

THE EFFECT OF SHOULDER GIRDLE COORDINATION ON UPPER EXTREMITY WORKSPACE IN STROKE

¹Ana Maria Acosta and ^{1,2}Jules Dewald

¹Northwestern University, Department of Physical Therapy and Human Movement Sciences, ²Department of Physical Medicine and Rehabilitation, Department of Biomedical Engineering ; email: a-acosta@northwestern.edu

INTRODUCTION

The extensive range of motion of the healthy shoulder is a result of the integrated movements of the sternoclavicular, acromioclavicular, glenohumeral, and scapulothoracic joints. These movements are achieved by the coordinated action of the shoulder muscles that also act to maintain joint stability. It is generally accepted that the interaction between rotator cuff muscles and the deltoid muscles is crucial for effective arm elevation while the coordinated action of trapezius, rhomboids and serratus anterior allows scapular rotation necessary for full arm elevation [1]. However, it is not known how these patterns of muscle activation change following stroke, and how this impacts the shoulder range of motion in this population. The goal of this work is to understand the effect of brain injury due to stroke on shoulder muscle coordination and its impact on arm workspace.

METHODS

The shoulder kinematics were measured in one subject with unilateral brain injury resulting in left hemiparesis. The AROM in the affected limb was limited to 90° abduction. The kinematics, including scapular rotations, were measured statically using the standardized protocol developed by van der Helm [2]. The 3D position of the trunk, arm, forearm, and scapula was measured using a motion analysis system that tracked the location of infrared LEDs (IREDs) mounted on rigid bodies. Segment kinematics were calculated based on bony landmarks [3] that define their location and orientation. The locations of the scapular bony landmarks were recorded by placing a tripod instrumented with IREDs on the bone. In addition, surface EMG of a set of shoulder and elbow muscles was recorded simultaneously with the kinematics. The subject was asked to abduct (coronal plane) and flex (sagittal plane) the arm to specific angles in increments of 15° up to the maximum voluntary elevation in the affected side and maintain each posture for 5 s while the 3D location of the IREDs was recorded. The measurements were repeated twice with resting periods between trials to avoid muscle fatigue.

RESULTS AND DISCUSSION

Figure 1 shows the scapular kinematics calculated for the

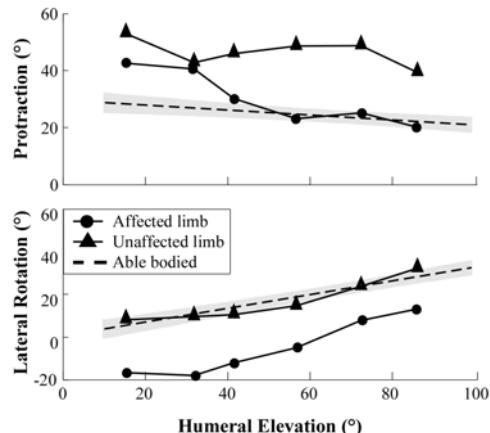


Figure 1. Scapular kinematics in a subject with stroke compared to able-bodied.

Table 1. Average EMG (%MVC) activity in upper and middle heads of the trapezius muscle in the stroke subject.

	Utrap	MTrap
Unaffected arm	84.4 ± 10.9	7.6 ± 1.6
Affected arm	42.2 ± 9.8	16.8 ± 3.7

affected (solid circles) and unaffected (solid triangles) limbs compared to able bodied (dashed line – gray area indicates 95% confidence interval). The able bodied kinematics are represented as the linear regression of the experimental data calculated from both limbs five subjects [3] with the associated 95% confidence interval. Note that the lateral rotation of the scapula, typically associated with the scapulo-humeral rhythm, in the unaffected side is similar to able bodied. However, in the affected side, the scapula is more medially rotated across arm elevations, indicating a more medial resting position. The scapulo-humeral rhythm or the relationship between the lateral rotation and the humeral elevation is similar between the affected and unaffected limbs. The mean or resting protraction angle of the scapula also differed across limbs, especially at higher elevation angles. In contrast, the tipping angle (not shown) was similar between limbs and to able bodied. Both of these angles represent the scapular winging. These results from a single subject are preliminary evidence to the alteration in muscle activation patterns. The scapular kinematics could be explained by increased activity in the Middle Trapezius (MTrap) and Rhomboid muscles and reduced activity of Upper Trapezius (UTrap) muscle as was actually observed in the EMG measurements. The average normalized MTrap EMG while supporting the arm at 90° abduction was twice as large for the affected arm than for the unaffected arm (see Table 1), while the activity of UTrap in the affected limb was approximately half of the activity measured in the unaffected limb. These results support the notion of altered coactivation patterns responsible for changes in the scapular kinematics. The increased activity in the affected side MTrap muscle pulls the scapula to a more medial rotation compared to the unaffected side. Note that although the activity in UTrap increased with arm elevation, it is still lower in the affected side, which may provide an explanation for the reduced range of motion in the affected arm. Under normal conditions, the scapula rotates laterally to allow the head of the humerus to clear the acromion. However, if the scapular rotation is not sufficient, the head of the humerus will encounter a physical stop that prevents the arm from being elevated above this point. Future work will provide further evidence for abnormal muscle coactivation patterns by measuring the activity of additional key muscles including rhomboids and rotator cuff muscles in a representative sample of stroke subjects.

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