

# Motions or muscles? Some behavioral factors underlying robotic assistance of motor recovery

Neville Hogan, PhD;<sup>1–2\*</sup> Hermano I. Krebs, PhD;<sup>1,3</sup> Brandon Rohrer, PhD;<sup>1</sup> Jerome J. Palazzolo, PhD;<sup>1</sup> Laura Dipietro, PhD;<sup>1</sup> Susan E. Fasoli, ScD;<sup>1</sup> Joel Stein, MD;<sup>4</sup> Richard Hughes, PT, NCS;<sup>4</sup> Walter R. Frontera, MD, PhD;<sup>4</sup> Daniel Lynch, OTR/L;<sup>5</sup> Bruce T. Volpe, MD<sup>3,5</sup>

<sup>1</sup>Newman Laboratory, Department of Mechanical Engineering and <sup>2</sup>Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology, Cambridge, MA; <sup>3</sup>Department of Neurology and Neuroscience, Weill Medical College of Cornell University, White Plains, NY; <sup>4</sup>Department of Physical Medicine and Rehabilitation, Spaulding Rehabilitation Hospital and Harvard Medical School, Boston, MA; <sup>5</sup>Burke Medical Research Institute, White Plains, NY

Abstract-Robotics and related technologies have begun to realize their promise to improve the delivery of rehabilitation therapy. However, the mechanism by which they enhance recovery remains unclear. Ultimately, recovery depends on biology, yet the details of the recovery process remain largely unknown; a deeper understanding is important to accelerate refinements of robotic therapy or suggest new approaches. Fortunately, robots provide an excellent instrument platform from which to study recovery at the behavioral level. This article reviews some initial insights about the process of upper-limb behavioral recovery that have emerged from our work. Evidence to date suggests that the form of therapy may be more important than its intensity: muscle strengthening offers no advantage over movement training. Passive movement is insufficient; active participation is required. Progressive training based on measures of movement coordination yields substantially improved outcomes. Together these results indicate that movement coordination rather than muscle activation may be the most appropriate focus for robotic therapy.

# **INTRODUCTION: RECOVERY AS LEARNING**

In our work on robotic rehabilitation, we have chosen to emphasize stroke as it continues to be the leading cause of disability: in fact, recent studies report an increase in its prevalence [1–2], a trend likely to continue because of increasing life expectancy, aging of the "baby boom" generation, and improved medical treatment that increases stroke survivability. Ninety percent of stroke survivors are left with significant impairment and require therapy. Motor deficits persist chronically in about half of stroke survivors [3], and sensorimotor rehabilitation is the most promising application for robotics technology.

**Key words:** active-assistance therapy, adaptation, cerebrovascular accident, hemiparesis, motor learning, passive-resistance therapy, rehabilitation, robotic therapy, submovements, upper limb.

**Abbreviations:** CIMT = constraint-induced movement therapy, CPM = continuous passive motion, FM = Fugl-Meyer, MSS = Motor Status Scale, PBPT = performance-based progressive therapy, PM = performance measure, ROM = range of motion. \*Address all correspondence to Neville Hogan, PhD; Department of Mechanical Engineering, Massachusetts Institute of Technology, Room 3-146, 77 Massachusetts Ave, Cambridge, MA 02139; 617-253-2277; fax: 617-258-7018. Email: neville@mit.edu

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A common assumption is that sensorimotor therapy<sup>\*</sup> works by helping patients "relearn" motor control. Though intuitively sensible, this notion may need to be refined. First, normal motor learning does not have to contend with the neuromuscular abnormalities that are common sequelae of neurological injury, including spasticity, abnormal tone, disrupted or unbalanced sensory pathways, and muscular weakness. Though stroke is essentially a lesion of the central nervous system, these deficits appear to involve the peripheral nervous system and might suggest that muscles should be the focus of therapy. Nevertheless, central nervous system plasticity appears to underlie recovery. Thus recovery may resemble motor learning in some respects, but it is likely to be a more complex process.

Second, normal motor learning is far from fully understood. Topics of ongoing vigorous debate include questions such as: What variables or parameters of action does the brain command and control? How are these encoded and represented in the brain? How are these encodings or representations acquired and retained? What training schedule optimizes acquisition? Is a period of consolidation between training sessions (e.g., sleep) required for long-term retention? These deep questions have practical relevance for therapy. For example, if the brain represents action as a sequence of muscle activations, focusing sensorimotor therapy on muscles would seem profitable. However, a large and growing body of evidence (briefly reviewed here) indicates that under many circumstances the brain does not directly control muscles: instead, it controls the upper limb primarily to meet kinematic specifications (such as a simple motion of the hand in a visually relevant coordinate frame) that adjust muscle forces to compensate for movement-by-movement variation of mechanical loads, which suggests that focusing sensorimotor therapy on motions rather than muscles may be more profitable. Of course, these are only two of a large number of possible therapy variations. In our research on robotic stroke rehabilitation, we have attempted to assess some of these possibilities.

