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EDUCATIONAL REVIEW

HOW TO CLINICALLY ASSESS AND TREAT MUSCLE OVERACTIVITY IN SPASTIC PARESIS*

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Objective: **This educational paper aims to describe, in adult patients, the different aspects of muscle overactivity after a central nervous system lesion, including spasticity, spastic dystonia and spastic co-contraction, the assessment of their symptoms and consequences, and therapeutic options.**

Discussion and Conclusion: **Clinical evaluation involves the assessment of passive range of motion, angle of catch or clonus, active range of motion, rapid alternating movements and functional consequences. A number of scales have been developed to assess patients with spastic paresis, involving both patient and caregivers. Not all persons with spasticity require treatment, which is considered only when muscle overactivity is disabling or problematic. A list of personal objectives may be proposed for each patient, which will drive assessment and treatment. Prior to treatment the patient must be informed of the intended benefits and possible adverse events. Clinical evaluation may be supported by the use of transient neuromuscular blocks and/or instrumental analysis. Physical therapies usually represent the mainstay of treatment. Self-rehabilitation with stretching and active exercises, intramuscular injections of botulinum toxin, alcohol or phenol injections, oral or intrathecal drugs, and surgery comprise the treatment options available to the clinician. Follow-up must be scheduled in order to assess the benefits of treatment and possible adverse events.**

Key words: spastic paresis; muscle overactivity; assessment; treatment.

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INTRODUCTION

The aim of this educational paper is to set out the essential points of management of spasticity in patients with spastic paresis, for Physical and Rehabilitation Medicine specialists and other physicians. Many central nervous system (CNS) acquired lesions, traumatic, vascular, tumoural or infectious, lead to spastic paresis, i.e. paresis associated with stretch-sensitive muscle overactivity. "Spasticity" is a term that is often used beyond its definition, to refer to various types of muscle overactivity. The term "muscle overactivity" is more appropriate and should be used preferentially. This article describes the different aspects of muscle overactivity after a CNS lesion, the means of clinical evaluation of its consequences, the different therapeutic options, the usual need for a multidisciplinary approach, and the need for a scheduled follow-up.

This paper has 4 main educational objectives: (*i*) to explain the pathophysiology of spastic paresis and the definition of the different types of spastic muscle overactivity; (*ii*) to describe the means of assessing spastic paresis and the consequences of muscle overactivity; (*iii*) to describe how to disentangle the roles played by various types of muscle overactivity, musculoskeletal complications and other neurological disorders potentially associated with spastic paresis; and (*iv*) to describe the different treatments available and their value.

PATHOPHYSIOLOGY OF SPASTIC PARESIS

Three fundamental phenomena occur after a lesion to the central motor pathways assigned to motor command execution.

Paresis

After an acquired brain injury causing a lesion to the corticospinal pathways, the central execution of motor command is disrupted, resulting in immediate paresis (1). Paresis is defined as the quantitative lack of command directed to agonist muscles when attempting to generate force or movement. This lack of command can occur through an insufficient number of motor units synchronously recruited and/or an insufficient discharge frequency of the recruited motor units.

Soft tissue contracture and changes in contractile muscle properties

The relative immobilization of the paretic body parts leaves some of the muscles and their surrounding soft tissues immobilized in a shortened position. Plasticity subsequently causes the soft tissue to shorten and adapt to its new length. This phenomenon leads to soft tissue contracture, which is defined by: (*i*) physical shortening; and (*ii*) reduction in extensibility of soft

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tissue, including muscles, tendons, ligaments, joint capsules, skin, vessels and nerves (1). This process begins with gene changes and transcriptional events, leading to protein synthesis reduction a few hours after the onset of immobilization, and only intensifies in the days, weeks and months that follow if insufficient preventative treatment is implemented (2, 3). In addition, changes in contractile muscle properties (particularly slow-to-fast) are observed depending on the initial motor unit type and duration of disuse (3).

