Quantification of Force Abnormalities During Passive and Active-Assisted Upper-Limb Reaching Movements in Post-Stroke Hemiparesis

Peter S. Lum,* Charles G. Burgar, *Member, IEEE,* Deborah E. Kenney, and H. F. Machiel Van der Loos, *Member, IEEE*

***Abstract*— We evaluated a method for measuring abnormal upper-limb motor performance in post-stroke hemiparetic subjects. A servomechanism (MIME) moved the forearm in simple planar trajectories, directly controlling hand position and forearm orientation. Design specifications are presented, along with system performance data during an initial test of 13 stroke subjects with a wide range of impairment levels. Performance of subjects was quantified by measuring the forces and torques between the paretic limb and the servomechanism as the subjects relaxed (passive), or attempted to generate force in the direction of movement (active). During passive movements, the more severely impaired subjects resisted movement, producing higher levels of negative work than less-impaired subjects and neurologically normal controls. During active movements, the more severely impaired subjects produced forces with larger directional errors, and were less efficient in producing work. These metrics had significant test–retest repeatability. These motor performance metrics can potentially detect smaller within-subject changes than motor function scales. This method could complement currently used measurement tools for the evaluation of subjects during recovery from stroke, or during therapeutic interventions.**

***Index Terms*— Force abnormalities, hemiparesis, motor function, motor performance, reaching movements, stroke, upper-limb.**

I. Introduction

A MAJOR difficulty in assessing treatment strategies for motor impairments in patients with neurologic injury is the lack of sensitive techniques to quantify the effect of the treatment. Treatment efficacy is usually determined by subjective evaluations of *motor function*, defined as the ability to accomplish movements or tasks that are the essential components of activities of daily living. While restoring motor function is the ultimate goal of rehabilitation, there is wide agreement that more objective and sensitive measures are needed to aid in guiding treatment protocols, evaluating the efficacy of specific treatments, and charting the recovery process [1]. *Motor performance* is defined as the kinematics or forces during the movement or task being tested. Quantification of motor performance with continuous variables can potentially detect smaller differences in ability than motor function scales. Abnormal motor performance can be determined by comparison with the performance of neurological normals, or in the case of hemiparesis, performance of the opposite limb. In this study, we focused on abnormal motor performance during passive and active-assisted movements, therapeutic techniques used following neurologic injury. These exercises normally involve movement of the paretic upper-limb by a therapist as the patient relaxes (passive), or actively attempts to contribute to the movement (active). We used the interaction forces/torques during these movements to quantify abnormal motor performance in the paretic limbs of post-stroke subjects.

In order to produce repeatable movement patterns in the paretic limb, we developed a servomechanism (MIME) that moves the limb in simple predetermined trajectories by directly controlling the position and orientation of the forearm. MIME consists of left and right mobile arm supports that limit movement to the horizontal plane, and a six degree-of-freedom (DOF) Puma robot arm that is attached to the arm support on the paretic side (Fig. 1). The robot applies forces/torques to the paretic forearm through the support. Subjects are instructed to relax during the movements (passive), or to assist the movement by generating force in the direction of movement (active). A six-axis force/torque transducer measures the forces/torques applied to the paretic limb.

Fig. 2 lists the mechanisms which can produce abnormal externally measured forces (in the following, we use the term forces to refer to both forces and torques). Force abnormalities during both passive and active movements can be due to altered mechanical properties of the limb and/or mechanisms associated with abnormal muscle activity. We define muscle activity as the development of muscle force due to activation. In passive movements, abnormal muscle activity can be due to hyperreflexia or abnormal descending commands. All of these mechanisms contribute to hypertonia, defined as increased resistance to passive movement. In active movements, abnormal muscle activity could be due to weakness, inability to grade contractions in individual muscles, or incoordination (defined as abnormalities in the temporal pattern or spatial distribution of activity across different muscles). A paradigm

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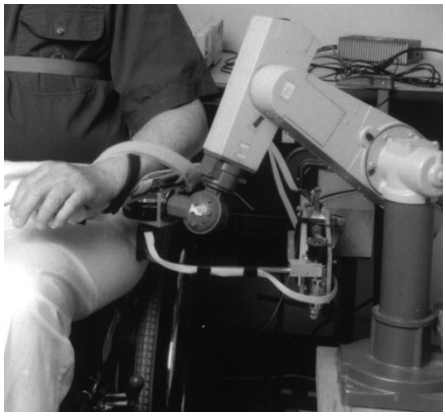
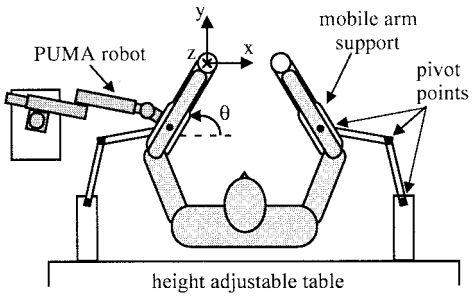


