

# **CAN ROBOTS IMPROVE ARM MOVEMENT RECOVERY AFTER CHRONIC BRAIN INJURY? A RATIONALE FOR THEIR USE BASED ON EXPERIMENTALLY IDENTIFIED MOTOR IMPAIRMENTS**

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## **ABSTRACT**

Significant potential exists for robotic and mechatronic devices to deliver therapy to individuals with a movement disability following stroke, traumatic brain injury, or cerebral palsy. We performed a series of experiments in order to identify which motor impairments should be targeted by such devices, in the context of a common functional deficit – decreased active range of motion of reaching – after chronic brain-injury. Our findings were that passive tissue restraint and agonist weakness, rather than spasticity or antagonist restraint, were the key contributors to decreased active range of motion across subjects. In addition, we observed striking patterns of abnormal contact force generation during guided reaching. Based on these results, we suggest that active assistance exercise is a rational therapeutic approach to improve arm movement recovery after chronic brain injury. We briefly discuss a simple, cost-effective way that such exercise could be implemented using robotic/mechatronic technology, and how such exercise could be adapted to treat abnormal muscle coordination.

## **BACKGROUND**

Recently there has been a surge of interest in bringing robotic and mechatronic technology to bear on rehabilitation of movement after brain injury [1]. Stroke is currently the leading cause of severe disability in the U.S., and arm and hand movements are often preferentially impaired after stroke. A significant amount of recent research has therefore been focused on devices for therapy of the arm after stroke. Such devices could ultimately benefit approximately 300,000 new stroke survivors per year, as well as the more than 1.5 million chronic stroke survivors with movement disability in the U.S.

A current difficulty in designing appropriate robotic technology for movement therapy of brain-injured individuals is that the optimal therapy techniques are unknown. More fundamentally, it is unclear what induces the observed movement impairments. Brain injury is often accompanied by a series of motor impairments, including weakness, spasticity, impaired movement range and impaired motor coordination. These impairments are mediated, in

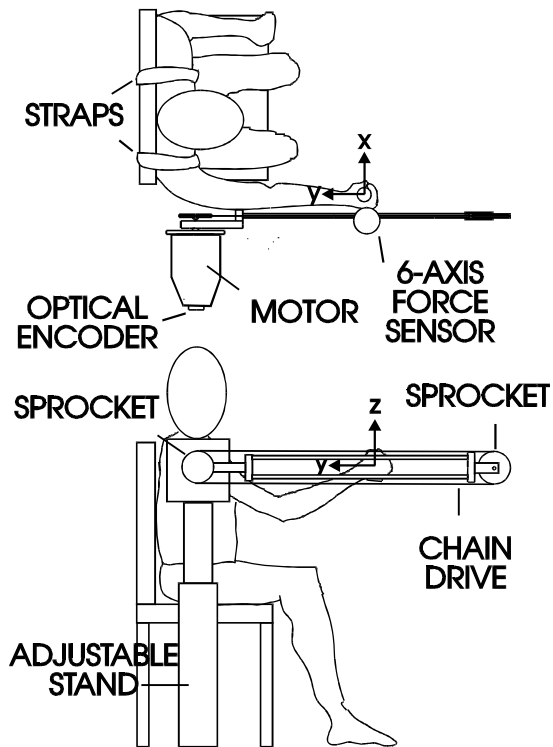
part, by changes to neural pathways, reflex systems, muscle, and connective tissue. Physical rehabilitation – and robotic therapy devices – could be targeted at any of these impairments.

The goal of this study was therefore to identify the role of three motor impairments to a common functional deficit – decreased active range of motion of reaching (or decreased active “workspace”). Briefly, the three impairments were:

1. *Increased passive tissue restraint*, which may arise due to disuse and persistent abnormal posture of the spastic arm [2], and could cause an increased resistance to voluntary movement of the arm.
2. *Antagonist muscle restraint*, which could arise from reflex activation of antagonists (spasticity), or abnormal antagonist coactivation [3].
3. *Agonist muscle weakness*, arising from destruction of key motor centers and outflow pathways and potentially by disuse atrophy [4].