Muscle overactivity

Lesion- and activity-dependent adaptive changes subsequently occur within the higher centres and the spinal cord (2). Brainstem descending pathways (rubro-, tecto-, reticulo-, vestibulospinal pathways) are increasingly recruited (with potential disinhibition due to frontal disconnections in brain lesions) to take over some of the execution of motor command following disruption of the corticospinal pathway. Most of these brainstem descending pathways tend to be constantly active, thus generating permanent muscle activity. At the spinal cord level, lower motor neurons are now deprived of their regular descending excitation from the corticospinal pathway and release growth factors locally (4). These tend to promote local sprouting from neighbouring interneurons, thus creating conditions for the formation of new abnormal synapses between these interneurons and the somatic membrane of the deprived motor neurons, thus leading to the creation of new abnormal or exaggerated reflex pathways (5).

Spasticity. Spasticity is the most commonly recognized manifestation among these gradually occurring changes. The definition of spasticity has been simplified as an *increase in velocity-dependent stretch reflexes* (2, 6), which is clinically manifested at rest by excessive responses to muscle stretch or tendon taps. Stretch-induced contraction at rest occurs at a lower threshold, and with an increased amplitude in patients with spastic paresis compared with normal subjects. Thus, the primary triggering factor for spasticity is phasic stretch, and this phenomenon is detected and measured at rest. Spasticity is unlikely to be a disabling form of muscle overactivity, except when clonus interferes with posture or movement. Clonus can be triggered by passive movement during nursing, dressing or washing, or simply during attempts to maintain a relaxed seated position, causing discomfort or difficulty in keeping the feet on the footplates of a wheelchair. Clonus can also be triggered by active movements, with a potentially negative impact on prehension or walking.

Spastic dystonia. The term "spastic dystonia" was coined by Denny-Brown to represent tonic chronic muscle activity that is present in the context of spasticity (2, 7). Thus, spastic dystonia is spontaneous overactivity at rest for which there is no primary triggering factor. It is the type of muscle overactivity that is most easily recognizable when looking only at patients with spastic paresis, as spastic dystonia deforms joints and body postures and is a major cause of disfigurement and social handicap in these patients.

In hemiparetic patients after stroke or traumatic brain injury various types of spastic dystonia may be observed, leading to abnormal postures. For example, in the upper limb the shoulder can stay internally rotated and adducted with a flexed and pronated elbow and flexed wrist and fingers. In the lower limb, spastic dystonia may often involve the plantar flexors and cause equinovarus deformity and/or toe flexors, causing toe clawing that can be painful and disabling during walking.

Spastic co-contraction. Spastic co-contraction is defined as an "unwanted, excessive, level of antagonistic muscle activity during voluntary command on an agonist muscle, which is aggravated by tonic stretch in the co-contracting muscle" (2, 8). Spastic co-contraction in patients with spastic paresis is a descending phenomenon, most probably due to misdirection of the supraspinal drive. It may be facilitated by increased recurrent inhibition, causing loss of reciprocal inhibition during voluntary command (2, 9). Thus, the primary triggering factor for spastic co-contraction is voluntary command on an agonist, and spastic co-contraction is detected and measured during voluntary effort.

In patients with good or fairly good motor control, spastic co-contraction is likely to be the most disabling form of muscle overactivity in spastic paresis, as it impedes generation of force or movement: range of active motion is diminished and rapid alternating movements are slowed. In the upper limb spastic co-contraction may often be observed in the flexors during attempts at elbow, wrist or finger extension. Thus, reaching becomes difficult as well as opening the hand to grab an object or drop it. In the lower limb during the swing phase of walking, spastic co-contraction of the hip extensors may restrict active hip flexion; in the knee flexors and extensors it may restrict knee motion; similarly, spastic co-contraction of the ankle plantar flexors may restrict active dorsiflexion in the swing phase (10).

Other types of muscle overactivity. Other types of muscle overactivity exist, which have not been shown to be stretch-sensitive. Pathological extra-segmental co-contraction (also known, depending on its pattern, as syncinesia, associated reactions, choreic or athetotic movements) is an unwanted, abnormal level of activity in muscles that are distant (at a different segmental level) from the agonist involved in the voluntary command (2). Excessive cutaneous or nociceptive responses and other forms of inappropriate muscle recruitment during yawning, breathing or coughing are also common in spastic paresis.

