Surgical Management of Spasticity of the Shoulder

Dan A. Zlotolow, MD^{a,b,*}

KEYWORDS

• Shoulder • Spasticity • Hemiplegia • Quadriplegia • Cerebral palsy • Traumatic brain injury

Stroke

KEY POINTS

- There are multiple etiologies for developing spasticity of the shoulder.
- All etiologies involve deregulation of the lower motor neurons.
- Athetoid movements are difficult to manage.
- All or some muscles of the shoulder can be involved.
- Treatment depends on the needs of the patient and the muscles involved.

INTRODUCTION

Although much has been written about cerebral palsy (CP) and spasticity of the upper limb, relatively little has been devoted to the shoulder.¹⁻⁴ Even the textbook, The Pediatric Upper Extremity, contains a chapter that only covers surgical options distal to and including the elbow.⁵ Perhaps this is because most surgery for spasticity is performed by either general pediatric orthopedists or adult hand surgeons, both of whom are generally less comfortable treating the shoulder. More likely, however, is that the shoulder is less problematic in this patient population than other joints and merits less attention. The more common position of shoulder adduction and internal rotation is rarely the limiting factor for patient function; this more commonly depends on terminal limb (hand) control (Fig. 1). Hygiene may be an issue if the adduction contracture is severe, particularly if accompanied by an internal rotation contracture. Donning and doffing clothing also may be an issue. Even more rarely, we have seen patients with difficulty fitting into chairs or getting through doorways because of bilateral external rotation and abduction contractures. Although shoulder spasticity is relatively uncommon, it may present significant functional problems for those affected.

PREOPERATIVE ASSESSMENT

Regardless of etiology, injuries to the brain are difficult to manage peripherally with the tools available to a hand surgeon. Although we can rearrange the muscles that remain under the control of the central nervous system and release, replace, or remove those muscles that are not, we are not able to directly impact the underlying problem of control. After splinting and therapy have failed to provide sufficient improvement, surgery may be appropriate in some cases.

Patients tend to fall into 2 broad categories: those with some volitional motion and those without (Fig. 2). For patients with no volitional motion, the goals are not to improve function (that is not possible), but rather to facilitate care. Patients with volitional motion can be further subdivided into those limited by spasticity and those limited by lack of coordination (athetoid movements);

E-mail address: DZLOTOLOW@YAHOO.COM



Disclosure Statement: Ownership or Royalty Agreement: Osteomed, Arthrex, McGinley Orthopaedics, Elsevier, Springer.

^a Department of Orthopaedics, The Hospital for Special Surgery, 535 East 70th Street, New York City, NY 10021, USA; ^b Shriners Hospital for Children Philadelphia, 3551 North Broad Street, Philadelphia, PA 19140, USA * 3551 North Broad Street, Philadelphia, PA 19140.



Fig. 1. Typical posture of the shoulder in patients with spastic shoulder girdle muscles. (Used with permission of Shriners Hospitals for Children–Philadelphia, Philadelphia, PA. All rights reserved.)

athetoid movements are a relative contraindication for surgery.

The cause of spasticity needs to be considered before any surgical intervention. Function in patients with progressive brain lesions, such as tumors and multiple sclerosis, will deteriorate over time, making any gains from surgical intervention less enduring. Relatively static lesions, such as



Fig. 2. Testing the volitional strength of the deltoid. (Used with permission of Shriners Hospitals for Children–Philadelphia, Philadelphia, PA. All rights reserved.)

CP, may also have peaks of spasticity that will impact timing of surgical intervention. We tend to delay definitive surgery for CP until after the peak of spasticity, typically approximately 12 years of age. Lesions that are in evolution, such as in the acute period following a stroke or traumatic brain injury, may require a wait-and-see approach until spasticity stabilizes. The goals in the acute period are instead to minimize joint contractures with therapy, splinting, and chemodenervation. Afterward, antispasmodic agents should be titrated until spasticity is minimized without compromising global function.

Once the level of spasticity has relatively stabilized, a thorough assessment of the patient's functional goals and neurologic, muscular, and joint assets and liabilities is performed; goals may need to be curtailed or expanded based on what is realistically possible. Managing expectations of patients and family members becomes a primary concern during the surgical planning phase.

Preoperative assessment tools, such as electrodiagnostic studies, motion analysis, and spasticityspecific instruments, such as the Shriners Hospital for Children Upper Extremity Evaluation (SHUEE),⁶ can be very helpful. In particular, the SHUEE can be used to assess the efficacy of the surgical intervention by comparing preoperative and postoperative scores. Electromyography (EMG) is helpful for ascertaining volitional control and baseline spasticity of individual muscles. Motion analysis can demonstrate joint contractures and movement patterns.

We also consider botulinum toxin A (Botox) to be a preoperative assessment tool. Injection of an affected muscle with Botox offers a preview of what a release or permanent denervation may yield. If the improvements in function or posture are clinically significant after chemodenervation, the results provide greater confidence that a permanent solution may also yield similar benefits.

SURGICAL PLANNING

Surgical plans are built from the inside out; the first concern is to release restrictive joint contractures. More than almost any other joint in the body, the shoulder relies on capsular and muscular constraints for stability. When peri-glenoid muscles lose their excursion, the resultant loss of motion leads to capsular contracture. If the patient's active range of motion matches his or her passive motion, then no tendon or nerve transfer will augment motion. However, if there is a motion lag, muscles can then be transferred or differentially innervated or denervated, as needed, to augment active motion. Last, the skin may need Download English Version:

https://daneshyari.com/en/article/11013667

Download Persian Version:

https://daneshyari.com/article/11013667

Daneshyari.com