J Shoulder Elbow Surg (2012) 21, 691-698

ELSEVIER

Journal of Shoulder and Elbow Surgery

www.elsevier.com/locate/ymse

Outcomes of tendon fractional lengthenings to improve shoulder function in patients with spastic hemiparesis

Surena Namdari, MD, MSc, Hassan Alosh, MD, Keith Baldwin, MD, MPH, MSPT, Samir Mehta, MD, Mary Ann Keenan, MD*

Department of Orthopaedic Surgery, Hospital of the University of Pennsylvania, Philadelphia, PA, USA

Background: Patients with spastic hemiparesis after upper motor neuron (UMN) injury often exhibit limited shoulder movement. We evaluated the outcomes of shoulder tendon fractional lengthenings in patients with spasticity and preserved volitional control.

Methods: A consecutive series of 34 adults with spastic hemiparesis from UMN injury (23 post-stroke, 11 post-traumatic brain injury) and limited shoulder movement with preserved volitional motor control who underwent shoulder tendon fractional lengthenings (pectoralis major, latissimus dorsi, teres major) were evaluated. Active and passive shoulder motion, spasticity, pain, and satisfaction were considered pre- and postoperatively.

Results: There were 15 males and 19 females with a mean age of 44.1 years. Mean follow-up was 12.2 months. Mean Modified Ashworth spasticity score was 2.4 preoperatively compared to 1.9 postoperatively ($P = .001$). Active flexion, abduction, and external rotation improved compared to the normal contralateral side ($P < .001$) with most dramatic gains in external rotation. Similarly, passive extension, flexion, abduction, and external rotation improved compared to the normal contralateral side ($P < .01$). Ninety-four percent (15/16) with preoperative pain had improved pain relief postoperatively with 14 (88%) being pain-free. Thirty-one (92%) were satisfied with the outcome.

Conclusion: Shoulder tendon lengthenings can be an effective means of pain-relief, improved motion, enhanced active motor function, and decreased spasticity in patients with spastic hemiparesis from UMN injury.

Level of evidence: Level IV, Case Series, Treatment.

Keywords: Tendon lengthening; spasticity; hemiparesis

Patients with spastic hemiparesis after upper motor neuron (UMN) injury often exhibit limited active and passive shoulder movement. Disorders of acquired spasticity most commonly include traumatic brain injury (TBI) and cerebrovascular accidents. These central nervous system disorders cause disruption of UMN inhibitory pathways. The result is UMN syndrome. Characteristics of UMN syndrome include the presence of spasticity and other forms of involuntary muscle overactivity, voluntary weakness, and a variety of motor control abnormalities that impair the regulation of voluntary movement. These can lead to static and/or dynamic components of deformities.

Institutional Review Board of Hospital of University of Pennsylvania, IRB Protocol # 810370.

*Reprint requests: Mary Ann Keenan, MD, Hospital of University of Pennsylvania, 2 Silverstein, 3400 Spruce Street, Philadelphia, PA, USA 19104.

E-mail address: MaryAnn.Keenan@uphs.upenn.edu (M. Keenan)

1058-2746/$ - see front matter © 2012 Journal of Shoulder and Elbow Surgery Board of Trustees.
doi:10.1016/j.jse.2011.03.026

In the upper extremity, a flexion synergy pattern is common, which results in a position of shoulder adduction and internal rotation with the arm held at the side.[6,20] Spasticity, impaired muscle control and weakness lead to muscle imbalance and are considered to be the dynamic components of the shoulder deformity. With time, the adducted, internally rotated position of the shoulder can result in soft tissue shortening and the formation of a static contracture. These upper limb deformities result in many dynamic and static functional problems for the patient, including difficulty with volitional movement of the limb.

Patients with spastic shoulders after UMN injury can either lack all motor control (hemiplegia) or have variable levels of preserved motor control (hemiparesis).When a patient has a contracture in the absence of any volitional shoulder movement, adduction and internal rotation of the shoulder can lead to maceration or inadequate hygiene in the axilla and difficulty with dressing. We have previously shown that a release of the contracted muscles in patients without motor control can restore passive mobility and relieve pain.[14,15] However, the hemiparetic patient with a spastic shoulder and preserved active motor control presents unique challenges.