Factors causing variation. The time of day and the position of other joints, including the neck, may impact on the different types of stretch-sensitive muscle overactivity. Thus, when assessing muscle overactivity in spastic paresis, the clinician must make every effort to repeat assessments at a similar time of the day and in the same body position. Other common factors in variation are stress level, external temperature and any nociceptive factor. The mere measurement of spasticity in a patient at rest is far from fully reflects the impact of the condition during movement.

CLINICAL EVALUATION

Whatever the different aspects and terms in use to describe muscle overactivity, what is important is to determine its real

impact, and this requires a careful clinical evaluation. This assessment can then be used to argue the need for a treatment and will be the basis of the required follow-up.

Passive range of motion

For each movement evaluated, the clinician stretches the corresponding muscles and joints at a very slow speed (called V1, slow velocity) that is kept below the threshold for eliciting a stretch reflex. The angle at which soft tissue offers a maximum resistance is defined as the passive range of motion for that joint. A common and important clinical issue is to distinguish retraction from severe dystonia, which may require transient motor blocks (see below).

Angle of catch or clonus and spasticity grade

For each movement evaluated, the clinician should stretch the corresponding muscles and joints at the fastest speed possible for the examiner (V3, fast velocity). According to the Tardieu scale, two parameters are then determined: the *angle* at which catch or clonus occurs represents the threshold to elicit the reflex, and the type of muscle reaction occurring at that angle defines the spasticity *grade* (11, 12).

Active range of motion

For each passive movement evaluated, the clinician asks the patient to perform an active movement as far as possible along the range, until the active force produced by the agonist is balanced by the passive resistance from the stretched structures together with the spastic co-contraction in the antagonist. This third angle is the active range of motion.

Rapid alternating movement frequency

The patient performs the same active movements over the maximal range as measured above then returns to the starting position again, as many times as possible in a limited period of time (e.g. 15 s). The number of movements performed indicates the ability of the subject to repeat fast active movements (13), which is required for some everyday activities, but moreover is a good way, in our experience, to reveal spastic dystonia or co-contraction during a simple examination.

Function: consequences of paresis, contracture and muscle overactivity

Individualized, patient specific outcomes may be considered, using a goal-setting process. To describe the tools that can be used to assess function, including the consequences of muscle overactivity, patients can be divided into two approximate main categories according to the magnitude of impairment.

Assessing function in patients with severe paresis. Contractures and/or spastic dystonia may lead to abnormal joint positions, causing disfigurement, pain, discomfort, impairment in washing, dressing, urinary function, sitting position, walking impairment with potential consequences on perineal hygiene or a risk of bed sores. Clonus may also bear functional impact, assessed on: spontaneous position (sitting or standing), passive motion, active motion (reaching, walking).

Scales intended for caregivers or patients evaluate difficulties during nursing, discomfort or disfigurement. A tool has been proposed by Bhakta et al. (14); the patient's disability (PD) and the carer burden rating scale (CBRS). These scales have been developed to measure upper limb activity limitation in patients with no active function in the arm. The PD consists of 8 items, such as cleaning the palm, cutting fingernails, putting the paretic arm through sleeves, etc. The CBRS consists of 4 items: cleaning the palm, cutting the fingernails, dressing and cleaning under the armpit. With a similar approach, Brashear et al. (15) proposed the Disability Assessment Scale, which assesses 4 domains of potential disability: hygiene, dressing, limb position, and pain. The patient, together with the physiatrist, selects one of the 4 domains as the principal target of treatment.

However, when motor control is strongly impaired, overactivity in some muscles may be functionally useful. In the lower limb, patients with severe paresis may sometimes stand using quadriceps overactivity; in the upper limb, elbow flexor overactivity may allow the patient to carry a purse or bag. The patient must be evaluated keeping in mind the potential usefulness of muscle overactivity in some areas before a decision is reached on treatment.