# WEAKNESS

Upper-limb weakness after stroke is common and results in substantial disability. Motor outcomes following conventional treatment are poor, with 30 to 66 percent of those who receive conventional therapy not able to use their paretic arm functionally [4-5]. Gowland stated that only 5 percent of persons who receive intensive therapy for severe upper-limb weakness poststroke regain functional use of their paretic upper limb during rehabilitation [4]. Several approaches to provide upperlimb exercise after stroke have been studied, including constraint-induced movement therapy (CIMT) [6-10]. While CIMT appears to be a promising therapy, it requires a significant level of residual motor function and is not feasible for individuals with more severe weakness after stroke. Alternative approaches are needed for the large number of people who are unable to achieve this threshold level of motor function at the initiation of therapy. Robotic therapy can provide therapeutic exercise to stroke survivors with a broader range of motor impairments: assisted exercises for individuals with severe weakness as well as resistive exercises for stroke survivors with greater motor abilities.

Robot-aided rehabilitation reduces upper-limb motor impairment when provided early after stroke [11–13] and also when provided to persons with chronic, stable deficits after stroke [14–16]. However, the optimal exercise regimen for improving motor function after stroke remains to be determined. Evidence suggests that progressiveresistance (strength) training of the lower limbs may provide functional benefits [17–18]. Studies of upper-limb resistive exercises after stroke have examined finger strengthening rather than more proximal upper-limb resistance therapy [19–20]. To better understand the role of muscle weakness and to what extent it may be ameliorated by exercise, we conducted a study to evaluate the effects of incorporating resistive exercises into robotic therapy [21].

# **PROGRESSIVE-RESISTANCE EXERCISE**

Our primary hypothesis was that combining progressiveresistance exercises with robotic therapy would result in a greater improvement in motor control than activeassistance robotic therapy alone. The latter consisted of horizontal reaching tasks toward computer-generated virtual targets displayed on a monitor. The targets were

<sup>\*</sup>In this article, the term "sensorimotor therapy" is intended to encompass physical and occupational therapy, especially those aspects that robotic technologies might conceivably contribute to.

arranged in a circular array, and reaching tasks proceeded clockwise from each target to the next. If a subject was unable to reach a target independently, the robot (InMotion2, Interactive Motion Technologies, Cambridge, Massachusetts) provided assistance to reach it. For subjects able to reach the target independently, the robot provided guidance, gently opposing inappropriate (lateral) motions not directed toward the target.

Robotic progressive-resistance therapy was of the same form as active-assistance therapy, except that the robot was programmed to provide resistance to the desired movement. The amount of resistance was determined and modified by a control algorithm that used robotic measures of the subject's muscle strength to increase or decrease the effort required to reach the targets. These measures were obtained at the end of each treatment session to determine the amount of force to be delivered by the robot during the next session. A maximum force of 28 N was provided by the robot as resistance during therapy exercises. The number of repetitions of the task was the same for subjects receiving active-assistance therapy and those receiving resistance therapy [21].

After giving their informed consent, subjects underwent a battery of assessments performed by a single therapist not otherwise involved in the study who was blinded to the subjects' group assignments. We performed serial assessments at study enrollment, after 2 weeks, and after 4 weeks to establish a reliable and stable baseline for motor function. A robot-based assessment determined eligibility for progressive-resistance therapy. Robotic therapy began after the completion of the assessments and consisted of three 1-hour sessions a week for 6 weeks.

Subjects able to participate in progressive-resistance therapy were randomized to receive either active-assistance robot-aided exercises or progressive-resistance robotic therapy. Subjects who were unable to participate in resistance therapy underwent active-assistance robotic therapy and were again screened for eligibility after 3 weeks of robotic therapy. After 3 weeks, those subjects able to participate in progressive-resistance therapy were then randomized to receive either progressive-resistance therapy or to continue with active-assistance therapy.

Forty-six subjects at least 1 year poststroke completed the 6-week treatment protocol. The main result was that although subjects in all groups showed improvement in measures of motor control (mean increase in Fugl-Meyer [FM] score of 3.3, 95% confidence interval 2.2 to 4.4) and maximal force (mean increase in maximal force 3.5 N, p = 0.03) over the course of robotic therapy, no significant differences in outcome measures were found between subjects who received progressive-resistance therapy for all or part of the therapy program and the matched active-assistance therapy subjects [21].

The absence of any difference between groups receiving progressive-resistance therapy and active-assistance therapy may simply mean that this robotic form of progressive-resistance exercise was not optimal in terms of duration, repetition, or intensity; for example, one limitation of this study was the relatively modest amount of resistance provided by the robot (for safety reasons). However, while larger resistive forces might evoke greater benefits, persons with deficits comparable to our study population are unlikely to overcome substantially larger resistive forces. Instead, the fact that both types of robotic therapy yielded modest increases in maximal force (13.7% from baseline to study completion) suggests that active-assistance therapy may be as effective as progressive-resistance exercise for ameliorating upperlimb weakness in this population.