## METHODS

To distinguish these three motor impairments, detailed mechanical measurements were made of the arms of five spastic hemiparetic subjects during reaching along a motorized guide. The device, which was used in the configuration shown in Fig. 1, allowed measurement of hand position and multi-axial force generation during guided reaching movements in the horizontal plane, and application of



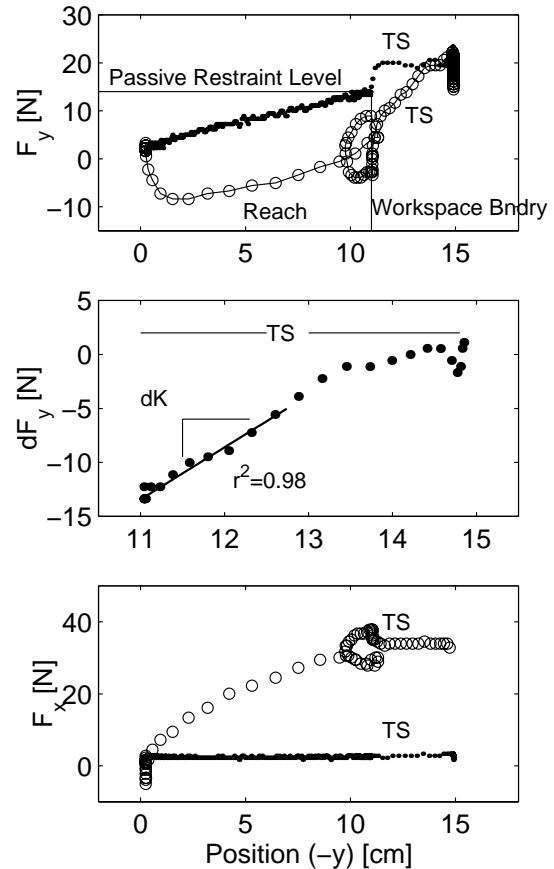
**Figure 1:** The Assisted Rehabilitation and Measurement Guide (“ARM Guide”). The subject’s forearm/hand was attached to a handle/splint that slid along a linear constraint via a low-friction, linear bearing. A six-axis force/torque sensor sensed contact forces between the hand and the constraint in the coordinate frame shown. A computer-controlled motor attached to a chain drive was used to drive the hand along the constraint. An optical encoder measured the position of the hand along the constraint.

motorized stretches to the arm. After establishing workspace deficits along the device by the subjects, two tests were performed to elucidate the causes of these deficits. Each test was applied following individual reaches by each subject, across a set of twelve reaches:

**Passive Restraint Test:** To evaluate the level of passive tissue restraint at the workspace boundary, the ARM Guide

returned the subject's hand to the position from which the most recent reach was initiated. The arm was then moved slowly ( $< 4$  cm/sec) back to the workspace boundary achieved by the most recent reach, and the force needed to hold the passive arm at the boundary was measured (Fig. 2, top). For comparison, the passive force generated by the contralateral arm (which was ostensibly normal) at a matched position was also evaluated. During these slow passive movements, EMG recordings of seven muscles surrounding the shoulder and elbow were used to verify that muscles were inactive.

**Active Restraint Test:** We hypothesized that any active restraint arising from activation of antagonist muscles during reaching would manifest itself as an increased stiffness following reaching, while the subject was still activating muscles and trying to move beyond the boundary. To evaluate this stiffness, a small stretch (the "terminal stretch", 4 cm amplitude, bell-shaped velocity trajectory with a peak velocity of 15 cm/sec) was applied to the arm when hand velocity had dropped and remained below 1 mm/sec for 150 msec. An identical small stretch was applied following the slow passive movement of the arm through the same range (Fig. 2 top). The restraint force measured following the passive movement was then subtracted from the restraint force measured following reaching, in order to subtract out any



**Figure 2:** *Top:* Example of force measured along ARM Guide in y direction (see Fig. 1) during an active reach with a spastic arm (open circles), and during a slow passive movement through the same range (filled circles). Each movement was followed by an identical 4 cm terminal stretch (labeled TS). *Middle:* Expanded view of differential force (i.e.  $F_y$  for TS following following reach minus  $F_y$  for TS following passive movement.)  $dK$  = active stiffness of arm. Regression to find  $dK$  was performed only over first 200 msec to minimize possible effects of voluntary intervention by subject. *Bottom:* Horizontal off-axis force during reach (open circles) and during passive movement (filled circles).