Assessing function in patients with fairly good motor control. Paresis, soft tissue contracture, spastic co-contraction, dystonia, and clonus may interfere with active movement. Evaluating the real impact of overactivity on active movement is required to determine the need for and type of treatment. Different levels of assessment can be used, measuring motor control, limb function or global independence.

Generic personal functional scales, such as the Barthel Index after stroke, Expanded Disability Status Scale in multiple sclerosis or the Functional Independence Measure, may be useful in clinical practice. However, as Wade et al. (16) noted, these activities of daily living (ADL) scales measure the ability to perform tasks without necessarily involving the affected arm. They may therefore rate mainly adaptive strategies learned by patients, and are not designed to measure the consequences of overactivity and the effects of treatment.

Emphasis may be put specifically on the assessment of the motor impairment of the affected limb (17, 18), which involves comprehensive testing of active abilities of the paretic limbs. The limitations of extensive impairment scores, such as the Fugl-Meyer, include time consumption and the absence of real-life tasks tested. Its sensitivity may be insufficient to observe change after treatment with botulinum toxin (BTX) (19). *Upper limb function* has been assessed using different scales (e.g. Rivermead Motor Assessment, Frenchay arm test, Motor Activity Log) and by motor impairment tests, such as the box-and-block test or the nine-hole peg test. However, these standard scales for upper limb function are not usually sensitive enough to demonstrate efficacy of treatment, and contradictory results have been published (20–23). With the purpose of improving sensitivity, reliability and validity in the functional assessment of the upper limb, a modified Frenchay Arm Test, termed the Modified Frenchay Scale has been proposed (24). This tool involves 10 real-life uni- and bi-manual tasks, which are videotaped and rated on a visual analogue scale.

Many authors have used a global assessment of the result, proposed to patients, caregivers or investigators in research studies. While negative in some studies (22), in most this global assessment of benefit showed positive effects of BTX treatment (15, 19–21, 25). Such a global assessment may be based on previous goals for the treatment, previously specified with the patient.

The main function of the lower limb is walking. Clinical functional scales, such as the Functional Ambulation Classification, do not have sufficient sensibility. To rate actual function in the lower limb, walking tests can be performed (10 metres walking, 2-min or 6-min endurance test) as well as tests of stair-climbing performance and walking on uneven ground. Walking speed and step length can be measured, as well as the physiological cost index, which is the speed divided by the difference between the heart rate before and after the effort (26). The quality of the movement is difficult to assess clinically and can be usefully completed by instrumental analysis (notably kinematic analysis) especially when surgery is considered.

Other evaluations

Combined assessment of soft tissue contracture and spastic dystonia. Tools such as the Ashworth and the Modified Ashworth scales have been widely used in clinical trials under the initial assumption that they measure spasticity (27, 28). However, it is now established that these instruments evaluate a combination of soft tissue contracture and spastic dystonia, in addition to spasticity itself (12, 29).

Assessment of pain. Pain directly related to muscle overactivity is possible, due to prolonged muscle contractions, but also to tendon lesions and abnormal postures. Pain may be an important component of the patient's difficulties. Apart from scales in which pain assessment is included, as in the Disability Assessment Scale (15), pain can be assessed non-specifically using visual analogue scales when it is deemed necessary.

TREATMENT

Indications for treatment

The treatment of muscle overactivity may be considered when the condition is disabling. Whatever its aetiology, muscle overactivity usually has a negative impact on motor command, and this justifies treatment of the symptom itself, i.e. independent of the aetiological context, as a function of the patient's neurological disorders (30, 31). However, not all spastic patients necessarily require treatment for muscle overactivity. Treating muscle overactivity must only be considered after rigorous clinical analysis, in order to determine the severity of the condition, its true consequences and distribution. This careful assessment often requires a multidisciplinary approach, varying from patient to patient, including physician, physical therapist, occupational therapist, nurse or caregiver. A list of personal, separately measurable objectives may be proposed for each patient, to drive therapeutic strategy. The follow-up must be scheduled to document benefits and possible adverse events.