The gains in force generation primarily occurred during the first 3 weeks of therapy, without further significant increases during the second 3 weeks of therapy. This finding is in contrast to the results for motor impairment, where the improvements in FM scores seen were evenly distributed between the first and second 3 weeks of therapy. This is consistent with the finding in neuroscience research that motor control is organized hierarchically, with muscle force production being subordinate to kinematic coordination (hence a recovery of force generation alone would not ensure a recovery of motor coordination), but further investigation is needed to distinguish this result from other possible interpretations.

# **CONTINUOUS PASSIVE MOTION**

As the recovery of kinematic coordination appears to be an important component of recovery, to what extent may it be achieved by passive limb exercise? In the clinic, passive range of motion (ROM) exercise is a standard part of treatment and is considered effective at preventing contractures [22]. Recent clinical experiments demonstrated that passive movement altered the inhibitory state of the central nervous system and subsequently affected behavioral responses [23]. In these experiments, subjects who received passive rhythmic flexion and

extension (i.e., movements of the wrist systematically delivered by a passive movement apparatus) were found to have disinhibited local cortical regions that were independent of spinal cord activity [23–24]. In other work that concentrated on the influence of gamma-aminobutryic acid neurotransmission, practice-dependent plasticity altered cortical activity to favor improved motor performance [25]. Accordingly, we tested in patients with stroke and a paretic upper limb whether the addition of a daily treatment session with a device that moves the upper limb passively would alter motor outcome, spasticity, shoulder joint integrity, pain, and disability [26–27].

We used a commercial continuous passive motion (CPM) device (Shoulder 600, OrthoLogic, Tempe, Arizona) to mobilize the glenohumeral joint repetitively and reproducibly, while avoiding extremes of shoulder joint excursion. Patients consecutively admitted to a poststroke rehabilitation unit were screened for inclusion, which required a single image-verified first stroke within 3 weeks and significant motor impairment of the arm. Thirty-two subjects met the criteria and gave informed consent to an approved protocol that randomly assigned them to receive an extra daily treatment of CPM or occupational group therapy in addition to the standard poststroke therapy that all patients received. For the CPM treatment, the patient sat upright in the chair to which the device was attached so that the axis of the CPM device shoulder motor was aligned with the patient's shoulder, while the patient's arm was supported by a rigid padded brace. The daily treatment period (5 days a week) lasted 25 minutes and combined shoulder elevation, abduction, and external rotation. Control subjects received an extra 25-minute occupational group therapy session daily (5 days a week) that included a standard regimen of stretching and mobility exercises.

Both groups began with severe flaccid hemiparesis, but the treatment group was younger and less impaired, although the impairment motor scores were not significantly different. Complete details of the attempt to control for group imbalance are presented elsewhere [27]. The main result showed that the change in motor impairment as measured by the FM for the upper limbs was equivalent for CPM-treated and control subjects. Data are presented as mean  $\pm$  standard error of the mean unless otherwise noted. CPM-treated patients had slightly higher FM on admission (7.5  $\pm$  1.7) compared with control subjects (5.2  $\pm$  1.5) and less change by discharge (44% change compared with 77% change for control subjects; discharge FM = 10.9  $\pm$  1.1 for CPM subjects; discharge FM =  $8.9 \pm 1.2$  for control subjects). These changes were not significant (p = 0.26).

Other measures, particularly of joint stability [27], suggest that the daily CPM treatment was of sufficient intensity and duration  $(22.0 \pm 1.0 \text{ days})$  to yield measurable results. In fact, a trend was noted indicating that joint stability improved more for the CPM-treated group than for control subjects. The fact that no significant effects on motor outcome were noted suggests that passive motion is not sufficient. Given that interactive robotic therapy produced a significant effect on motor outcome [11–16], the conclusion seems inescapable: recovery requires active participation.

# **ACTIVITY-DEPENDENT PLASTICITY**

The importance of active participation is consistent with what is known about the neurobiology of recovery after neurological injury. Candidate mechanisms include recovery of undamaged brain from functional inactivation caused by the damage, activation of undamaged regions of brain in the opposite hemisphere, and reorganization of synaptic connections. Functional brain imaging in patients recovering and recovered from stroke [28-32] showed increased blood flow in areas around the lesion. Recovery of function depends in part on the postinjury experience, and plasticity or reorganizational potential may be enhanced by activity. Enhanced movement therapy for the paretic arm of recovering stroke patients led to significant regional cerebral blood flow improvements compared with those receiving standard care [33]. Work with animal models also indicates that training enhances recovery after central nervous system damage. After focal cortical injury, animals exposed to enriched or challenging sensorimotor environments registered greater anatomical responses during recovery and improved eventual functional outcomes [34–35]. Though many details of the biology of recovery are unknown or controversial, the combination of clinical and animal studies indicates that motor activity of appropriate structure and intensity enhances or guides a neuroplastic recovery process after brain injury.

