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Shoulder abduction-induced reductions in reaching work area following hemiparetic stroke: neuroscientific implications

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Abstract

A stroke-related loss of corticospinal and corticobulbar pathways is postulated to result in an increased use of remaining neural substrates such as bulbospinal pathways as individuals with stroke are required to generate greater volitional shoulder abduction torques. The effect of shoulder abduction on upper extremity reaching range of motion (work area) was measured in 18 individuals with stroke using the Arm Coordination Training 3-D (ACT^{3D}) device. This robotic system is capable of quantifying movement kinematics when a subject attempts to reach while simultaneously generating various levels of active shoulder abduction torque. We have provided data demonstrating an incremental increase of abnormal coupling of elbow flexion for greater levels of shoulder abduction in the paretic limb that results in a reduction in available work area as a function of active limb support. The progressive increase in the expression of abnormal shoulder/elbow coupling can be explained by a progressive reliance on the indirect cortico-bulbospinal connections that remain in individuals following a stroke-induced brain injury.

Keywords

Stroke; Arm; Coordination; Kinematic; Synergies; Strength

Introduction

In individuals with hemiparetic stroke, reaching with the paretic arm can be impaired by abnormal muscle coactivation of shoulder abductors with elbow flexors (Dewald et al. 1995). This coactivation is implicated in stereotyped movement patterns that were first described as a “flexion synergy” (Twitchell 1951; Brunnstrom 1970). It is well documented that abnormal muscle coactivation is present in individuals with moderate to severe stroke (Dewald et al.

1995, 2001; Dewald and Beer 2001; Beer et al. 2007; Ellis et al. 2007) and that these coactivations result in a reduced active range of motion when reaching against gravity (Beer et al. 1999, 2000, 2004). Although Beer and colleagues were able to elegantly quantify reaching kinematics as a function of supported versus unsupported reaching, the methodologies were limited to only two reaching conditions, and a center-out reaching task that did not represent the entire range of motion or work area of the subject. In this study, we defined work area as the largest region that a subject can access with their hand in the horizontal plane at 90° of shoulder abduction. The present study was designed to quantify the effects of progressively greater shoulder abductor activation on total work area of the paretic arm and more importantly, the constituent kinematic attributes that will ultimately point to the underlying neurophysiological mechanisms responsible for the impairment.

We hypothesized that total work area of the paretic arm would decrease in response to progressively greater shoulder abduction requirements in a manner consistent with an increasing expression of the flexion synergy. This may be attributed to an incremental substitution of corticospinal by corticobulbospinal motor pathways (Beer et al. 1999, 2007; Dewald and Beer 2001; Ellis et al. 2007). To fully characterize the nature of this substitution, it was necessary to test an array of abduction requirements ranging from cases where the subject's arm was fully supported (i.e. no active limb support) to cases where subjects were required to actively support loads beyond the weight of their limb. If upper limb movement impairment following stroke is related to corticobulbospinal takeover and associated abnormal muscle and joint torque coupling, as documented in our previous isometric studies, one would expect a progressive reduction in work area as subjects are required to actively support more and more of the weight of their limb in a dynamic task. Conversely, if the impairment is the result of a different mechanism such as shoulder weakness/paresis, full reaching work area would be expected to remain relatively constant until an abduction level is reached where subjects are unable to produce enough torque with the shoulder abductors to lift the paretic arm. Results of this study support the hypothesis that reaching reductions in individuals with stroke are caused by abnormal shoulder abductor/elbow flexor coupling and may be explained by an increased contribution of bulbospinal systems as reported previously (Beer et al. 2007; Ellis et al. 2007). Portions of this work have been published in abstract form (Sukal et al. 2005).

Methods

Subjects

Eighteen subjects (ranging 39–65 years in age) with chronic hemiparesis, 2–25 years post infarct, were recruited for this study. All subjects were screened for inclusion in the study; subjects were excluded if they had difficulty with sitting for long durations, recent changes in the medical management of hypertension, any acute or chronic painful condition in the upper limbs or spine, or greater than minimal sensory loss in the paretic upper limb. All subjects were evaluated with the arm motor portion of the Fugl-Meyer motor assessment (FMA) (Fugl-Meyer et al. 1975) and the Chedoke-McMaster motor assessment (CMA) arm scale (Gowland et al. 1993). The inclusion criteria for the research study required mild to severe impairment equivalent to scoring within the range of 2–6 out of a maximum of 7 points on the CMA, or between 15 and 50 out of a possible 66 on the FMA. Additionally, subjects had to be able to lift their arm and volitionally extend the elbow slightly. Each subject's passive range of motion of the paretic upper limb was also measured. A passive range of motion of at least 90° of shoulder flexion, abduction, and neutral internal/external rotation was required to participate in the study. Overpressure at the end of the range of motion was used as a medical screening to verify the absence of an inflammatory condition at the shoulder, elbow, wrist and fingers. All subjects provided informed consent in accordance with the Declaration of Helsinki prior