Muscle overactivity is probably the only motor disorder that can benefit from a drug treatment, but it is not the only motor disturbance in spastic paresis. Paresis can be treated by motor training. Soft tissue shortening can be treated by aggressive stretch programmes. The following 3 questions must be addressed:

- Is muscle overactivity problematic and, if so, in what respects? Only a detailed analysis of the impact of overactivity in all its passive and active functional aspects enables the clinician to decide on the appropriateness of a given treatment and to set reasonable patient objectives.
- Is muscle overactivity the main cause of the disability or only one of the causes? In the latter case, which components are involved? It is important to specify quality of motor control and hypoesthesia. If muscle overactivity is considered to be an important culprit for the motor deficiency, then its treatment is likely to be helpful to the patient (32).
- Is the problematic muscle overactivity limited to one muscle group or is it more widely spread? The correct treatment depends on the answer.

Transient motor blocks

Transient neuromuscular blocks by intramuscular injections (motor point injections) or by perineural injections (trunk nerve anaesthesia) using local anaesthetic drugs may be performed prior to treatment in order to address the following issues (28): (*i*) How severe is muscle contracture? If it is severe, it is obvious that drug treatment of overactivity alone or physiotherapy cannot aim to restore the normal range of motion; (*ii*) How could the patient manage functionally (walking, reaching, grasping) after local weakening treatment?; (*iii*) Is there any active command available in the antagonist muscles that can be trained or what will be unwanted? This information may be fundamental when surgery is considered (33, 34) but the motor block has a transient effect, too short to allow the patient to adjust to a new motor pattern, thus the latter two issues may be incompletely solved.

In the upper limb, the following blocks can be carried out easily: pectoral nerve loop to examine the role of *pectoralis* major and minor in the adduction and medial rotation; musculocutaneus nerve to release the elbow flexors except for *brachioradialis*; ulnar and median nerves to release wrist and finger flexors. In the lower limb, the obturator nerve can be blocked to release hip adductors, posterior *tibialis* nerve and its branches can be separately blocked to examine the roles of *gastrocnemius*, *soleus*, *tibialis* posterior and toe flexors.

Anaesthetic drugs available include lidocaine, ropivacaine and bupivacaine. Apart from potential allergic reactions, the main risks are toxic dose-related effects, such as hypotension, bradycardia and cardiac arrest. The clinician must therefore have emergency equipment available. These complications are fortunately rare.

Treatment of nociceptive triggering factors

Potential nociceptive triggering factors must be treated before treating muscle overactivity, bearing in mind that the patient

may be unaware of them because of hypoesthetic areas. Common factors are: bed sores, urinary infection or lithiasis, constipation, haemorrhoids, fractures or ingrown toenails. Muscle overactivity is sometimes associated with these factors, especially with sores and pain, and treatment of one may help the other. For example, intrathecal baclofen infusion via a transient catheter may be help with large bedsores in spastic paraparesis, leading to a reduction in abnormal posture.

Treatment options

The therapeutic programme may combine, in various proportions, physical therapy, occupational therapy, self-rehabilitation, the use of orthoses and assistive devices, drug treatment, orthopaedic surgery and neurosurgery.

Physical therapies. Physiotherapy is the basic treatment for all patients with spasticity (35, 36). It may help to limit muscle contractures and reduce overactivity for a short period. Physiotherapy is essential to help patients to adapt to changes, and sometimes drug-induced reduction in overactivity can allow a new rehabilitation programme to be started. In all cases, physiotherapy must be considered as complementary to drugs and surgery. Maintained stretch must remain a mainstay in a physiotherapy session, as its efficacy has been largely demonstrated (37). Heat or cold applications have also been proposed, as well as positional interventions, such as inhibitory casting. Electrical stimulation has been shown to allow spasticity reduction in muscles antagonist to the stimulated muscles (38). One interesting use of electrical stimulation is the stimulation of hand and fingers extensors during prehension training, mixing overactive flexor inhibition with extensor activation (39). Finally, one of the most important parts of overactivity treatment and contracture prevention is self-rehabilitation with stretching postures, active exercises taught to patients and caregivers and sometimes orthoses.

