AN ELECTROMYOGRAPHIC ANALYSIS OF HORIZONTAL REACHING FOLLOWING THE TRANSFER OF THE POSTERIOR DELTOID IN TETRAPLEGICS

¹ Mark Robinson, ¹Gabor Barton, ¹Adrian Lees and ²Pradip Sett

¹ Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, UK.

² North West Regional Spinal Injuries Centre, Southport and Ormskirk Hospital NHS Trust, UK.

Email: m.a.robinson@ljmu.ac.uk Web: www.ljmu.ac.uk/RISES

INTRODUCTION

Following a spinal cord injury (SCI) at the C5/6 level the triceps is left paralysed. Any simple reaching movement therefore requires an alternative muscle activation pattern. Koshland et al. [1] identified that individuals with tetraplegia compensate for their lack of triceps activation by reducing shoulder accelerations and only activating the shoulder agonist. This is of contrast to a healthy person who would typically use a reciprocal (tri-phasic) muscle activation pattern. To restore active elbow extension, the unaffected posterior deltoid (PD) muscle can be surgically transferred. People with tetraplegia can then perform reaching movements kinematically and temporally comparable to neurologically healthy individuals [2]. The adaptation of the nervous system to this new muscular arrangement, specifically in terms of the activation of the PD and biceps is unknown. The aim of this pilot study was to test the hypothesis that the transferred PD works reciprocally with the biceps to control the shoulder and elbow during reaching.

METHODS

One C5/6 tetraplegic participated, one arm had the PD transferred onto the triceps tendon, the other arm had normal PD but no triceps function. A 20 cm centre-out horizontal reaching movement was performed to 6 directions spaced at 60° intervals. This allowed the PD to be assessed when acting as both an agonist and antagonist. A ball transfer unit was secured to the wrist to allow a low friction movement over the horizontal surface. Force sensitive resistors were used to determine the start and end of the reach (Figure 1).

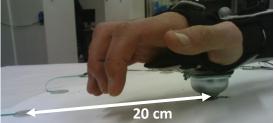


Figure 1: A ball transfer unit supported the wrist to provide a smooth reaching movement to 6 directions at 60° intervals.

Surface electromyography (EMG) of the Biceps Brachii, Posterior Deltoid, Pectoralis Major (sternal head) and the Triceps Brachii was collected. The EMG signal was sampled at 1920 Hz then band pass filtered at 20-400 Hz, rectified and a linear envelope created using a 4 Hz low-pass filter. EMG data was normalised to the peak value within each muscle. Kinematics of the reach were assessed using retroreflective markers on the left and right acromion, the elbow and wrist (Qualisys, optoelectronic camera system, 240 Hz).

RESULTS AND DISCUSSION

Normalised EMG and kinematic data for one target direction which required humeral adduction and elbow extension are presented in figures 2 and 3. Vertical lines represent the start and end of the reach movement, data 0.25 s before and after reach was also included then normalised to 101 points.

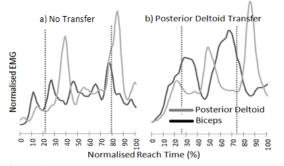


Figure 2: Biceps and PD muscle activations. No synergy is seen without transfer (a) but following transfer (b) a reciprocal muscle action is evident.

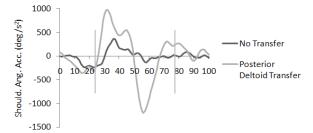


Figure 3: Shoulder angular acceleration in tetraplegic arms with and without PD transfer. A lack of active deceleration (antagonist activity) is seen in the arm with no PD transfer.

Before transfer, the PD has an antagonistic role to control the movement of the humerus and there is little synergy between the biceps and PD (Figure 2a). After the PD transfer, the biceps now works reciprocally with the PD (Figure 2b) to control humeral and elbow movement. As both the PD and biceps now articulate the shoulder and elbow joint, the central nervous system utilises this new arrangement to reciprocally activate the PD and biceps to provide improved control of the shoulder and elbow. Improved control is also evident in the kinematics of the reach as the reciprocal action allows active acceleration and deceleration of both the shoulder and elbow which was not apparent before the transfer (Figure 3). The original hypothesis is supported although a full study is required to confirm this in other tetraplegic patients.

REFERENCES

- 1. Koshland et al., Exp Brain Res. 164: 133-147, 2005.
- 2. Robinson, M.A. et al., Proceedings of ESB XVI, Lucerne, Switzerland, 2008.