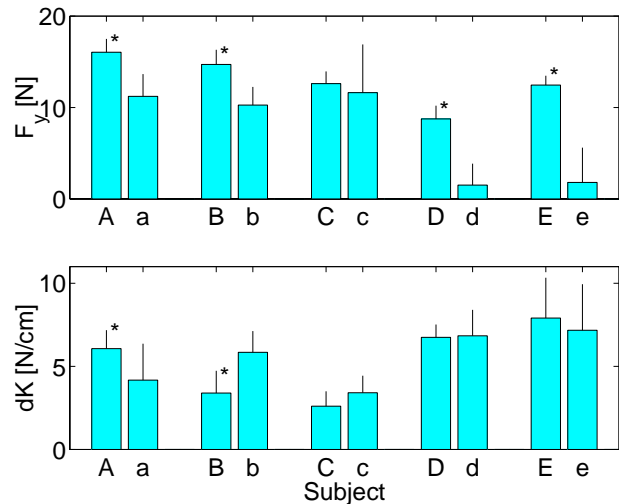
passive forces common to the two conditions, such as those arising from passive stiffness, inertia, and damping.

The result was the restraint force due solely to coactivation of muscles at the workspace boundary (Fig. 2 middle). For comparison, the terminal stiffness of the contralateral arm following matched, targeted, reaching movements, and following slow passive movement through the same range, were evaluated in a similar fashion.

Five subjects were tested, each having suffered a hemispheric brain injury (four ischemic stroke, one traumatic brain injury) at least two years previously. The subjects had a wide range of movement ability as gauged by a standard clinical exam. The two subjects with the greatest movement ability exhibited workspace deficits during free movement, yet had a full active range of motion during reaching along the ARM Guide. To induce a workspace deficit along the ARM Guide, these subjects (D and E) were loaded with a light spring load (stiffness 2.5 N/cm). All subjects had mild to moderate spasticity in elbow flexor muscles as detected manually.

## RESULTS

All subjects showed highly repeatable active range of motion as they reached along the ARM Guide: the standard deviation of the final hand resting position was less than 1.5 cm for all subjects, while mean movement amplitudes ranged from 7.0 to 16.0 cm across subjects. The well-defined limit to active range of motion occurred well before the end of the passive range of



**Figure 3: Top:** Passive restraint force for subjects A – E at the workspace boundary. upper case = spastic arm; lower case = contralateral arm **Bottom:** Active stiffness at the workspace boundary. Asterisks denote significant difference between spastic and contralateral arms (*t*-test,  $p < .05$ ). Bars = 1 SD.

motion of the arm. Specifically, the subjects' arms stopped moving at least 7.0 cm before the mechanical limit to passive range of motion determined manually by the experimenter. Thus, the cause of the workspace boundary was not a passive mechanical limit to either elbow extension or shoulder flexion.

A striking feature of force development during reaching was that all subjects generated large, perpendicular forces against the ARM Guide with the spastic arm. The forces were greatest in the horizontal plane, were medially directed, and reached a maximum near the end of the range of motion (Fig. 2 bottom). For all subjects, the horizontal contact force at the end of the range of

motion was significantly more medial by more than 20.0 N than the horizontal force generated by the contralateral arm (one-sided t-test,  $p < .0001$ ). We have shown previously [5] that such medial contact force generation is consistent with clinical descriptions of the abnormal extension muscle synergy (i.e. elbow extension coupled with shoulder internal rotation and adduction).

### ***Mechanical Tests to Determine Origins of Workspace Deficits***

Interspersed with reaches to the workspace boundary, two mechanical tests were performed on each subject's arm (Fig. 2). For the passive restraint test, the subject's relaxed arm was slowly moved to the workspace boundary achieved by the previous reach. For four of five subjects, the level of passive restraint force generated by the spastic arm at the workspace boundary was significantly greater than the restraint force generated by the contralateral arm at a matched position (Fig. 3 top, t-test,  $p < .05$ ). The average increase across subjects was 4.6 N (SD 0.8).

