Management of Joint Contractures in the Spastic Upper Extremity

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KEYWORDS

• Cerebral palsy • Joint contracture • Spastic contracture • Flexion contracture

KEY POINTS

- Limited joint range of motion in a patient with upper extremity spasticity not due to bony constraint can result from muscle spasticity or contractures of the muscle or joint.
- Splinting or serial casting can be a useful initial treatment.
- Chronic spasticity can lead to secondary changes in the muscle resulting in muscle contracture, which can subsequently lead to joint contracture.
- Identifying the cause of the deformity is critical for successful treatment.
- Botulinum toxin A (Botox) is a useful adjunct to differentiate between spasticity and contracture and to guide treatment; when Botox no longer provides relief of a spastic deformity, surgical intervention may be warranted.

INTRODUCTION

Joint contractures are common in patients with upper extremity spasticity and, as such, any treatment of spasticity should include means to prevent contractures. This lack of joint mobility is due to both primary spasticity and secondary contractures resulting from longstanding spasticity. Contractures can result in poor limb function, difficulties with activities of daily living, such as hygiene and dressing, and psychological distress due to appearance. The overarching goals of treatment for these patients include improving function, which allows for greater independence, hygiene, and appearance.

CAUSE OF JOINT CONTRACTURES

Joint contractures are defined by decreased range of motion (ROM) of the affected joint. Although this term is nonspecific, patients with upper extremity spasticity-related joint contractures typically result from 3 interrelated physiologic mechanisms:

1. Primary spasticity
2. Muscular fibrosis/contraction
3. Intrinsic joint contracture

These mechanisms typically occur sequentially and are summarized in **Fig. 1**.

Spasticity

As referenced in O'Dwyer[1], Lance concisely defined spasticity as a "motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes ("muscle tone") with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome".[1] Spasticity of particular

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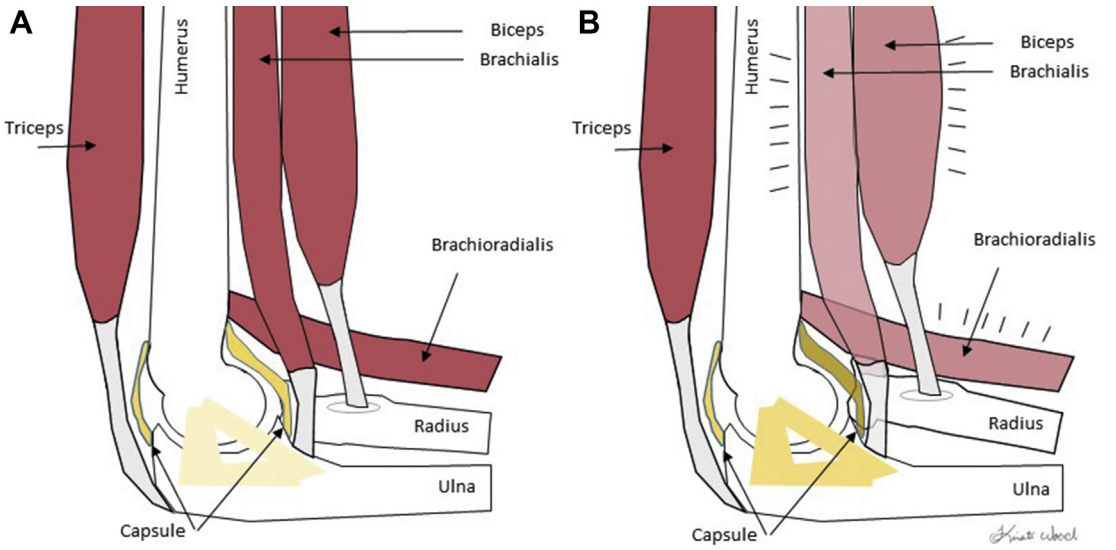

Fig. 1. (*A*) A normal elbow and (*B*) an elbow with spastic contractures. Contracture can be due to spastic and/or fibrotic flexors (biceps, brachialis, brachioradialis), tight capsule, tight ligaments, and/or dislocated radial head.

muscle groups or imbalance of muscular forces around a joint leads to limited ROM of a joint. It is important to appreciate the role of spasticity as the initiating factor in muscle and joint contracture development.

Muscle Contracture

A muscle contracture refers to a shortening of muscle length due to a decrease in the number of sarcomeres in series along the myofibrils, accompanied by an increase in the resistance to passive stretch.[1–5] Immobilization with the muscle in a shortened position produces decreased muscle fiber length, along with shortening of muscle connective tissue and an increase in muscle stiffness.[1] In patients with disruption of central command execution (ie, those at risk for spasticity), numerous peripheral effects occur because of immobilization of a joint in a fixed position or maintaining a muscle in a shortened position. In addition to actual spastic shortening, secondary causes for muscle shortening or contraction include muscle atrophy, sarcomere loss, accumulation of intramuscular connective tissue, increased muscular fat content, degenerative changes at the myotendinous junction, and an increase in mechanical spindle stimulation by stretch.[6] One study found that the muscle shortening occurs at the fascicle level in patients after spinal cord injury.[7] Taken together, these suggest that chronic spasticity will lead to changes in the muscle resulting in less contractility and elasticity due to increased muscle stiffness; this ultimately can lead to contracture. Maintaining muscle length and joint mobility is therefore important in preventing the onset of contractures.[8]

Once an upper extremity joint contracture occurs, successful treatment requires an understanding of the primary cause. Elucidating whether the cause of the limited ROM is due to spasticity alone or the secondary effects of muscle contracture can be challenging. Botulinum toxin type A (Botox) can be a useful diagnostic tool in making this distinction.

Shear wave elastography is an ultrasound tool that provides direct quantitative in vivo measurement of tissue material properties.[9–11] Multiple ultrasound push beams are used to induce the shear waves, and subsequently, to measure the shear wave speed in muscle. The shear wave speed is related to material properties, such that shear waves travel faster through stiffer tissues. As such, higher velocities indicate stiffer tissues[9] and can be associated with increased spasticity.[12]

Studies on the use of shear wave sonoelastography evaluation of muscle have shown potential for assessing structural changes in muscles, which may in turn prove useful in prognostication, evaluation, and treatment of spasticity.[11,13–16] When combined with the Modified Ashworth Scale, spasticity can be better quantified.[14] In addition, it may become a readily available tool that can assess the effect of Botox on a spastic muscle[17] or potentially predict poor candidates through assessing the extent of chronic changes within muscle structure.[18]

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