In this group, limitation in movement can be secondary to tendon contractures, abnormal patterns of muscle activation, and spasticity. The clinical issue is whether the limited motion is a result of absent or weak activity of the shoulder muscles or the result of inappropriate activity (dyssynergy or co-contraction) of the antagonist muscles. If the limitation is caused by restriction of movement by the antagonist muscles, then theoretically, these muscles can be selectively lengthened and movement improved. Historically, clinical examination has been the mainstay of evaluation and decision-making for patients who have spastic limb deformities. Clinical assessment supplemented by instrumented laboratory analysis with dynamic poly-electromyography (poly-EMG) has helped characterize movement disorders and has been shown to improve the outcomes of treatment.[3,6] Poly-EMG data can interpret whether effort-related initiation, modulation, and termination of voluntary activity are present in a given muscle and whether the behavior of a particular muscle is dyssynergic.[20] We evaluated outcomes of selective fractional lengthening in patients with adduction and internal rotation deformity of the shoulder to improve motion and function in patients with preserved volitional active movement after UMN injury.

Methods

Study sample

This was a retrospective case series of 34 consecutive hemiplegic patients. All procedures were performed by the senior author (MAK) between the years 2003 and 2008. All patients had an underlying UMN injury from stroke or TBI. Inclusion criteria included all patients with spastic hemiparesis and some preserved baseline volitional motor control who underwent selective fractional lengthening to treat spastic contractures of the shoulder. Motor control was graded in the extremity using a clinical scale.[11] In this scale, the extremity can be hypotonic and without any volitional movement (Grade 1) or rigid without any volitional movement (Grade 2). The extremity may exhibit mass flexion or extension patterned motor control. This can be reflexive (Grade 3) or volitional (Grade 4). Alternatively, motor control can be selective with pattern overlay (Grade 5), allowing movement of a single joint with minimal movement in the adjacent joints, or volitional (Grade 6), allowing movement of a single joint independently of the adjacent joints. Patients with Grades 1-4 motor control were deemed to lack the level of baseline control necessary to obtain substantial improvement from selective tendon lengthening. Patients with Grades 5 and 6 motor control were considered most appropriate for selective tendon lengthenings and were surgical candidates. Exclusion criteria included patients with heterotopic ossification, glenohumeral arthritis, contralateral shoulder injury or pain, and Grades 1-4 motor control. Patients were not considered for surgery if dynamic poly-EMG showed muscle weakness, lack of muscle activity, and/or absence of muscle co-contraction or dyssynergy.

Preoperative evaluation

All patients were evaluated before and after surgery using a standard, detailed format. The static and dynamic components of the deformity were evaluated in detail prior to surgery with the help of thorough clinical evaluation. In our practice, patients with Grade 5 or 6 motor control are generally treated with selective tendon lengthening rather than tendon releases. These patients were all of sufficient cognitive ability to follow commands and demonstrated some volitional movement in the upper extremity. Dynamic poly-EMG was utilized to determine surgical candidacy and planning. In patients who demonstrate some volitional control in the arm, the clinical question is whether the limited forward flexion is a result of absent or weak activity of the shoulder flexor muscles or the result of inappropriate activity (dyssynergy or co-contraction) of the shoulder extensor muscles during forward reach (Fig 1, *A*). If the limitation of forward reach is caused by restriction of movement by the posterior muscles, then theoretically, these muscles can be selectively lengthened and movement improved. If the limited flexion is the result of weakness (Fig 1, *B*), then a strengthening program could potentially be more helpful. If there is no activity of the shoulder flexors, then no treatment is currently available to correct this problem and improve active function. Poly-EMG was used to identify dyssynergy or co-contraction, defined as inappropriate muscle firing during antagonist motion. EMG recordings from specific muscles, the lateral head of the triceps, pectoralis major, teres major, and latissimus dorsi in conjunction with the movement tracing allowed the clinician to obtain detailed information regarding the activity of each muscle, both during active efforts by the patient and passive efforts by the examiner. In cases in which there was a question regarding whether contractures were too rigid to result in substantial active motor function with lengthening, selective bupivicaine blocks of the dyssynergic muscles were used to demonstrate temporary improvement of active flexion and external