to participation in this study, which was approved by the Institutional Review Board of Northwestern University.

Measurement device

The Arm Coordination Training 3-D (ACT^{3D}) device, shown in Figs. 1 and 2, was recently developed in our lab. It consists of a modified HapticMASTER robot (Moog-FCS B.V., The Netherlands) with an instrumented end effector, integrated with a Biodex experimental chair (Biodex Medical Systems, Shirley, NY). The HapticMASTER (HM) is an admittance controlled robot (Van der Linde et al. 2002), and therefore imparts a negligible amount of inertia during a reaching task. The end effector consists of a six degree of freedom measurement device (JR3 load cell, Woodland, CA) to monitor forces and torques and an instrumented gimbal to record joint angles. Additionally, the ACT^{3D} was used to provide a frictionless, stiff haptic surface (referred to as the “haptic table” hereafter) and impose forces on the arm to either increase or decrease the amount of limb support required of the subject during the reaching task, as explained further in the protocol. A rigid forearm-hand orthosis with Velcro straps was used to couple the arm directly to the robot without the need for external slings or other supports. Position of the subject in relation to the robot was controlled via a common support track to which the chair and robot were both mounted.

An OpenGL rendered representation of the arm on a computer monitor provided the subject with online feedback about limb configuration and target location during experiment tasks. The feedback display and target locations, shown in Fig. 3, were tailored to the individual subject's limb length. The shoulder was located by back-calculating from the HM endpoint while the arm was in a known position, and subsequently shoulder and elbow angles were determined from that location. Auditory feedback was also used when the endpoint of the device made contact with the haptic table during tasks where the arm was required to stay above the table. This will be explained further in the protocol.

Protocol

Subjects sat in the experimental chair with their arm resting in a forearm-hand orthosis attached to the ACT^{3D}. The orthosis maintained the wrist and hand in a neutral position and the subject's trunk was immobilized to prevent shoulder girdle movement by a set of straps attached to the experimental chair (see Fig. 2). Small shoulder movements were visually monitored by the experimenters by observing both the subject's shoulder as well as the visual feedback display that could indicate shoulder protraction or retraction to the experimenter. The shoulder was positioned at 90° of abduction when the tested arm was resting on the haptically rendered table. The mechanical interface, or gimbal, between the subject and the HM component of the ACT^{3D} was locked to constrain movements to a plane such that during reaching or retrieving movements, the subject's arm and hand were in line with the center of rotation of the shoulder, thus preventing internal and external rotation at the shoulder. Subjects were manually placed in an initial position of 90° elbow flexion and 40° shoulder flexion using the acromium, ulnar and radial epicondyles, and the distal end of the third phalanx as anatomical landmarks (Zatsiorsky and Seluyanov 1985; Shiba et al. 1988) with a goniometer. Custom software calculated the position of the shoulder and a display of an avatar of the arm was adjusted to mimic the subject's view of their actual arm configuration.

Subjects were asked to make the largest circle they could with their arm fully supported by and gliding on the haptic table, similar to the conditions of the air bearing table in previous studies (Beer et al. 2000, 2004). The movement task was performed slowly (~5°/s joint velocity) to minimize the effects of hyperactive stretch reflexes or spasticity. Subjects performed approximately three circles in a clockwise direction and three circles in a counterclockwise direction, the order of which was randomized, in an effort to capture the full reaching work

area. Rest was given between the two directions to eliminate fatigue, and verbal feedback was given to encourage the subject to achieve maximal movement extent. Total available work area was captured in two stages because the range of the current ACT^{3D} system is insufficient to accommodate the total work area of the test arm in adults. These stages consisted of determining the left and the right halves of the work area separately by changing the subject/ACT^{3D} configuration, but setting the manual home position in the same way for each side of the work area.

