Spasticity is a common and debilitating condition associated with various neurologic disorders such as multiple sclerosis, stroke, cerebral palsy, spinal cord, and brain injury. While the incidence of spasticity is not known with certainty, it is likely that it affects over half a million people in the United States alone, and over 12 million worldwide.\(^1\) With the increasing number of available options in managing spasticity and most notably, the considerable increase over the last few years in the use of botulinum toxin type A (BTX-A) as a safe and effective option in spasticity management,\(^2\) there is an urgent need for a national consensus and guidelines for spasticity management in Arab countries. A group of experts in rehabilitation medicine and neurological sciences representing different Arab countries met in Cairo in May 2005 to evaluate the developing experience with botulinum toxin in the treatment of various clinical disorders characterized by spasticity, and to develop a consensus statement to guide the use of BTX-A in the region. The practice of developing a regional consensus statement for the use of BTX-A in spasticity management has many precedents in North America (National Institutes of Health consensus statement) and Europe (European consensus statement).\(^9,10\) In this paper, we provide an overview of spasticity, its current management options, the history of BTX-A use, and describe the consensus statement.

**Definition and effects of spasticity.** Spasticity was defined by Lance as a "velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex."\(^1\) Spasticity can produce impairment of active functions performed by the patient such as reaching, transferring, and walking, as well as disabling symptoms of pain, spasm, and disfigurement, disturbance in passive functions when the patient is being assisted by a caregiver, such as excessive tone in a limb during washing, bathing, and dressing. Spasticity may also lead to musculoskeletal complications such as contracture, peripheral neuropathy, and heterotopic ossification.\(^5\)

**Management options.** Spasticity treatment is best approached in a multi-disciplinary fashion. The goals and benefits to the patient are very important when considering the path of treatment. Common goals are to decrease pain, prevent or decrease contractures, improve ambulation, facilitate activities of daily living (ADL), facilitate rehabilitation participation, save caregiver’s time, improve the ease of care, and increase safety. Appropriate management choices are based on realistic therapeutic objectives. Both the patient’s and the caregiver’s goals must be considered. Traditional treatments for spasticity include physical and occupational therapy where the patient is stretched from one to several times per day, but this has only limited effect on the patient’s spasticity. Rehabilitation treatment options include casting, orthotics, electrical stimulation, practice of functional tasks, sensory integration, muscle stretching, and targeted muscle training. Oral medications can be used to decrease spasticity, however, many have unwanted side effects such as drowsiness, sedation, confusion, and
fatigue. Intrathecal baclofen results in a greater decrease in spasticity by allowing higher concentrations of baclofen in the cerebrospinal fluid at approximately 1% of the daily oral dosage. To be considered for intrathecal baclofen pump placement, the patient must have severe lower limb spasticity that does not respond to other less invasive treatments. Other treatments include chemical neurolysis, in which the nerve conduction is impaired through the use of chemical agents, and therapeutic nerve block using phenol or alcohol. Major side effects include damage to sensory and motor nerves, pain at injection site, scarring, and dysesthesias.5 Another treatment used to alleviate spasticity is rhizotomy. Studies have shown that performing selective dorsal rhizotomy at a young age can reduce the need for orthopedic surgery. The procedure is very specialized and difficult, requiring general anesthesia and a neurophysiologist, must be present to identify which nerve is to be severed. Orthopedic surgical approaches and principles include lengthening or transferring tendons, tendonotomy, relocating or fusing joints, rotating bones, neurectomy, osteotomy, and skin release.

**Botulinum toxin type A use in spasticity management.**

Among the breakthroughs in the history of medicine is the evolving clinical use of BTX-A as a therapeutic tool. The BTX-A injections have been used as a safe and effective treatment for a variety of disorders of abnormal muscle tone, including muscle overactivity, or spasticity.11