An underlying activity-dependent neural plasticity is probably a key mechanism through which robotic therapy produces clinical benefits. On that basis, recovery should resemble motor learning. One distinctive feature of motor learning is its specificity: motor learning usually does not generalize broadly. Combining the results of two trials

with 96 acute-phase inpatients [11,13,36], we found a statistically significant change in Motor Status Scale (MSS) and Medical Research Council motor power scores that favored patients receiving robotic therapy. However, though the MSS score for shoulder and elbow reached significance (which demonstrates that this instrument can detect the effect of robotic therapy), the wrist and finger MSS score showed no significant difference between experimental and control groups. This result was almost certainly because our initial form of robotic therapy exercised the shoulder and elbow but not the wrist and fingers.

To the extent that the benefits of robotic therapy do not generalize broadly but are specific to the muscle groups and/or limb segments exercised, recovery resembles motor learning. However, the interpretation of this statement requires care. The assumption that a key benefit of robotic therapy derives from its intensity and the large number of repetitions it affords seems reasonable; learning requires practice, practice, practice. However, the form of practice matters. We have found that at least one form of robotic therapy can achieve greater benefits with fewer repetitions.

# PERFORMANCE-BASED PROGRESSIVE THERAPY

Given the apparent importance of a patient's active participation in therapy, we revised our robot control algorithm to test whether continuously challenging a patient would enhance recovery. The revised algorithm differs from our earlier sensorimotor therapy in three important ways.

First, during our earlier clinical trials, robotic therapy took the form of fixed, repetitive reaching exercises cued by a video display. An impedance controller with constant stiffness and damping made the therapy interactive: the force exerted by the robot varied continuously as a function of the deviation of the patient's motion from a minimum-jerk trajectory of constant duration that connected the start position to the goal position. This system suited patients with limited motor ability for whom it provided assistance; however, it would also impede patients who moved faster than the nominal trajectory. Our revised algorithm used nonlinear impedance control to implement a "virtual slot" that extended between the start and goal positions and defined the appropriate coordination. Lateral deviation from the desired path was discouraged by the stiffness and damping of the slot sidewalls. Desired motion was assisted by moving the back wall of the slot along a minimum-jerk virtual trajectory so that the slot progressively "collapsed" to a "virtual spring" centered on the goal position. However, motion along the "virtual slot" (well-aimed and faster than the nominal desired trajectory) was unimpeded.

A request for the subject to move was signaled by a target in the visual display changing color. If the patient failed to trigger the robot within 2 seconds, the robot began to act (i.e., the back wall of the "virtual slot" closed on the goal position). To trigger the robot, the patient had to move the handle (in any direction) at a speed above a modest threshold value. Even severely impaired patients with a paretic arm could trigger the robot. Although trunk motion was discouraged by restraining seat belts, in practice, sufficient trunk motion to move the handle and trigger the robot was possible. Subjects were given no particular instruction but to try to reach the target. Though ultimately inappropriate trunk motion is to be discouraged [37], this mode of triggering the robot encouraged severely impaired patients to participate actively, rather than passively allowing the robot to drive the arm.

Second, the revised algorithm continuously monitored the patient's performance. By combining records of the kinematics of actual patient motion and the kinetics of mechanical interaction between robot and patient, four performance measures (PMs) were computed: PM1 graded patients' ability to initiate movement, PM2 measured movement speed, PM3 measured aim or coordination, and PM4 measured movement range or extent. We used these measures to adjust the parameters of the controller during a therapy session. For the first five cycles of movements to the eight goal positions and back to the center position, the time allotted for a movement (the duration of the nominal minimum-jerk trajectory) and the stiffness (impedance) of the "virtual slot" sidewalls were adjusted approximately to match the patient's current performance and need for guidance, which was important because patient performance typically declined between the end of one therapy session and the beginning of the next. For every subsequent three cycles of reaches to and from the eight goal positions, the controller parameters were adjusted based on the patient's performance and its variability during the previous batch of moves. The intent was to challenge the patient to improve while compensating, if necessary, for lapses in performance.

As patients aimed better, the stiffness of the "virtual slot" sidewalls was decreased (and vice versa). As patients moved faster, the time allotted for movement was decreased (and vice versa). The speed threshold to trigger the robot was also adjusted to 10 percent of the peak speed of a minimum-jerk trajectory of that duration. Consequently, if nominal movement duration increased, the speed of motion required to trigger the robot decreased (and vice versa). Thus, the motor ability required to trigger the robot and move to the target was less demanding for more impaired patients and more demanding as performance improved. Again, this was intended to encourage active participation of even the most impaired patients, yet continuously challenge patients as they recovered.

Third, to provide motivation, positive reinforcement, and knowledge of results, our revised algorithm provided specific, movement-related feedback in the form of a simple graphical display that consisted of four vertical bars for which height and color changed to reflect recent patient performance. The height of each of the four bars was determined by the four PMs, expressed as a percentage, and scaled relative to the score achieved at the end of the initial five cycles of reaches so that patients nominally scored between 70 and 90 percent of maximum as performance changed throughout a therapy session. The intent was to avoid discouraging patients who could not yet move well without boring patients who could.