For the active restraint test, a terminal stretch was applied to the arm immediately following reaching, and compared to a terminal stretch following slow passive movement through the same range. For all subjects, the difference between the restraint force in the two conditions, plotted as a function of hand position, was well approximated by a linear

relationship (Fig. 2 middle). The mean variance accounted for by linear regression of this relationship across all subjects was 0.86 (SD 0.05) for the spastic arms, and 0.85 (SD 0.10) for the contralateral arms. As judged by the slope of the differential force response, the stiffness of the impaired arm following reaching was increased by an average of 5.3 N/cm (SD 2.3) across subjects compared to arm stiffness following passive movement (Fig. 3). Similarly, arm stiffness increased in the contralateral arm following matched reaching movements as compared to following passive movement by an average of 5.5 N/cm (SD 1.6). These differences were significantly different from zero (t-test,  $p < .001$ ), but not from each other. On a subject-by-subject basis, only one subject showed a statistically greater active stiffness in the spastic arm.

### **DISCUSSION AND CONCLUSION**

The increased passive tissue restraint we measured most likely resulted from disuse of the spastic arm. Muscle, tendon, and joint capsules tend to shorten and stiffen when held in a shortened position for an extended time period [2]. Since spastic hemiparetic patients often have difficulty moving their arm across the full workspace, and typically decline to use the spastic arm in favor of the contralateral arm, one would expect to observe changed passive tissue properties. Such changes have been frequently observed in the lower extremity after brain injury [e.g.

6], and have been suggested to occur at the elbow [7].

The finding that active stiffness of the spastic arms was comparable to that of the contralateral arms was surprising. All subjects had clinically detectable spasticity in their elbow flexor muscles. Also, all subjects exhibited gross patterns of abnormal muscle coactivation during reaching, as witnessed by the generation of large off-axis contact forces. Despite these possible indicators of antagonist restraint, however, the stiffness measurements demonstrated that the net effect of reflex-based antagonist activation and abnormal antagonist coactivation was not excessive, compared to antagonist levels during normal movement (i.e. with the contralateral arm).

### ***A Rationale for Robotic Therapy***

Based on these results, we suggest that a rational plan for treating workspace deficits in chronic brain injury is to target agonist weakness and passive tissue restraint. Robotic therapy devices could help implement such treatment by providing active assist exercise. The principle of active assist exercise is to complete a desired movement for the patient if the patient is unable. The effect of such exercise is to interleave repetitive movement attempts and passive range of motion exercise. Repetitive movement exercise, in which an individual attempts repeatedly to activate damaged motor pathways, has

shown promise in improving agonist strength in the hand [8]. Passive range of motion exercise, in which shortened soft tissues are extended and held in a lengthened position, can help alleviate passive tissue restraint [2]. By interleaving these two exercises via active assistance, robotic therapy devices could address both passive tissue restraint and agonist weakness in a single, efficient exercise.

The reaching guide used in this study provides an example of a simple, cost-effective means to provide active assist therapy for reaching movements across the user's workspace. The device makes use of a passive linear constraint to guide movement along desired straight-line reaching trajectories. The passive constraint can be moved and locked to allow reaching in different directions across the workspace. Thus, only a single actuator is required to assist reaching in a wide variety of directions.

A final consideration is the abnormal coordination patterns we observed in the subjects. Mechanically completing a movement for a person may encourage use of abnormal muscle synergy patterns, since the person may develop more force for reaching when using the pattern, and since any misdirected (i.e. off-axis) forces will be counteracted by the mechanical assistance. Incorporating feedback of off-axis force generation during guided reaching may enhance development of coordinated movement. One approach is to provide

visual or auditory feedback of off-axis contact forces. Another approach is to reduce the stiffness of the guiding mechanism, so that if a user exerts large off-axis forces, the arm will deviate from the desired reaching path.

**Acknowledgements:** The authors gratefully acknowledge support of NIDRR Field-Initiated Grant H133G80052, and Whitaker Foundation Biomedical Eng. Research Grant to DJR.

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