The Future of Upper Extremity Spasticity Management

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KEYWORDS

• Spasticity • Upper extremity • Nerve transfers • Contralateral C7

KEY POINTS

- Treatment of upper limb spasticity has historically relied on rebalancing the affected musculotendinous units and joints.
- Future management of upper limb spasticity may focus on reestablishing a normal neuronal impulse pathway to the dysfunctional musculotendinous unit.
- Transfer of the contralateral C7 nerve root to the injured C7 nerve root may open the potential for simultaneously releasing flexor spasticity while improving extensor function for reach and grasp activities.
- On the nonsurgical frontier, robotic exoskeletons offer the potential to facilitate rehabilitation of upper extremity spasticity.

INTRODUCTION

Treatment of upper limb spasticity has historically relied on rebalancing the affected musculotendinous units and joints[1–8] by means of a wide variety of medical and surgical methods. Botulinum toxin A (Botox) therapy has been useful as a stand-alone or adjunctive modality for temporarily relaxing spasticity.[9–13] Tendon release, lengthening, or transfer procedures may help correct the resultant abnormal postures. Arthrodesis can address severe joint deformities, with static placement into a more functional position.

Although these approaches tackle the downstream effects of spasticity at the muscle, tendon, and joint levels, they do not address the primary pathologic condition within the nerve. Muscle tension is normally maintained by the γ-neuron circuit,[14–16] which is located in the spinal cord (**Fig. 1**). The activity of the γ-neuron circuit is normally inhibited by upper motor neurons within the cerebral cortex; this process regulates muscle tension and prevents spasticity. When there is an insult to the cerebral cortex, the negative feedback loop between the upper motor neuron and γ-neuron circuit is disconnected. As a result, the γ-neuron circuit runs uninhibited and muscle spasticity ensues. Peripheral procedures to address the manifestations of upper extremity spasticity do not influence the disrupted feedback loop and, as such, the pathologic neuronal pathways persist. As a result, functional return is only temporary in the case of Botox or, with respect to tendon procedures, progressively less apparent over time.

When upper extremity spasticity develops, functional placement of the hand in space is no longer achievable. This is due to the fact that the delicate balance between flexor, extensor, pronator, and supinator muscles is disrupted. Classically, patients with upper limb spasticity present with a flexed elbow, pronated forearm, flexed wrist, thumb in palm, and a flexed finger posture. This abnormal set of postures limits the

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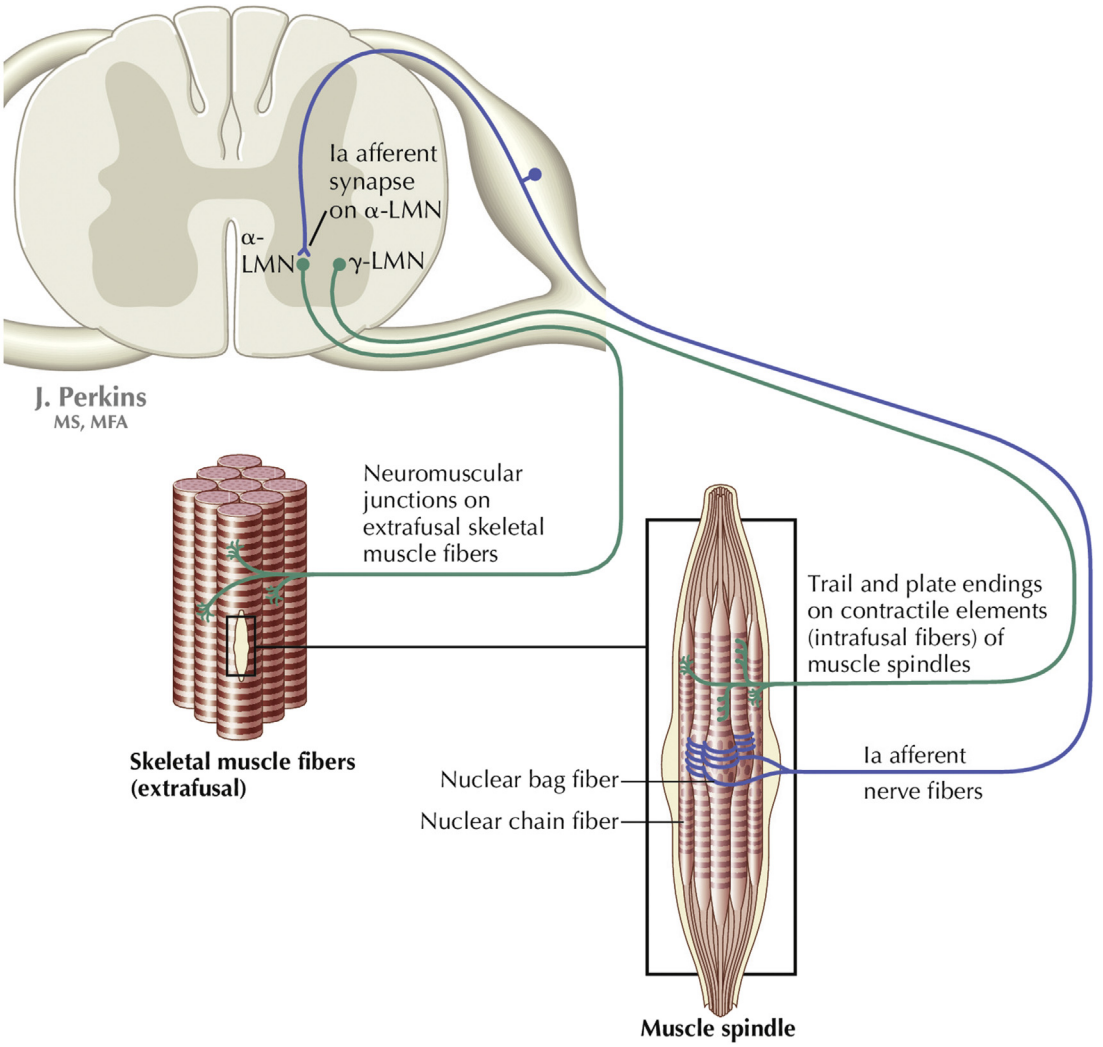

Fig. 1. Located in the spinal cord, the γ-neuron circuit maintains muscle tension and is modulated by a negative feedback loop from upper motor neurons in the CNS. (Netter illustration used with permission of Elsevier, Inc. All rights reserved. Available at: www.netterimages.com.)

interaction of the upper limb with the environment (Fig. 2).[17,18]

Depending on the severity, the flexor spasticity may produce a hygienic, cosmetic, and/or functional deformity.[1,19] Severe flexion deformities can promote skin intertrigo and eventually lead to skin breakdown, thus requiring wound management. Physical appearance is also an important consideration, because the hyperflexion deformity represents one of the key stigmata of upper limb spasticity. The functional implications of flexor spasticity are also numerous and include limitations on reaching a wider radius of objects, 2-handed manipulation of objects away from the body, and grasp, pinch, and release activities.

NOVEL SURGICAL APPROACH FOR UPPER LIMB SPASTICITY

In 2011, Xu and colleagues[20] introduced a novel surgical approach for treating upper limb spastic hemiplegia. Rather than focusing on the downstream effects of spasticity on the muscle, tendon, and/or joint, the investigators argued for intervening at the nerve level. Because the flexor muscles are spastic while the extensor groups are weak in upper limb spasticity, the investigators proposed a nerve procedure that could partially release flexor spasticity while simultaneously improving extensor function.

The investigators based their approach on previous literature demonstrating the benefits of