drive) at least in part. We define this stretch-sensitive, misdirected descending command as spastic cocontraction. While spastic cocontraction is really the main antagonistic mechanism impeding movement, in particular leg movements during gait, muscle overactivity is often assumed to be represented by mere measurements of resistance to passive movement. A five-step clinical assessment, including a quantitative assessment of active range of motion, may better serve the clinician to reflect the amount of cocontraction.

Another critical phenomenon is stretch-sensitive paresis, which is the aggravation of paresis of agonist command by the stretched position of the antagonist. The physiological upregulations of spastic cocontraction and stretch-sensitive paresis probably involve neuronal rarefaction in agonist command, hypo-excitability of the remaining upper motor neurons, hypereexcitability of the lower motor neurons subserving the more shortened muscles and reduction or reversal into facilitation of Ia and groups II reciprocal inhibition. Considering its determinants (agonist effort and muscle position), treatment of spastic cocontraction and stretch-sensitive paresis – and management of deforming spastic paresis in general – should logically involve a combination of:

– prolonged stretching postures for the more overactive and shortened antagonists, potentially in association with focal weakening agents such as botulinum toxin;
– intensive motor training (e.g. non assisted maximal amplitude rapid alternating movements and task-related exercises) of the less overactive muscles.

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Methods. Forty-five patients suffering from LAS with either paraparesia (n = 10) or tetraparesia (n = 25) were referred to the specialized consultation in Physical Rehabilitation Medicine department in order to treat their spasticity. Some patients walked independently (n = 4), some required human or technical help for deambulation (n = 38), some were unable to walk (n = 3). Their medical charts were retrospectively analyzed, to determine the efficacy, tolerance and side-effects of botulinum toxin therapy.

Results.– Thirty-nine patients received botulinum toxin injections in their inferior members, in order to ameliorate ambulation (n = 36), sitting or nursing (n = 3). In 16 patients, a motor block of rectus femoris muscle was made. Five patients out of them were considered as ineligible for botulinum toxin therapy. Twenty-one patients were satisfied with the treatment that was continued. Eighteen patients stopped botulinum toxin therapy after the first injection either because of lack of improvement either by diseased progression. One patient experienced a transient respiratory degradation after treatment.

Discussion and conclusion.– Botulinum toxin therapy provided clinical amelioration of spasticity in more than a half of our patients. One patient had a transient respiratory degradation. To our knowledge, no study has been reported about the efficacy and tolerance of toxin therapy in spasticity management of LAS patients. This treatment is used to treat hypersialorrhea, without severe side effect [2]. Nevertheless, the treatment should be started with low doses. As LAS patients often suffer from severe motor loss, motor block of the nerve of rectus femoris muscle should be perform before of botulinum toxin injections in this muscle.

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Evaluation of a treatment of focused spasticity with toxin botulinum

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Keywords: Focused spasticity; Clinical evaluation; Botulinum toxin; Vas satisfaction

Introduction.– Botulinum toxin is the first-line therapy in the management of focal spasticity.

Through this work we want to evaluate our modest experience with the use of this therapeutic method.

Materials and methods.– Fifty-two patients with focused spasticity due to various pathologies were treated with botulinum toxin in the year 2011. A pre therapeutic assessment and three weeks after injection of predetermined muscles was performed taking into account the following parameters:

– the pain intensity assessed by visual analog scale;
– spasticity measured by Ashworth scale;
– the range of motion measured by goniometry;