. If possible, an instrumented gait analysis collecting kinetic, kinematic and dynamic electromyographic data may improve gait evaluation and therapeutic decision-making, as it facilitates agreement between surgical procedures planned by different surgeons (3).

Finally, the most concrete and realistic goals that can be achieved by an appropriate treatment must be determined with the patient and their caregivers by means of the goal attainment scaling (GAS) scale, which allows person-centred improvements to be measured (13). Improving transfer, stability and gait, walking without shoes, pain reduction, facilitating use of braces and/or shoe wear, and orthosis release are all possible goals. Therapeutic decisions must be taken with regard to patient specificity, clinical examination and expected goals. The evaluation performed after treatment will determine whether the goal has been achieved according to the goal achievement strategy.

DIAGNOSTIC NERVE BLOCKS WITH ANAESTHETICS

Determination of the role of the spasticity of different calf muscles in SEVF by means of diagnostic nerve block (DNB) with anaesthetics is the first step before any therapeutic decision is taken (14). The DNB consists of injecting a small dose of local anaesthetic at the level of the motor nerve innervating the spastic muscles in order to temporarily suppress their spasticity. The DNB can be made at the level of the tibial nerve (mixed sensorimotor nerve block), inducing a non-selective decrease in the spasticity of all the muscles innervated by the tibial nerve (soleus, gastrocnemii, tibialis posterior and flexor digitorum muscles) and a sensory deficit in the sole of the feet that interferes

with gait (15). Such a global tibial nerve DNB is only used to differentiate spasticity from contracture. To determine the respective role of the different muscles of the calf in SEVF, the DNB has to be selectively performed on one nerve after another, at the level of the motor nerve branches to the soleus, the tibialis posterior and the lateral and medial gastrocnemius, respectively. According to the clinical examination (i.e. spasticity predominant at the gastrocnemius) the order may vary. The precise location of the soleus and tibialis posterior motor nerve branches has been determined by computed tomography (CT) scanning according to the bony prominences (respectively 10 mm and 45 mm above the upper extremity of the fibula and 17 mm lateral to the middle of the popliteal fossa) (16). Localization of the motor nerve branches to the soleus and gastrocnemius muscles has also been determined in cadavers (17).

The DNB technique is simple, low cost and takes approximately 10–15 min: a disposable needle for conduction anaesthesia, gauge 23 (50 or 100 mm length) is used, coupled to a portable electrical stimulator or electromyograpy apparatus. The needle transmits electrical stimulation. A 1–2 ml dose of local anaesthetic (lidocaine 2% or bupivacaine 0.5%) is injected at the site where clinical muscular contraction of the selected muscles is seen at 4 mA intensity and 0.01 ms duration of stimulation, which is consistent with close contact of the needle with the nerve. H-reflex monitoring may help to target the appropriate motor nerve branch to the soleus and gastrocnemius. After each selective DNB, the patient is assessed to evaluate spasticity reduction and gait improvement. If necessary, the session can be renewed a few days later, using a different nerve selection order. The DNB leads to a decrease in spasticity (from 2–3 points on the Ashworth scale) and clonus disappearance in the selected muscles within a few minutes, lasting for 2 h. Such reduction is correlated with a decrease in the soleus Hmax/Mmax ratio, indicating preferential susceptibility of muscle spindle afferents to local anaesthetics (18). The procedure is safe. Only Parziale et al. reported a compartment syndrome developed in the volar forearm after completion of a median nerve block under anticoagulation therapy (19). Although such a complication has not been reported in the leg, we recommend stopping anticoagulant medications before DNB.