After completing the trials while sliding on the haptic table, the experimental chair was elevated by approximately one inch, and subjects were required to actively support their arm just above the haptic table resulting in 90° of shoulder abduction as it was when supported by the haptic table. The gimbal remained locked during these trials to prevent internal and external rotation at the shoulder, as during the table-supported trials. Subjects were again asked to generate the largest possible slow circles with their arm in clockwise and counterclockwise directions with adequate rest on the table allowed between each trial. The ACT^{3D} was used to provide forces along its vertical axis to alter the amount of active limb support the subject was required to generate. For example, in the 0% active support condition, the ACT^{3D} provided a force equal to that of the subject's relaxed limb such that no net abduction torque was required for the subject to maintain their arm in a position above the table. A total of nine support levels were randomized for testing. They ranged from 0% to 200% of required active limb weight support, in increments of 25% of limb weight. If subjects were not providing adequate abduction torque to stay above the table during a trial, an auditory signal cued them to lift more. In the event that they did touch the table, subjects were instructed to return to the starting position and begin again, and the associated trial was excluded from further analysis. Experimenters monitored subjects for excessive lifting and verbally cued them to lower the arm if they significantly exceeded 90° of shoulder abduction.

In 15 of the subjects, this entire procedure was completed for both the paretic and non-paretic limb within one week of one another; the remaining three were only tested on the paretic side due to scheduling complications.

Analysis

All trials were normalized to the same coordinate system in which the shoulder was located at the origin, and the x-axis was aligned through the center of rotation of both shoulders. For each testing condition (on the table and each of the active support levels), the circles from all trials of the left and right sides of the work area were overlaid by matching the home target positions from each stage of testing. Data points where the arm touched the table were eliminated (except in the table-supported condition), and the outermost path was selected and saved. This outermost path was translated into polar coordinates and a triangular approximation was used to calculate the area of the circle for each condition. Additionally, at each point on the outermost path, shoulder and elbow angles were back-calculated using measured segment lengths and shoulder position. Peak flexion and extension angles for the shoulder and elbow were determined for each limb at all support levels.

Areas for all subjects were normalized to the area they were able to achieve while supported on the table to account for differences in limb length. Data Desk statistical analysis software (Ithaca, NY) was used for all statistical analysis. Two-factor ANOVAs were used to determine if there was an effect of limb and active support level and an interaction effect of limb and active support level on work area and peak shoulder and elbow flexion/extension angles. If a significant effect was found, post hoc testing was implemented using the Bonferroni test. Maximum values of elbow flexion, elbow extension, shoulder flexion and shoulder extension of the paretic limb were also identified for each support level and grouped across subjects. Correlation analysis was done using Pearson correlation coefficient to determine the

relationship between work area and peak elbow/shoulder range of motion. Finally, a linear regression was used to estimate the effect of abduction level on each of these four joint angles. Effects were deemed significant if the *P*-value was less than or equal to 0.05.

Results

Work area

A representative sample of measured work area from one subject is shown in Fig. 4. This subject has left hemiparesis, however, for an easier comparison between arms, the work area of the paretic arm has been spatially inverted along the y-axis. Therefore, the areas on the right side of the graph are in the direction of elbow extension from the home position in graphs representing both the paretic and non-paretic results. There is a continual reduction in work area available to the subject as shoulder abduction requirements increase, beginning already between the conditions where they were tested on the haptic table versus when providing 0% of active limb support. The subject is left with less than 0.1 m² at 200% of active limb support. This is only 12% of the area available to him under the table-supported condition. The work area close to the trunk is preserved in the paretic arm across levels of active limb support and actually increased slightly towards the body as abduction requirements increase. Conversely, in the non-paretic side, there is no apparent cost of shoulder abduction in the available work area. The subject generates nearly identical outer path tracings across active support levels, as well as when resting on the haptic table.