**Mechanism and uses.** Previously known only as a cause of a serious, but often fatal, paralysis acquired through ingestion of contaminated food, botulinum toxin is a complex protein produced by the anaerobic bacterium *Clostridium botulinum*. There are several serologically distinct botulinum neurotoxins, which have slightly different specific effects. The majority of laboratory and clinical experience is with botulinum neurotoxin type A. There are 3 steps in the neurotoxic action. First, the heavy chain of the toxic protein attaches to the external membrane of motor cholinergic neurons (presynaptic nerve membrane). The toxin molecule is then absorbed into the nerve terminal by a process called endocytosis (crosses the presynaptic plasma membrane, following which the toxin inhibits release of vesicle-bound acetylcholine). The light chain of the Botulinum toxin type A cleaves SNAP-25, a cytosolic protein attached to the presynaptic membrane, which is one of the intracellular SNARE proteins required for the neurotransmitter release (these proteins are responsible for the attachment of the vesicle to the presynaptic membrane to facilitate exocytosis). Cleavage of any of the proteins results in inhibition of acetylcholine release, disruption of neuromuscular transmission, and paralysis of the muscle.12 Although botulinum toxin has been shown to enter the central nervous system in animals by retrograde axonal transport, the therapeutic effects in humans are due primarily to the blockade of peripheral neuromuscular transmission, leading to a condition known as chemodenervation. Botulinum toxin produces its therapeutic effect by a long-term blockade that leads to changes very similar to those produced by surgical denervation. These changes include muscle paralysis, atrophy, and electromyographic abnormalities. In most situations, the clinical effects of botulinum toxin are of limited duration. In experimental animals and humans, recovery is accompanied by the sprouting of new nerve terminals. The formation of new neuromuscular junctions adjacent to these sprouts parallels the recovery (effective return) of neuromuscular activity, until finally, the original functional end-plate is re-established and collateral axonal sprouts regress.13 In December 1989, the United States Food and Drug Administration approved the use of BTX-A for use in strabismus, blepharospasm, hemifacial spasm, and other disorders of the第七 cranial nerve in patients 12 years of age or older. Chemodenervation has been found to be beneficial in patients with spasticity due to multiple sclerosis, stroke, cerebral palsy, and brain or spinal cord injury in a multitude of clinical reports and trials.2-8 This has stimulated the interest and the increase use of BTX-A in both children and adults with spasticity. In most European countries, BTX-A is licensed for post-stroke wrist and hand spasticity. In some countries, such as France, Italy, and Switzerland, it is also licensed for lower limb spasticity.

**Methods used in creating the consensus.** A group of adult and pediatric neurologists and rehabilitation medicine specialists met for 2 days in Cairo in May 2005 to review all available data regarding the role of BTX-A in the management of spasticity in adults and children in Arab countries. There was an overview and presentation by Professor Franco Molteni, who participated in drafting the European Consensus Statement.10 The meeting was followed by a group discussion and drafting of the consensus statement. This was incorporated into the full document by the first 2 authors. The document was circulated among all participating authors for revision. Suggestions and corrections were included in the final version. It is imperative to mention that although the Cairo meeting was financially sponsored by Allergan, there was no influence or involvement from the drug company representatives on drafting or revising the consensus statement. All opinions in this document are based on scientific facts and clinical experience of the participating authors.
**Consensus Statement**

1. Spasticity management is an inter-disciplinary activity and should be undertaken where appropriate personnel and facilities are available.

2. Whilst the management of spasticity is a long-term process, a defined period of BTX-A treatment can guide patients and caregivers in achieving their goals.

3. When planning to use BTX-A, the team must ensure that an appropriate rehabilitation program is in place, and available post-injection to assist in achieving the treatment goals.

4. When selecting patients for spasticity treatment using BTX-A, the team has to clearly identify goals of treatments depending on the pattern of spasticity, the dynamic spastic component, posture, related symptoms like pain, limitation of activities of daily living such as personal hygiene and dressing.

5. Patients, their families and caregivers should receive appropriate information and should agree with the treatment goals (consent may be required in some countries).

6. Clinicians injecting BTX-A must have experience in diagnosis and management of spasticity including appropriate knowledge of functional anatomy and proper dosing.

7. A program of exercise, muscle stretching and splinting should follow injections of BTX-A to ensure the possibility of optimal functional benefit.

8. BTX-A is currently commercially available as BOTOX (Allergan) and Dysport (Ipsen). These 2 preparations are different. One unit of BOTOX is not the same as one unit of Dysport. A proven dose ratio has not been established. Some countries in the Middle East region have both BTX-A products. While other BTX-A preparations are available in some Arab countries, their use should be avoided until approved by the appropriate health authorities.

9. The treating team should use a formal set of evaluation measures pre- and post-injections, such as Ashworth Scale, range of motion, visual pain scale, and measures of ADL. We encourage the use of visual documentation.

### Acknowledgments

*The authors would also like to acknowledge the valuable contribution from Dr. Etribi, Ain Shams University, Egypt.*

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