Selective DNB has several advantages. First, it helps to determine the role of each spastic muscle in the SEVF deformity. This is useful, especially before surgery (neurotomy for instance) and chemodenervation (botulinum toxin and phenol). For instance, determination of the respective role of soleus and gastrocnemius spasticity is important: soleus muscle spasticity is usually clonic and responsible for triceps clonus in 80% of cases (20), while gastrocnemius muscle spasticity is usually more tonic and much more susceptible to develop spastic co-contraction. Buffenoir et al. showed that soleus nerve block appeared more appropriate than gastrocnemius nerves block in relieving spasticity (14). Effective correction of SEVF deformity after selective block of the soleus nerve is consistent with a classical predominant role for soleus spasticity, avoiding inappropriate and unnecessary treatment of other muscles. If the improvement is incomplete, evaluation must be completed by tibialis posterior and/or gastrocnemius nerve blocks. The role of tibialis posterior muscle spasticity in forefoot varus must be established, since inappropriate treatment may lead to iatrogenic valgus of the foot and increased toe flexion in the stance phase. Secondly, tibial DNB (a mixed sensorimotor block) also affects sensory fibres innervating the sole of the foot and induces transient disturbances in the sensation of the plantar foot, which could adversely affect walking ability (15). Performing selective motor nerve branch blocks without affecting sensory fibres reduces the risk of sensory disturbances and improves functional evaluation. Thirdly, treatment of equinus deformity may frequently, but not always, lead to a painful claw toe, which may appear after DNB, allowing the flexor digitorum muscles to be treated before the claw toe becomes symptomatic. Finally, DNB allows the result of a treatment to be predicted (21).

IF DIAGNOSTIC NERVE BLOCK IMPROVES THE DEFORMITY THEN SPASTICITY IS THE PROBLEM

A complete correction of SEVF in the swing and stance phase of gait after DNB indicates that spasticity is fully responsible for the SEVF and that appropriate treatment is indicated. Systemic pharmacological treatments (baclofen and tizanidine) of the spasticity are usually recommended when the muscle overactivity is diffused to involve a large number of affected muscles. However, adverse effects (drowsiness, sedation, hypotension and weakness) usually arise at lower doses than those required to reduce spasticity, which limits the benefit of the medication. Furthermore, no studies have dealt with the specific effects of oral medications on SEVF. Therefore, we do not prescribe oral antispastic drugs as a specific treatment of the SEVF. Since SEVF is a focal problem, it should be treated by a focal chemodenervation technique, such as alcohol–phenol block or BoNT-A injections. First described by Tardieu, chemodenervation with alcohol and phenol was extensively used before the development of BoNT-A

and functional neurosurgery (22). Alcohol and phenol denature proteins and injure nerve cells, producing non-selective neuronal degeneration and surrounding fibrosis. Several uncontrolled studies demonstrated reduction in spasticity after motor nerve and neuromuscular block with alcohol and phenol, with a large range of reduction in spasticity and duration of action (23). A randomized controlled trial (RCT) demonstrated that a 3-ml injection of 5% phenol into the tibial nerve is less effective in reducing plantar flexor spasticity at week 4 than 400 U onabotulinum toxin A in the soleus, gastrocnemius and tibialis posterior muscles. Moreover, 30% of patients reported dysaesthesia of the sole of the foot, interfering with gait after phenol injections (9). Due to the risk of neuropathic pain, we do not recommend injecting the tibial sensory-motor nerve trunk, but only the collateral motor nerve branches to the soleus and tibialis posterior if DNB improves SEVF without additional sensory deficit related to diffusion of the anaesthetics to the tibial nerve.

In the present guidance, botulinum toxin type A (BoNT-A) is the first-line treatment of SEVF related to spasticity. BoNT-A is the most potent neurotoxin known to induce a reversible neuromuscular junction blockade of the injected muscles lasting for 3–6 months. BoNT-A is the treatment of focal spasticity with the highest level of evidence. Several double-blind RCTs have demonstrated the ability of BoNT-A to reduce spasticity of the ankle plantar flexors and invertors, pain and the need for walking aids in a dose-dependent manner, and to increase active dorsiflexion of the ankle compared with placebo (24, 25). A systematic review revealed a small, but significant, increase in gait velocity (6). However, since there is no consensus concerning which muscles to treat and the dose, the subjective opinions of the treating doctor prevail over objective analysis of the patient's characteristics. In our practice, BoNT-A is injected at the soleus, lateral and medial gastrocnemius, tibialis posterior and/or flexor hallucis and digitorum muscles, based on clinical evaluation and on improvements after DNB. Some colleagues recommend the use of BoNT-A injections as a long-lasting block instead of DNB. However, the improvement will be delayed (thus making appropriate assessment more complex) and the cause of a lack of improvement (e.g. not the best choice of muscles, too low dose) will not be determined. BoNT-A injections should be made under electromyography, electrostimulation (ES) or ultrasound (US) guidance. Although the method of choice remains a matter of debate, US appears to be more effective than ES (26). In our practice, we use both techniques. After BoNT-A injections, early physiotherapy, neuromuscular electrical stimulation of the injected muscles, and stretching seem effective in increasing the effects of BoNT-A (27). BoNT-A is safe, but the maximal dose that can be injected is limited, while its benefits are reversible. This leads to the use of repeat injections, which supports the need for a permanent surgical solution, such as neurotomy.