(a)



(b)

Fig. 1. (a) Front view picture of the left mobile arm support and robot and (b) top view diagram.

mechanisms that can cause abnormal forces

passive movements
- abnormal muscle activity
  - hyperreflexia
  - abnormal descending commands
- altered mechanical properties of the limb

active movements
- abnormal muscle activity
  - weakness (MVC, isometric force rise time, etc.)
  - inability to smoothly grade contractions
  - incoordination (synergies, co-contraction, overflow, etc.)
- altered mechanical properties of the limb

Fig. 2. List of mechanisms which can produce abnormal forces during passive and active trials.

which can isolate and quantify the severity of each of these mechanisms would be a useful tool for clinical evaluations and motor control studies. Clearly, isolation of individual mechanisms is difficult without electromyogram (EMG) measures. For example, separating weakness from incoordination is not possible with measures of external force alone, since what might appear to be weakness in an agonist might actually be due to agonist/antagonist co-contraction, which would be considered incoordination.

In this study, we measured external forces during constrained movement and compared these forces with those from the opposite limb. In free movement tasks, the kinematics of the paretic limb are compared with kinematics from the opposite limb or from normal subjects. However, Fig. 3 illustrates that this comparison is confounded by the fact that kinematics can affect nearly every stage of the motor control model: gravity is position dependent; passive mechanical forces are dependent upon both position and velocity of movement; inertial forces are dependent upon position and acceleration; centripetal and Coriolis forces are position and velocity dependent; muscle moment arms vary with position; muscle forces are dependent upon the force-velocity and length-tension relations; kinematics directly affect the output from muscle stretch receptors. As a result, it is difficult to determine the contribution of abnormal muscle activity to abnormalities in kinematics. However, in the MIME paradigm, the kinematics are guaranteed to be consistent, and the influence they have on all stages of the model will be constant for all movements in a particular pattern (illustrated in Fig. 3 by the dotted lines), even with dramatically different descending commands. Thus, paretic/contralateral limb differences in the forces measured cannot be attributed to any of these factors, and must instead be due to altered passive mechanical properties and/or abnormal muscle activity in the paretic limb.

The effects of altered passive mechanical properties can be removed by biasing forces measured in the active trials by forces measured in the passive trials. In passive movements, MIME moves the limb in the selected kinematic pattern as the subject is instructed to relax. The external forces would reflect the summed effects of gravity, inertia, centrifugal forces, Coriolis forces, passive mechanical properties, and forces related to muscle activity: hyperreflexia and abnormal descending commands. In active movements, voluntary descending commands are added to the picture and directly affect muscle activity and external forces. *Voluntary generated forces* are defined as the forces measured in active trials biased by forces measured in passive trials. Since the kinematics of active and passive trials are the same, all forces not related to muscle activity (including passive mechanical forces) are common to the two measurements and eliminated with this subtraction. Thus, abnormalities in voluntary generated forces can only be due to abnormal muscle activity. However, it should be noted that not all muscle activity abnormalities will appear as abnormalities in voluntary generated forces (i.e., abnormal co-contraction), and abnormalities in the voluntary generated forces could be due to abnormal muscle activity in passive and/or active movements. Nevertheless, from a global input-output perspective, this quantity can be thought of as the ability of the subject to control the neuromuscular apparatus and actively generate a force in the direction of movement, relative to forces in the passive condition.

We tested the hypothesis that force abnormalities during passive and active-assisted movements will increase as the level of upper-limb motor function decreases. By proving this hypothesis we verify that subjects who are more severely impaired functionally perform worse in the MIME paradigm. This is the minimal requirement for a useful paradigm. From a basic science perspective, this provides evidence that the mechanisms responsible for abnormal performance in the MIME task also contribute to lost motor function. If the

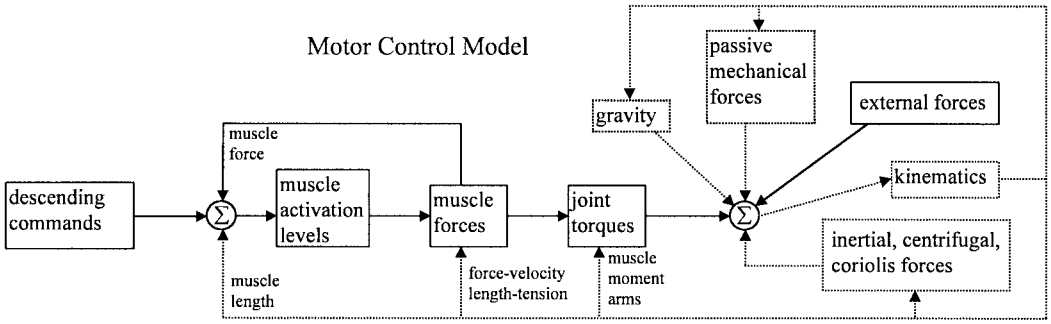


Fig. 3. Simplified block diagram of a model of motor control. The dotted lines are explained in the text.