This performance-based progressive-therapy (PBPT) algorithm provided support for patients to progress from complete plegia to normal arm movement. PM1, which measured ability to initiate movement, was probably most important for severely impaired patients and helped to ensure appropriate timing of afferent and efferent signals, which may be important for reestablishing the excitability of corticospinal projections [38–42]. PM4 measured active ROM, an important clinical measure of function, but also rewarded hypertonic patients for relaxing their arms, which allowed the impedance controller to move their hands closer to the target. PM2 and PM3, which respectively measured movement speed and aim or coordination, quantified the tradeoff between speed and accuracy that is characteristic of unimpaired movement and probably most important for patients with mild-to-moderate impairment.

The PBPT protocol was evaluated by a clinical study of 30 stroke patients between the ages of 39 and 81 with chronic motor impairment following a single stroke that had occurred between 8 and 95 months before the initial assessment [43]. All patients were evaluated six times: three times in a 2-month period before the start of therapy to assess baseline performance, at the midpoint and discharge from robotic therapy (18, 1-hour sessions of robotic training, 3 times a week for 6 weeks), and finally at a follow-up evaluation session 3 months after robotic training. Evaluating therapists were different from treating therapists.

The first three evaluations showed no significant changes on any of the impairment scales, which verified that subjects were indeed at the chronic phase of their recovery in which no spontaneous improvement is observed. Subsequent evaluations showed that the PBPT protocol evoked a statistically significant improvement in motor performance that was maintained at the 3-month follow-up (**Table**). This result confirms earlier studies of

#### Table.

Motor impairment outcomes of performance-based progressive robotic therapy.

-	*	Impairment Measure			
Severity*	Assessment	Fugl-Meyer Shoulder/ Elbow Component <sup>†</sup> (Max = 42)	Percent Change	Medical Research Council Motor Power <sup>†</sup> (Max = 70)	Percent Change
Moderate	Pretreatment	$17.0 \pm 1.3$		$37.2 \pm 2.5$	
( <i>n</i> = 12)	Posttreatment	$22.5 \pm 1.3^{\ddagger}$	32	$45.4\pm1.7^{\ddagger}$	22
	3 mo follow-up	$24.5\pm0.9^\ddagger$	44	$46.5 \pm 1.9^{\ddagger}$	25
Severe	Pretreatment	$8.2 \pm 0.7$	—	$17.3 \pm 1.8$	_
( <i>n</i> = 16)	Posttreatment	$10.9\pm0.9^{\ddagger}$	33	$23.7 \pm 2.0^{\ddagger}$	37
	3 mo follow-up	$12.5\pm0.9^{\ddagger}$	52	$26.3\pm2.2^\ddagger$	52

\*Moderate defined by score of >4 on Canadian Neurological Scale (CNS) and <15 on National Institutes of Health Stroke Scale (NIHSS). Severe defined by score of <4 on CNS and >15 on NIHSS.

<sup>†</sup>Data presented as mean  $\pm$  standard error of the mean.

<sup>‡</sup>Significant change (p < 0.001). Max = maximum.

chronic-phase patients [14–16] and shows that amelioration of chronic neurological impairments long after the expected period for recovery following stroke is possible. More important for our understanding of recovery, the magnitude of the improvement achieved with PBPT was many times greater than achieved with our previous robotic therapy. The only change was the therapy protocol: the same robot assisted with the same set of reaching movements. A treatment protocol that adapted to the patient's motor ability and presented a continuous challenge substantially enhanced recovery.

An important and informative detail is that this enhancement of recovery was achieved with fewer repetitions. Because the adaptive PBPT protocol adjusted the time allotted for a movement and allowed long movement durations as needed, fewer repetitions could be accomplished in a 1-hour therapy session. Under this adaptive protocol, patients made just over 12,000 movements over the course of treatment. Under the previous sensorimotor protocol, patients made just over 18,000 movements in the same number of sessions. PBPT achieved significantly greater impairment reduction with more than 6,000 fewer movements.

This result shows that although the process of recovery may share some features of motor learning (such as specificity), the relationship between learning and recovery may be subtle. Though movement is beneficial, movement alone is not sufficient. Active involvement of the patient is important. Though repetition may be beneficial, repetition alone is not sufficient; the benefits of robotic therapy do not exclusively derive from the high "dosage" of movement delivered.

# **MOTOR BEHAVIOR IN RECOVERY**

Neuroanatomical studies can provide important clues about the process of neurorecovery; for example, activitydependent neural plasticity may explain why movement matters. However, that such studies will prove sufficient to predict the behavioral course of recovery with enough detail to guide the design of robots or treatment protocols seems unlikely. Equally important is to quantify the behavioral details of recovery. Fortunately, robots provide an excellent instrument platform for this endeavor.