Selective tibial neurotomy (STN) involves partial section of the motor nerve branches of the tibial nerve (soleus, lateral and medial gastrocnemius, tibialis posterior and/or flexor hallucis longus) innervating the spastic muscles. This neurosurgical procedure is performed under general anaesthesia without muscle relaxant drugs in order to preserve nerve stimulation response. Through a vertical cutaneous incision below the popliteal fossa, the motor nerve branches of the tibial nerve are identified with intraoperative tripolar electrical stimulation. The motor nerve branches to treat are defined according to the DNB assessment and preoperative discussion (21). The degree of nerve section usually ranges from 50% to 80% and depends on the extent of the spasticity and muscular contraction under electrical stimulation. Partial section of the motor nerve branch involves the afferent Ia fibres mediating the spastic monosynaptic arc reflex, explaining a permanent reduction in spasticity correlated with a permanent decrease in Hmax/Mmax ratio. The section also concerns the efferent α motor fibres inducing a muscle weakness which resolves after 8–12 months due to collateral reinnervation (18). The sensory fibres of the tibial nerve must be carefully spared in order to avoid sensory deficits and neuropathic pain. As the motor nerve branches to the flexor digitorum longus are mixed with the sensory fibres innervating the sole of the foot at the level of the surgical approach, we recommend avoiding neurotomy for this muscle and treating the claw toe by means of tendon surgery. A systematic review of uncontrolled studies demonstrated the long-lasting effects of STN and suggested that STN is effective to reduce triceps spasticity, to improve gait kinematics and to achieve pre-operatively defined goals (7). Furthermore, the beneficial effect of STN can be predicted by means of DNB (21). An RCT showed that STN induces a greater reduction in ankle spasticity compared with BoNT-A injections, whereas both treatments induced a comparable improvement in ankle kinematics during gait (11). The complications rate of the technique is low (approximately 2–5%). Adverse effects include transient oedematous swelling of the ankle and foot, wound infection and hypoesthesia and/or neuropathic pain, probably related to excessive manipulation or inappropriate section of the sensory fibres of the tibial nerve, especially if the fascicles innervating the flexor digitorum longus muscle are dissected. One publication reported foot deformity recurrence after STN related to contracture, supporting

the need for a rehabilitation programme including triceps auto-stretching to maintain the improvement (28). If the patient does not want and/or cannot benefit from BoNT-A injections or STN, a rigid ankle-foot orthosis (AFO) strong enough to constraint spasticity or an orthotic shoe can be an option. However, the level of constraint frequently limits the AFO tolerance so that one of the most frequent goals for BoNT-A a or STN treatment is AFO release or improved tolerance.

IF DIAGNOSTIC NERVE BLOCK DOES NOT IMPROVE THE DEFORMITY THEN SPASTICITY IS NOT THE PROBLEM

In case of mixed participation of triceps spasticity, triceps surae – Achilles tendon shortening and dorsiflexor muscles weakness and/or imbalance, DNB is only partially effective or ineffective to improve SEVF. The main difficulty is to determine the respective role of these different causes, as the treatments will differ accordingly. If DNB helps to determine the implication of the spasticity, clinical examination (or gait analysis) can determine the other responsibilities. Such determination is probably the most challenging parameter in SEVF management. This also explains why publications dealing with SEVF surgery have reported different surgical techniques (i.e. lengthening, transfer, neurotomy) during the same procedure making comparison hazardous.

Triceps surae – Achilles tendon shortening

SEVF persistence in the swing and stance phase of the gait after DNB indicates that a triceps surae – Achilles tendon complex shortening is responsible. The clinical assessment must differentiate soleus from gastrocnemius contracture. Limited dorsal ankle flexion with extended knee is related to the gastrocnemius shortening only, while limited dorsal ankle flexion with flexed and extended knee is related to soleus-gastrocnemius complex shortening. If the ankle passive ROM limitation does not exceed 20° of fixed plantar flexion, we propose an intensive stretching programme, performed daily with the patient in the standing position. Several uncontrolled studies have suggested a potential benefit from prolonged muscle stretching on the viscous and elastic components of the triceps. However, a recent systematic review highlighted the lack of scientific evidence concerning the effect of stretching to treat or prevent contracture (29).