To further distinguish the effect of abduction, areas were also normalized within the limb tested. The composite of the 18 subjects' normalized areas is shown in the bar graph in Fig. 5. In all subjects, there is a reduction in work area when they were asked to support larger percentages of their limb weight. Not every subject, however, reached the 200% active support point; in some subjects their available work area had shrunk to zero prior to this level of active support. There is little difference seen in the non-paretic side regardless of support level. However, in the paretic limb there is a clear effect with increasing levels of abduction. The two-factor ANOVA reveals a significant effect of limb ($P < 0.001$) and active support level ($P < 0.001$) on normalized area as well as an interaction effect between limb and active support level ($P < 0.001$). Post hoc testing indicates a significant difference between areas separated by 75% or more of limb support starting at 25% active support condition ($P < 0.05$) with the change reaching lesser *P*-values ($P < 0.001$) starting at 50% of active limb support. The results of this comparison are shown in Table 1.

Joint angles

Elbow and shoulder angles achieved during these trials also change in a systematic way across abduction levels. An example of a shoulder angle-elbow angle plot for the same subject in Fig. 4 is shown in Fig. 6. As expected, the extent of joint excursion utilized also decreases as a function of shoulder abduction torque. Specifically, it can be seen in the figure that there is a large reduction in the elbow extension degree of freedom as active support of the limb increases (Fig. 6). Maximal joint excursions were identified in four directions and summarized for all subjects in Fig. 7. Implementation of a two-factor ANOVA indicates a significant effect of active support level and limb on peak elbow extension ($P < 0.001$) and peak shoulder flexion ($P < 0.001$), but not for peak shoulder extension ($P = 0.69$) and peak elbow flexion ($P = 0.19$). Post hoc testing results for peak elbow extension and peak shoulder flexion are given in Table 1. For these two variables, a larger amount of active support is required between statistically significant levels than the overall work area. The significances in elbow extension and shoulder flexion were due to relative reductions in these degrees of freedom as a function of increases in shoulder abduction (i.e., active limb support); conversely an interesting trend, albeit non-significant, is the increase observed in elbow flexion as a function of active limb support.

Finally, correlation analysis of peak joint angle and work area shows that work area is significantly correlated with reductions in shoulder flexion ($r = 0.811$, $P < 0.001$), elbow extension ($r = 0.876$, $P < 0.001$), and shoulder extension ($r = -0.607$, $P < 0.05$) but not with elbow flexion ($r = -0.389$) range of motion in the paretic limb.

Discussion

This study shows the incremental effects of abnormal joint torque coupling during reaching as a result of increasing levels of required shoulder abduction in the paretic arm of individuals with chronic stroke. A progressively greater expression of the flexion synergy, clinically described as a stereotyped movement pattern which couples shoulder abduction with elbow flexion (Twitchell 1951; Brunnstrom 1970) in the paretic arm, is observed when subjects generate greater shoulder abduction and external rotation torques during reaching. Our results demonstrate that even submaximal levels of shoulder abduction produce deficits primarily in elbow extension and shoulder flexion, consistent with qualitative clinical observations and our previous isometric studies (Dewald et al. 1995; Beer et al. 2000, 2004; Dewald and Beer 2001; Ellis et al. 2007).

Loss of independent joint control in the paretic upper limb

Increased proximal drive of shoulder abductors appears to lead to an increased expression of the flexion synergy at both proximal (shoulder) and more distal (elbow) joints. During lifting tasks, range of motion is compromised in elbow extension and shoulder flexion, while elbow flexion and shoulder extension are either preserved or enhanced as a function of higher levels of background shoulder abduction activation. It stands to question why subjects were able to generate greater areas when gliding on the haptic table than at 0% of required active support. At the 0% active support condition, subjects do not have to generate abduction torques at the shoulder in order to remain elevated off the table. We believe that on the haptic table, subjects are able to take advantage of the table constraint by generating a shoulder adduction to assist in elbow extension, as was found in previous isometric single joint tasks (Dewald et al. 1995). The “extension synergy” is characterized by abnormal coupling between shoulder adduction with shoulder flexion and elbow extension, and is a probable strategy in use to generate larger work areas while performing movement tasks on the rigid haptic table.

Beyond the lack of movement area, there appears to be a systematic difference in the availability of elbow flexion and shoulder extension extent in the paretic limb as compared to the non-paretic limb. Although not statistically significant, these baseline differences may have contributed to the reduced area in the paretic limb on the table compared to the non-paretic limb on the table and may be explained by reduced joint range of motion related to passive tissue changes or joint instability (Lynch et al. 2005), rather than an abduction-dependent impairment. However, strong abduction-dependent reductions in elbow extension and shoulder flexion along with full elbow extension range of motion available on the haptic table indicate a mechanism beyond passive tissue changes or joint instability.