Drugs. This section refers mainly to the recommendations of France's drug authority (Agence Francaise de Securite des Produits de Sante) (30, 31).

Botulinum toxin. A double-blind study comparing BTX, tizanidine and placebo after stroke or traumatic brain injury has recently established BTX type A as the first-line treatment of multifocal muscle overactivity, for both better efficacy and better tolerance than systemic treatment (40). The efficacy of BTX type A has been documented in self-care improvement (washing and dressing) (15) and active movements in the leg with gait improvement (41, 42). No improvement in active arm function has been demonstrated to date except using kinematic analysis (43). An effect on pain reduction has also been demonstrated. There are currently 4 forms of BTX available in Europe: 3 type A (Botox®, Allergan, Dysport®, Ipsen-Pharma, Xeomin®, Merz) and 1 type B (Neurobloc®, Elan-Pharma), and it must be kept in mind that the units of these toxins are different, specific for each brand. We strongly recommend using electrical stimulation to localize injection sites, as using anatomical markers alone may lead to inaccurate targeting (30, 31, 44). There is interest in the use of ultrasound guidance to help to identify muscles, particularly in children, but this technique has not been evaluated against electrical stimulation for its efficiency.

No immediate post-injection complications have been reported (except for slight pain at the injection site). Patient and caregivers must be warned of a low risk of adverse effects that may occur during the first 3 weeks after each injection (swallowing disorders and botulism-like syndrome) and should be encouraged to consult if any doubt.

The results of the therapy may be assessed 1–6 weeks after the injection on the basis of the personalized objectives decided before treatment. Repeat injections are often justified due to the transient effect of the toxin, but a long-lasting effect is possible. No repeated treatment must be conducted without accurate assessment. When needed, a minimum delay of 2–3 months between injections is usually recommended, although no scientific evidence backs up any specific delay (30, 31). Each subsequent injection should be followed by an evaluation of the achievement of pre-therapeutic defined objectives, as well as tolerance, with a review of the dose and treated muscles. Repeat injections can be continued as long as beneficial effects are observed.

Alcohol and phenol. Alcohol or phenol have often been injected perineurally. This treatment should be used preferably in nerves with a low sensory activity and a high motor predominance (obturator, musculocutaneous, etc.). Intramuscular (motor point) injections may also be performed with the potential disadvantage of being painful at injection, but the advantage of avoiding the risk of post-injection dysesthaesia. This focal treatment by chemical neurolysis is usually not used as a first-line therapy, except in the case of particularly problematic overactivity affecting a large number of areas. Alcohol or phenol can be used for large muscles by chemical block of a whole nerve trunk (adductor muscles for example), while BTX is used for some other muscles because of the maximum permitted doses of BTX (30). Injection is performed using electrical stimulation. A transient motor block may be performed initially in order to indicate whether chemical neurolysis might be effective. The benefits of alcohol or phenol treatment must be weighed up initially, relative to those of surgery (alcohol or phenol induce tissue fibrosis, which may render subsequent surgeries difficult).

Oral drugs. Baclofen and tizanidine have reduced tone on Ashworth scores with a dose-dependent response in some studies, but there is no evidence on reducing the functional impact of spasticity (45, 46). Tizanidine has been shown to be no more effective than placebo in reducing tone and improving perceived function in the upper limb (40). Poor tolerance, with insidious adverse events, such as sedation, fatigability, drowsiness and reduction in seizure threshold, have led these to be considered as second-line therapies in stroke patients (30, 31). Introduction, dose adjustment and withdrawal must be performed gradually. All long-term treatments must be reappraised (e.g. for therapeutic windows, and dose changes) with a periodicity that depends on condition and time since onset. Other drugs, such as dantrolene, have no sufficient evidence in the literature to recommend their use. A combination of oral therapies is not recommended (30, 31).