When stretching performed intensively during 2 months fails to improve ankle mobility or when the ankle ROM limitation exceeds 20° of fixed plantar flexion, we recommend a lengthening surgical procedure. If only the gastrocnemius muscle is shortened, we perform a lateral and medial gastrocnemius aponevrotomy followed by a cast placed with a 10° ankle dorsal flexion for 3 weeks. If both gastrocnemius and soleus muscles are shortened, we perform an Achilles tendon lengthening followed by a cast placed with a 10° ankle dorsal flexion for 6 weeks. The gait is authorized with cast. In order to anticipate a painful claw toe that can be worsened by the equinus correction, which stretches the flexor digitorum muscles and tendon, a flexor hallucis longus and flexor digitorum longus lengthening is systematically performed during the same procedure at the same operative site. Keenan et al. estimated that residual claw toe, causing pain in 72% and callosities on the dorsum of the toes in 59% of cases, is frequently observed after Achilles tendon lengthening despite release of the flexor hallucis longus tendon (30). If the claw toe is already severe and painful before triceps lengthening, we recommend additional digitorum flexor tendon lengthening at the level of the internal malleolus or at the base of the toes. Lengthening the Achilles tendon, although necessary to correct the equinus deformity, weakens the triceps surae, resulting in insufficient stabilization of the tibia during the second part of the stance phase, which may necessitate the use of an AFO for propulsion. If at the pre-operative assessment the triceps surae muscle is weak, the addition of a flexor hallucis longus and flexor digitorum longus transfer to the os calcis may improve the strength of the gastrocnemius-soleus muscle complex and allow a greater increase in function and less reliance on orthotics. More rarely, a triple arthrodesis is proposed for severe rigid bony deformities. A systematic review of case series studies suggested that orthopaedic surgical correction of SEVF is a safe and effective procedure in terms of re-obtaining a balanced foot position, improving walking capacity and reducing the need for orthotic use (8).

Dorsiflexor muscles weakness

The lack of activation of the dorsiflexor muscles (tibialis anterior and extensor digitorum muscles) in the swing phase of gait is responsible for an equinus in the swing phase of gait also called “drop foot”.

Equinus in the swing phase of gait related to dorsiflexor muscles weakness is treated by AFO or functional electrical stimulation (FES). AFO assists foot clearance during the swing phase, improves the mode of initial contact, prevents ankle inversion injuries, and helps in advancing the body during midstance. Recent systematic reviews conclude that AFO can improve ankle and knee kinematics, kinetics, energy cost of walking and balance (31). However, different AFO are available, making appropriate choice challenging.

We recommend the use of a dynamic AFO for active patients without triceps weakness or spasticity and a carbon AFO for patients with an associated lack of propulsion at the push-off phase of gait in relation to triceps weakness.

Electrical stimulation (ES) is commonly used to improve muscle strength. ES can also be applied during gait (so called functional electrical stimulation; FES) by stimulating the common peroneal nerve to activate the tibialis anterior muscle during the swing phase of gait. A recent systematic review supports the ability of transcutaneous FES to improve gait kinematics and gait speed and to reduce energy cost (32). However, the improvement obtained with FES and AFO appears to be identical (33). Last, in selected patients, an implanted FES device may be proposed in order to suppress the external device of AFO or transcutaneous FES (34). Less frequently, the anterior transfer of the long toe flexors, using them as dorsiflexor tendons or for tenodesis, is proposed (35).

A triceps spastic co-contraction may also induce an equinus in the swing phase of gait, mimicking a lack of activation of the dorsiflexor muscles. The DNB will improve equinus in the swing phase due to the triceps spasticity, but not in case of dorsiflexor muscles weakness. A triceps surae – Achilles tendon complex shortening may also induce equinus in the swing phase. However, in such cases, equinus will also be present in the stance phase. Lastly, one of the most challenging difficulties is to determine the presence of dorsiflexor muscles weakness in addition to a triceps shortening, as it will be necessary to compensate (by means of AFO or FES) the dorsiflexor weakness after Achilles tendon lengthening.