Our earliest work on robotic therapy emphasized planar motion partly as a matter of convenience. Backdrivable robots are easier to implement in planar configuration, but our choice was also guided by neuroscience research. The study of planar reaching continues to be a

highly productive paradigm for studying the neuroscience of motor behavior. One robust finding that has emerged from such work is that, in the absence of any overriding requirement such as maximum speed or precision, unimpaired planar reaching motions are organized to meet kinematic goals. The form of these movements is competently described as though they were chosen to be maximally smooth [44] in visually relevant coordinates: the path of the hand is straight and the speed profile has a single peak. Exposure to mechanical perturbations (such as motion-dependent force fields) that perturb this kinematic pattern evokes a spontaneous adaptation that restores the original pattern [45–46]. Conversely, exposure to visual displays that distort the appearance of the motion also evokes adaptation, again restoring the original kinematic pattern, even though that may require substantially different patterns of actual limb motion and muscle force [47-48]. At least for this class of movements, the brain controls the kinematics of hand motion, subordinately adjusting muscle forces as needed.

# **SUBMOVEMENTS**

In contrast to the smoothness of unimpaired motion, a striking feature of the earliest arm movements made by acute-phase stroke patients as they recover is that they are highly fragmented. In earlier work, we studied the kinematics of these motion fragments, or submovements, and found that they have a highly stereotyped speed profile [49]. The shape of this speed profile did not vary with the duration or extent of the motion fragment and was the same for the stroke patients we studied, who had a wide variety of lesion territories and sizes. Interestingly, this shape was indistinguishable from the maximum-smooth speed profile typically observed in studies of unimpaired reaching movements.

As we reported previously, our observations suggested that as recovery progressed, these motion fragments, or submovements, progressively overlapped and merged to produce continuous motion [50]. From that observation and a comparison with unimpaired reaching, we expected patients' movements to become smoother as they recovered. To test whether this was so, we performed a detailed study of the kinematics of stroke patients' arm movement at various stages of their recovery [51]. A secondary goal was to determine if we could find useful robot-based measures of recovery to complement existing clinical scales.

We analyzed planar point-to-point reaching movements made with the hemiparetic arm by 31 patients recovering from stroke. Twelve were acute-stage inpatients who had suffered their first unilateral infarct less than 1 month before beginning the study, and 19 were chronic-stage outpatients between 12 and 53 months after stroke. Subjects were between 19 and 78 years, with no significant difference in age between inpatients and outpatients; 10 were women, 21 men. We calculated mean speed, peak speed, movement duration, and five measures of smoothness (all defined such that higher values denoted smoother movements [51]).

Subjects' mean speed (total distance traveled divided by total movement duration) tended to increase for both inpatients and outpatients, with inpatients as a group showing significantly larger changes (p < 0.001). Similarly, movement duration tended to decrease for both inpatients and outpatients, with significantly larger changes in inpatients as a group. However, whether these would make useful measures of individual recovery is unclear, because almost 20 percent of subjects (6/31) showed no significant change in duration, 32 percent (10/31) showed no significant change in mean speed, and 13 percent showed significant decreases in mean speed. Subjects' peak speed showed no consistent trend, increasing in some patients and decreasing in others, with significant decrease being more common. Insofar as faster movement requires greater muscle strength, this observation is consistent with our finding that strength training afforded no advantage over sensorimotor therapy. At least for these patients, recovery was not simply a matter of getting stronger or moving faster.

In contrast, all but 1 of our 31 subjects showed a significant increase in one or more of the smoothness measures, with over 70 percent (22/31) showing an improvement in four or more. The differences between first- and last-day values of each smoothness measure are plotted in **Figure 1**. For all but one of the measures, the change in smoothness differed significantly (p < 0.001) between inpatient and outpatient groups. For three of the five measures, inpatients showed greater increases in smoothness than outpatients. As smoothness is fundamentally a measure of movement coordination, these results reinforce the impression that motion, rather than muscle, should be the focus of therapy.

The most mathematically rigorous measure of smoothness (based on jerk, the rate of change of acceleration) presented an initially surprising result: while for outpatients it



#### Figure 1.

Changes in five smoothness measures [(a) jerk, (b) speed, (c) movement arrest period ratio (MAPR), (d) tent, and (e) peaks] for each subject over course of therapy. Positive changes indicate increasing smoothness. Filled circles denote statistically significant (p < 0.05) changes; open circles denote changes that did not reach statistical significance. Statistical significance of difference between inpatient (acute) and outpatient (chronic) smoothness changes are indicated (p-value). Reprinted by permission from Rohrer B, Fasoli SE, Krebs HI, Hughes R, Volpe BT, Frontera WR, Stein J, Hogan N. Movement smoothness changes during stroke recovery. J. Neurosci. 2002;22(18):8297–8304. [PMID: 12223584]. (© [2002] Society for Neuroscience.)

typically increased, for inpatients it typically decreased. However, this result ultimately turned out to be informative, because this characteristic can be explained by progressive merging of submovements. To illustrate this, we computersimulated a movement composed of two submovements with minimum-jerk speed profiles of the same amplitude and width, initiated an interval of time (T) in seconds apart. Simulated speed profiles along with comparable sample speed profiles of subjects are shown in **Figure 2**.