Possible mechanisms for progressive losses in independent joint control

Contribution of weakness—Weakness is often identified as a source of movement impairment following brain injury. Other studies have looked at the effect of impaired limb weakness and the ability to generate multi-joint forces (Mercier et al. 2004), strength imbalances across opposing muscle groups on torque production (Lum et al. 2003), and correlation to function while accounting for dexterity (Canning et al. 2004). Each of these studies evaluates elbow flexion and extension strength isometrically and without regard to surrounding joints; therefore, the relative contributions of the shoulder to movement and torque generation at the elbow cannot be distinguished. Although an overall weakness has been

documented quantitatively in the paretic arm of stroke subjects relative to the non-paretic arm (Dewald et al. 2001), simple weakness of the elbow extensors cannot contribute to the gradual reduction in work area, as none of the induced forces along the vertical act about the axis of elbow joint rotation. A lack of available reaching magnitude, path straightness and smoothness, and joint torque and angular acceleration, specifically toward targets involving elbow extension, have been previously reported however only during actively supported reaching (Beer et al. 2000, 2004, 2007; Kamper et al. 2002). We therefore attribute reductions in work area during active limb support to co-activation of shoulder abductors and elbow flexors.

The results of this report indicate that there is sufficient strength in shoulder flexion and elbow extension to generate a large work area, but only when less shoulder abduction activity is required. However, selective weakness of elbow extensors under higher shoulder loading conditions may also contribute to reductions in work area by amplifying the effects of shoulder abductor/elbow flexor coupling. In our previous isometric studies, higher levels of shoulder abduction inhibited elbow extensors (Ellis et al. 2007). Therefore, we conclude that coactivation of elbow flexors (Dewald et al. 1995) combined with inhibition of elbow extensors (Ellis et al. 2007) is responsible for the reduction of available work area during progressive abductor activation. Although measurement of EMG would be needed to confirm our hypothesis, based on earlier studies we believe that subjects were likely unable to overpower the steadily increasing activation of the biceps with a steadily inhibited triceps as shoulder abduction requirements increased.

Increased reliance on unaffected motor pathways—The progressive nature of work area reductions as a function of required active limb support suggests an incremental recruitment of unaffected neural substrates that are ultimately responsible for reaching range of motion reductions. The findings of this study may be explained by an increased influence of bulbospinal pathways on upper limb control. Specifically, we suggest that an incremental recruitment of bulbospinal pathways occurs as corticospinal resources become exhausted at higher required abduction levels. A functionally linked cortico-reticulospinal system is used in goal directed movements (Matsuyama et al. 2004), and new imaging techniques that allow non-invasive identification of corticofugal fibers in humans show a correlation between lesion location and deficit following stroke in a small group of subjects (Newton et al. 2006). Following brain injury, corticobulbar as well as direct corticospinal projections are often lost, but bulbospinal projections (Kuypers 1964) remain intact. These pathways are less specific than corticospinal projections and project mainly to axial and proximal limb muscles, exhibiting extensive branching and innervating neurons over many spinal segments (Matsuyama et al. 2004). Ipsilateral reticulospinal projections in particular have been proposed to be responsible for the shoulder abduction/elbow flexion patterns persistent across multiple arm configurations (Ellis et al. 2007).

Integration of sensory information is an integral component of movement and online correction. A limitation of this investigation is that we have only tested work area on one plane of movement, and therefore one set of possible sensory feedback. While other studies have looked at reaching tasks that are made within a parasagittal plane or across the body, and also found limitations in available joint movement combinations (Reisman and Scholz 2003), reduced inter-joint coordination (Cirstea and Levin 2000), and temporal deficits (Cirstea et al. 2003), none of these studies looked specifically at kinematic limitations associated with the generation of progressively greater shoulder abduction torques. Results from isometric experiments do indicate however that the flexion synergy is robustly preserved across limb configurations, despite differences in mechanoreceptor feedback (Ellis et al. 2007).