Tibialis anterior and peroneus muscles imbalance

The imbalance between activation of the tibialis anterior, extensor hallucis longus, extensor digitorum muscles and peroneus muscles (usually weaker) in the swing phase of gait is responsible for varus in the swing phase of gait, which places the foot in an unstable position at first foot contact, leading to ankle sprain and falls. As for the equinus related to dorsiflexor muscles weakness, AFO and FES are recommended. However, imbalance in the swing phase of gait can also be treated by tendon transfer, such as the split anterior tibial tendon transfer (SPLATT) procedure. The SPLATT procedure consists in dividing the tendon of the tibialis anterior muscle, maintaining half of the tendon on the medial aspect of the foot and transferring the other half to the lateral side of the foot, usually on the peroneus tendons, providing active dorsiflexion without varus of the forefoot. The hallux hyperextension caused by the spasticity of the extensor hallucis longus (EHL) muscle, which contributes to the varus deformity, may be transferred to the mid-dorsum of the foot, often in combination with a SPLATT procedure. An alternative to the SPLATT procedure is the Bardot procedure, which associates the fixation of the peroneus brevis distal tendon onto the tibialis anterior with Achilles and flexor digitorum tendons lengthening. Lastly, a posterior tibial tendon transfer from the navicular bone to the lateral cuneiform bone (Watkin's procedure) is described without iatrogenic valgus flatfoot (36). Several uncontrolled studies using different transfer techniques (SPLATT, entire tibialis anterior tendon, Bardot) frequently associated with other surgical procedures (Achilles lengthening, toe flexors lengthening, talonavicular arthrodesis and neurotomy) tend to demonstrate a varus correction leading to an improvement in ambulatory status, a reduction in requirement for walking aids and an increase in gait speed (37). The rate of surgical complications, including wound infection and pull-out of the tendon that required reoperation, appears to be low.

SPASTIC EQUINOVARUS FOOT IS PART OF A DIFFUSE SPASTICITY

In some cases, spasticity is diffuse and present distally, as well as proximally, at the hip and knee muscles, leading to a lack of hip and/or knee flexion, in addition to SEVF making intrathecal baclofen therapy (ITB) an option. First described by Penn, intrathecal administration of baclofen circumvents the blood-brain barrier, leading to a reduction in spasticity with a minimized risk of dose-related adverse events. Prior to pump implantation, candidates have to undergo a screening trial of intrathecal baclofen bolus or continuous (by means of external pump) injection in order to assess the benefit of the therapy. Numerous studies have demonstrated the efficiency of intrathecal baclofen (ITB) therapy in spastic patients with spinal cord injury, multiple sclerosis, traumatic brain injury and cerebral palsy. In contrast, only a few studies have concerned stroke patients, and none was focused specifically on the SEVF. A randomized, double-blind placebo-controlled crossover study showed spasticity decrease in both upper and lower limbs after continuous administration of baclofen (38). Uncontrolled studies demonstrated a decrease in triceps spasticity with slight improvements in walking speed, functional autonomy and quality of life (39). However, patients may experience functional deterioration if ITB weakens their paretic side such that the antigravity pattern they use for ambulation is suppressed (40). ITB complications are frequent and

are usually related to catheter dysfunction or surgical procedure complications.

CONCLUSION

SEVF is one of the most common deformities observed among hemiplegic patients. Selective DNB with anaesthetics helps clinicians to determine the respective role of the different spastic muscles and/or triceps surae – Achilles tendon shortening and/or weakness and/or imbalance of dorsiflexor muscles and to enhance the therapeutic decision. Alongside a rehabilitation programme and a patient-goal-oriented approach, treatments must be decided by an interdisciplinary medical and surgical spasticity team. Triceps spasticity is treated by BoNT-A and neurotomy, triceps surae – Achilles tendon complex shortening by stretching and tendon lengthening, and dorsiflexor muscles weakness and/or imbalance by AFO, FES and tendon transfer (SPLATT) procedure. In case of diffuse spasticity, ITB can be an option. Combined procedures are frequent.

In order to define an international consensus this monocentric proposed guidance should be taken as the basis for further research, including evidence from the practice of experienced teams from different countries.

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