#### Figure 2.

Simulated versus actual speed profiles. (**a–d**) Progressive merging of two minimum-jerk curves. Interval between submovements (*T*) shown as arrow on *x*-axis. Actual patient speed profiles from (**e**) first and (**f**) last therapy day for inpatient and (**g**) first and (**h**) last therapy day for outpatient. Simulated speed profiles qualitatively resemble actual patient data. Reprinted by permission from Rohrer B, Fasoli SE, Krebs HI, Hughes R, Volpe BT, Frontera WR, Stein J, Hogan N. Movement smoothness changes during stroke recovery. J. Neurosci. 2002;22(18):8297–8304. [PMID: 12223584]. (© [2002] Society for Neuroscience.)

We calculated each of the five smoothness measures for the simulated movements. As submovements merged, smoothness measures generally increased, except for the one based on jerk: starting from large values of T (no overlap), it initially decreased as T decreased, then reached a minimum and subsequently increased as T continued to decrease. Thus a recovery process that started by making separate submovements (as we observed with inpatients) and proceeded by progressively merging those submovements would competently describe both our inpatient and outpatient data. The sample speed profiles from subjects shown in **Figure 2** support this account.

# **EXTRACTING SUBMOVEMENTS**

Encouraged by this result, which indicated that the "fine-structure" of movement kinematics might yield insight about recovery, we wanted to develop methods to identify or "extract" submovements from continuous kinematic records. Unfortunately, this proved to be remarkably difficult. Two main problems existed: first, the shape to be extracted is unknown. Though we observed isolated submovements early in recovery, we have no a priori guarantee that these shapes remain unchanged as recovery proceeds. Second, and much more vexing, even if the submovement shape was known, the sequence of submovements obtained is exquisitely sensitive to the method used to identify them. We found that all of the prior methods that have been used were vulnerable to substantial misidentification [52]. By recasting submovement extraction as a global nonlinear optimization problem, we developed two reliable approaches. The first is based on a "branch-andbound" algorithm. It is powerful, with proven convergence properties, and can correctly identify submovements even in the presence of noise [52]. Unfortunately, it is computationally burdensome. The second method uses a stochastic "scattershot" global nonlinear minimization algorithm. It is probabilistic in nature; i.e., the results are globally optimal with probability close but not equal to unity, but it requires approximately four orders of magnitude less time to compute [53–54].

# SUBMOVEMENT CHANGES WITH RECOVERY

Using the scattershot algorithm, we extracted submovements from planar point-to-point reaching movements made with the hemiparetic arm by 41 patients recovering from stroke [55]. Fifteen were acute-stage inpatients who had suffered their first unilateral infarct less than 1 month before beginning the study, and twenty-six were chronic-stage outpatients between 12 and 54 months poststroke. Subjects ranged in age from 19 to 83; 12 were women and 29 men. A representative result is shown in **Figure 3**. Analysis of submovements extracted from all patients is summarized in **Figure 4**.

Over the course of therapy, the number of submovements required to reach a single target decreased. As may therefore be expected, submovements also tended to increase in amplitude (peak speed) and duration.



#### Figure 3.

Typical movements for (a) first and (b) last day of therapy. Bold lines show tangential speed during the movement; fine lines show underlying submovements. Data from last day show fewer submovements, which have greater peak speed, duration, and overlap than earlier movements.

Submovements became more overlapped for all patients, but more markedly for inpatients (significant at p < 0.05). Outpatients' values for overlap were all grouped tightly around a common mean, from 0.7 to 0.8 s. Inpatients' values appeared to converge to that mean during therapy; initial values for overlap tended to be lower than 0.7 s, but (with a few exceptions) increased markedly so that inpatients' final values of overlap were much more closely grouped around 0.7 s.

The time between the peaks of adjacent submovements decreased significantly (p < 0.05) for inpatients (those less than 1 month poststroke) but not for outpatients (those greater than 12 months poststroke). Furthermore, interpeak intervals appeared to converge to a limit. Although inpatients began with a wide range of interpeak intervals (from 0.4–0.8 s), at the completion of therapy, they all fell in a narrow band centered approximately at 0.45 s. Outpatients tended to begin and end therapy in that same band; the majority of them showed no significant change.

This last finding indicates that different aspects of sensorimotor recovery may follow different schedules. Recall that in our comparison of sensorimotor therapy and resistance exercises, we found that gains in strength appeared to reach a plateau within the first half of our treatment protocol. Here, a detailed study of kinematics shows that during recovery, the interval between submovements appears to approach a limiting minimum value and reaches it within a period of approximately 1 year. Other submovement parameters appear not to be limited in the same way, though further study is needed to test whether they too approach limiting values and, if so, on what timescales. Nevertheless, one observation of immediate practical impact (perhaps obvious but worth



Figure 4.

Number of patients who showed increases and decreases in each of five submovement (Submv.) characteristics, for all patients, inpatients, and outpatients, respectively. White bars indicate total number of participants in each group showing any change, and black bars indicate how many of those were statistically significant at p < 0.05 level. \*Indicates bar order on *x*-axis.

emphasizing) is that optimal treatment is a moving target: the details of therapy should adapt with the patient. The success of our adaptive PBPT algorithm indicates that this route can lead to substantial benefits.