Intrathecal baclofen. Intrathecal baclofen (ITB) is a long-term treatment with continuous, intra-spinal administration via an implanted pump that reduces spasticity, especially in spinal injury patients and in multiple sclerosis (47, 48). ITB has been used in patients whose leg muscle overactivity was considered diffuse. It should be reserved for severely disabled patients whose muscle overactivity interferes with posture, nursing, rest, personal independence or causes pain (30). Grave (potentially lethal) complications have occurred with this treatment and detailed information on the expected benefits and risks, notably in terms of potential loss of motor function (which can be reversed at withdrawal) must be provided to the patients and his or her caregiver and family (49).

Several assessments are usually performed before definitive pump implantation by after simple injection via lumbar puncture or via a temporary access device. Efficacy may be evaluated in the following 3–4 h. The usually recommended first test dose is 50 µg in adults, with a maximum dose of 150 µg that should be reached after 3 days. Caution should be exercised particularly with respect to the risk of overdosing, paying particular attention to vigilance and respiratory disorders. Thus, monitoring of vital signs by a specialized team must be performed during the 4 h following the test. Then, if the treatment is well-tolerated and deemed effective, the team may decide to implant the pump, and to monitor and follow up the treatment. It is critical to ensure maintenance, notably to detect hazards related to the procedure (displacement of the catheter, infection, etc.) and to prevent the occurrence of a withdrawal syndrome.

Surgery. Surgery may play an important role in the treatment of chronic muscle overactivity, but it is not the first-line treatment. Because of its potential adverse events and its remaining effects, surgical techniques must be strictly adapted to different goals: hygiene, standing, transferring, walking, use of assistive devices (e.g. shoes, orthoses, canes, wheelchair). It involves neurosurgery and orthopaedic surgery. Surgical procedures may include one or more of techniques described below.

Peripheral neurotomy (selective or hyperselective) consists in partial and segmental resection, involving motor collateral branches to overactive muscles. The goal of selective peripheral neurotomy is to balance agonists and antagonists without abolishing "useful" tone. For the lower limb, common targets are collateral branches of the posterior tibial nerves (e.g. ankle clonus, equinus, inversion of the foot) (50) and obturator nerves. In the upper limb, good results have been reported with neurotomy of the musculo-cutaneous, median and ulnar nerves (48). Other nerve surgeries, such as rhizotomies, have also been used but have potential complications.

Musculoskeletal surgery is treatment of the consequences of muscle overactivity, contracture and joint deformities, performed on the muscle or the tendon itself. Tendon transfers (e.g. *tibialis* anterior) or lengthening are conservative treatments commonly proposed (51). Tenotomy may be considered in the case of muscle contracture without active functional objectives. Osteotomies are sometimes proposed to correct hip displacements and foot deformities. Arthrodesis may be the only solution to stabilize joints, notably ankle and foot joints in case of severe paresis associated with strong muscle overactivity and hypoaesthesia.

CONCLUSION

Treatment of muscle overactivity should be considered only after careful evaluation of its consequences, often using a multidisciplinary approach, in terms of active and passive function, pain, discomfort and other symptoms. Not all patients with spasticity require treatment. Different scales may be used according to the goals, but none of them replaces personal objectives elaborated for and with each patient. The indication for and the choice of treatment among physical therapy, drug and surgery usually requires a multidisciplinary approach. Regardless of their value, focal treatments by chemical weakening should only be considered as an adjunct to physical therapies. Follow-up must be scheduled in order to assess the benefits or possible adverse events of the treatment.

Learning objectives

To understand the pathophysiology of spastic paresis and the definition of the different types of spastic muscle overactivity: spasticity, spastic dystonia, and spastic co-contractions.

To know how to assess spastic paresis and to understand the consequences of muscle overactivity.

To be able to distinguish between the assessment of muscle overactivity and the assessment of its consequences.

To understand the different scales, their usefulness and their limits.

To be able to assess with the patient and in a multidisciplinary approach the real consequences of muscle overactivity in activities and participation.

To know how to disentangle the roles played by various types of muscle overactivity, musculoskeletal complications and other neurological disorders potentially associated with spastic paresis.

To understand the different types of treatment available and their value.

To be able to assess the benefits and possible adverse events of the different treatments.

To be able to determine personal objectives for each patient before commencing treatment.

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