Implications and future research

An individual's degree of recovery is implicated by their work area available over a range of active support levels. This is most apparent in subjects with more severe impairment where work area is dramatically reduced at abduction levels at or below the weight of their arm. When less impaired individuals were asked to lift more than the weight of their limb (comparable to functional tasks such as lifting a book or a gallon of milk), a reduction is seen that is very consistent with that seen in the more impaired subjects at the lower levels of abduction. This is in agreement with findings that less impaired subjects may not exhibit the same patterns during free reaching tasks, but there are aspects of synergistic impairment that are still present (Reinkensmeyer et al. 2002; Reisman and Scholz 2003). When the system is driven more maximally, as with increased abduction requirements in our experiment, typical synergistic patterns emerge even in less impaired subjects despite being qualitatively categorized as not having synergistic movement impairment (Welmer et al. 2006).

We postulate that the emergence of synergistic patterns is directly related to the loss of fiber density in corticofugal and corticospinal tracts, and the associated reliance on bulbospinal tracts following a stroke. The less extensive the loss of fibers in these tracts, the greater the amount of abduction torque the subject will be able to generate before incremental recruitment of bulbospinal tracts occur, resulting in the graded appearance of the flexion synergy. This same logic follows in the reverse for a more extensive loss of fiber density. Future research is planned to measure fiber density in these tracts through imaging techniques such as diffusion tensor imaging (DTI) in order to determine the relationship between work area and corticofugal tract fiber density.

We have established previously that spontaneous joint torque coupling can be altered with a targeted isometric intervention (Ellis et al. 2005). Our future research will employ the ACT^{3D} in quantifying reaching impairment before and after a dynamic rehabilitation protocol designed to directly target abnormal torque coupling during reaching exercises (administered by the ACT^{3D}). We will also employ the ACT^{3D} to quantify abnormal joint torque coupling before and after pharmacologic applications that are currently being employed in an effort to directly identify and discriminate between contributions of brainstem and spinal cord to upper extremity movement following stroke.

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Fig. 1. ACT^{3D} setup: orientation of the subject and the ACT^{3D}. Changing the forearm-hand orthosis and the relative orientation of the subject to the robot allows both arms to be studied in the same setup. The line drawing shows the subject in the initial position used for location of the shoulder and configuration of the feedback

Fig. 2. Subject seated in the ACT^{3D} system. His trunk is secured by straps and the arm is attached to the HM with the lightweight forearm-hand orthosis. He is looking at the computer monitor for visual feedback, shown in Fig. 3

Fig. 3.

Example of the visual feedback a subject would receive when their left arm is being tested. In the same configuration as the subject's limb, the screen avatar shows joint movement and position with respect to the goal and the *dark gray* table surface. The *black* arc represents the longest reach they are able to make, as measured by adding the three limb segments and the *light gray* tracing shows the path of the 'fingertip' endpoint

Fig. 4. Envelope abilities during various levels of limb support in the left, paretic limb (a) of a single subject, inverted for comparison to the non-paretic limb shown in (b). Axes units are in meters, and an individual's outline is provided in the non-paretic (*right*) side for reference

Fig. 5.

Comparison of non-paretic and paretic limb work areas, normalized to the table supported condition within the arm measured revealed a significant difference between limbs for all levels of support. Bars represent standard deviations (* $P < 0.05$; ** $P < 0.001$; $n = 18$)

Fig. 6. Shoulder-elbow angle plot for the paretic limb of the subject shown in Fig. 4. As the percentage of active support increases, there is an overall decrease in elbow extension capabilities, specifically when the subject is flexing their shoulder at the same time (upper right portion). Both joints show a reduction in range as shoulder abduction requirements increase, with significant reductions in the direction of elbow extension and shoulder flexion

Fig. 7.

Maximal joint excursions as a function of limb support, with trend-lines across abduction levels. In each graph, the non-paretic limb (shown in a *dashed lighter gray*) remains relatively unchanged across active limb support requirements. By contrast, the paretic limb (shown in *solid black*) is affected, most strikingly in the elbow extension (a) and shoulder flexion (c) degrees of freedom

Table 1

Post hoc comparisons, paretic limb

	0	25	50	75	100	125	150	175	200
0	--			*	**	**§	**§	**§	**§§
25		--				*	**	**	**§
50			--				*	**	**§
75	*			--				*	**
100	**				--				*
125	**§	*				--			
150	**§	**	*				--		
175	**§	**	**	*				--	
200	**§§	**§	**§	**	*				--
	++	+	+						

Area: (*p<0.05; **p<0.001)
 Elbow Extension: (§p<0.05; §§p<0.001)
 Shoulder Flexion: (+p<0.05; ++p<0.001)