The pattern of submovement changes observed during recovery may hint at the neurological processes underlying recovery. To speculate briefly, we observed changes in submovement parameters during recovery that may reflect a motor controller that uses at least two distinct kinds of "models" or "maps:" "forward" maps that use sensory input to predict the consequences of ongoing movements (e.g., where the limb is likely to reach) and "inverse" maps that determine what motor output (e.g., pattern and timing of muscle activations) is required to produce a desired movement [56-57]. The larger interpeak intervals of acute patients may indicate that they initially adopt a "move a little and see what happens" strategy, while the forward maps are being recalibrated. Further, the fact that interpeak intervals approach a limiting value and do not change for outpatients may

indicate that the forward maps are recalibrated first, before the inverse maps [58]. But this is speculation; testing whether a model of this nature may account for the process of neurorecovery would require substantial further research.

# CONCLUSIONS

Should therapy focus on motions or muscles? Our investigations indicate that, at least for the upper limb, recovery of the normal pattern of kinematic coordination is preeminent. Passive motion does not affect impairment, and therapy to improve muscle strength appears to offer no advantage over sensorimotor training, whereas adaptive treatment that continuously challenges and assists a patient to improve coordination can yield substantial advantages.

Analysis of movement kinematics indicates that although recovery generally tends to be accompanied by increased speed and/or reduced movement duration, these quantities (which are most likely to depend on muscle strength) will not provide reliable measures of recovery. The recovery process appears to have a "finestructure" that is obscured by these aggregate measures. Measures of movement coordination (such as smoothness) appear to be more informative.

The fine-structure of behavioral recovery suggests that it proceeds by rapidly recovering an elementary "alphabet" of primitive movements then, over a longer time period, reacquiring the means to smoothly combine these elements. The process of recovering movement and deploying it to accomplish functional behavior may be loosely analogous to the acquisition of functional writing, first forming letters, then words, then sentences, paragraphs, and so forth.

Of course, recognizing that action requires more than motion is important. Though posture and movement are important, muscles do more than pull; they also change mechanical impedance (e.g., stiffness, viscosity), which is critically important for functional tasks requiring interaction with objects in the world [59]. Recent neuroscience research has demonstrated purposeful control of impedance [60]. As far as we know, methods to provide robotic therapy to restore this functional motor ability to stroke patients remain unexplored.

A preponderance of evidence now available indicates that appropriate forms of robotic therapy can provide sig-

nificant benefits. Nevertheless, the benefits demonstrated in the studies reviewed here are modest, generally confined to a reduction of impairment. While such a reduction has undeniable value—a few points change in upper-limb FM score may reflect sufficient arm movement to enable independent dressing and toileting-the probability of regaining full functional use of the limb (the ultimate goal of therapy) is harder to assess. However, we should recognize that robotic therapy is still an emerging technology. For pragmatic reasons this review primarily considered therapy focused on planar arm movements to enable clearer comparison of different forms of therapy (what worked and what did not). Further benefits may be anticipated with new technology (under development or already available) that address a richer repertoire of behavior. Initial results are promising: preliminary studies indicate that benefits accumulate when planar robotic therapy is supplemented with treatment by robot modules for spatial antigravity motions [61] and wrist rotations [62].

Though further improvements may reasonably be expected, the limitations of robotic technology should be acknowledged. For example, endowing a machine with the compassionate insight about an individual's needs that a skilled clinician provides would be difficult, and whether it would be worth the effort to try is unclear. In our view, robotics is no more than a toolset, albeit one with great versatility, that improves the resources available to clinicians as they facilitate recovery. The best treatment for an individual patient is most likely to be a combination of robotic therapy and other approaches. Though we have focused our analysis on how movement kinematics converge toward normal patterns and focused our robotic treatment on aiding the restoration of normal movement patterns, we recognize that normal movement may be an unreachable goal, especially for the most severely impaired patients. In those cases, functional recovery may require a combination of restorative and compensatory approaches, though the optimum balance of the two remains to be determined.

Further advances in treatment of sensorimotor dysfunction, whether by robots or by other means, require progress on many frontiers: clinical studies to establish which approaches are effective for which patients and to identify optimal treatment schedules, new technology to better serve patients' needs and capitalize on opportunities to conduct therapy in new venues (such as the home) or in new ways (such as via the Internet), and fundamental research to understand the biology of recovery at all scales,

from molecular to behavioral. Of course, these efforts are related: a deeper understanding of the recovery process will guide and inspire technology development, which in turn provides material for clinical evaluation.

In our view, the value of a systematic, theory-based approach cannot be overemphasized; nothing is as practical as a well-supported theory. The ongoing work summarized here (along with the work of many others we have omitted for brevity) indicates that, at least for the upper limb, the broad outlines of a quantitative, behavioral theory of recovery after stroke may be discernible.

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Brandon Rohrer is now with the Cybernetic Systems Integration Department, Intelligent Systems and Robotics Center, Sandia National Laboratories, Albuquerque, New Mexico.

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