hypothesis is false, the task may be too narrowly focused or the measurements inadequate, and analysis of performance data cannot yield insight into lost motor function. From a clinical perspective, one can use motor performance in a task to measure small changes in the status of a subject due to a therapeutic intervention. If there is a correlation between abnormal performance in this task and lost motor function, one can evaluate if these motor performance changes (which may be too small to affect motor function) are positive or negative. In order to test this hypothesis, the Fugl-Meyer (FM) examination was chosen as the "gold standard" for motor function. The validity and repeatability of the FM have been established [2]–[6].

Several studies have investigated the use of motor performance measures to augment clinical measures of motor function in subjects following neurologic injury. Cramer *et al.* used a computerized dynamometer to measure grip strength and index finger tapping rate in stroke subjects, and found strong correlations between motor parameters derived from these data and FM scores [7]. They emphasized the potential increase in sensitivity to small changes in hand function that a continuous linear scale offers. Turton *et al.* measured recovery of motor performance in a Fitts tapping task, and found that movement times continued to improve even when other subjective clinical scales had reached a plateau [8]. Heller *et al.* tested 56 subjects for the first three months post stroke, and found that a pass/fail activity scale (Frenchay arm test) had low sensitivity, but could be augmented with grip strength at the low end, and the nine-hole-peg test at the high end [9]. De Souza *et al.* followed stroke subjects for 64 weeks with an arm function test which used pass/fail scoring of passive movement, tone, pain, and several activities and movements [10]. They found good correlation between this test and motor performance in a pursuit tracking task [11]. However, when comparing these tests to standard clinical assessments, they concluded that the clinical assessments were inadequate for more severely disabled and slowly recovery patients. Jones *et al.* advocated using a preview tracking exam based on a steering-wheel task as a global measure of the sensorimotor system in conjunction with a more specific neurological assessment battery [12]. They tested 28 brain-damaged subjects repeatedly over several weeks and distinguished neurological recovery from the normal motor learning that occurs with repeated practice of a task. There have also been attempts to develop tools to measure quality-of-movement for persons with cerebral palsy; using subjective assessments based on ordinal scoring [13], [14], accelerometry [15], and video analysis to determine the number of movement segments in a reach [16]. Taken together, these studies suggest that assessments of motor function such as the Frenchay arm test, the arm function test, or the FM can be augmented at the high end with objective examinations of motor performance such as Fitts Tapping, pursuit tracking or the nine-hole-peg test. However, there is a lack of measurement tools which target the low end of the scale other than strength measures. In this study, we demonstrate MIME's ability to measure force abnormalities during attempted movements, especially in severely impaired subjects who have little or no isolated movement. These subjects typically have some movement in the flexor and extensor synergies, but have little capacity for isolated elbow rotation for example, and are unable to move in patterns that are out of synergy. The performance measures obtained from the MIME paradigm can augment motor function evaluations in these subjects.

Analysis of upper-limb kinematics has been useful for quantifying some aspects of abnormal motor performance following stroke. Wing *et al.* tested stroke subjects in reaching movements for one year and fitted smooth recovery curves to several kinematic parameters [17]. While most subjects showed improved peak velocity over time, there was no difference between recovery of isolated elbow movements and movements which were in or out of synergies. Trombly looked at recovery of reaching movements over a nine-week period, and found that increased speed of movement correlated with increased smoothness, not with increases in strength or agonist EMG [18]. They interpreted this as evidence that the increase in movement speed was due to motor learning. Levin also looked at reaching movements and found smaller movement amplitudes, longer movement times, deviations from straight lines, increased dispersion and segmentation [19]. Abnormalities in elbow/shoulder phase plots were attributed to loss of interjoint coordination. Ada *et al.* showed that in subjects with adequate strength and movement speed at the elbow, poor performance in a target tracking task was due to loss of dexterity [20]. In our study, we demonstrate the potential added insight into impaired motor performance that measurements from MIME afford: principally the ability to isolate muscle activity abnormalities

from altered passive mechanical properties during constrained reaching movements.

II. Methods

MIME incorporates a PUMA 260 robot (Stäubli-Unimation Inc.) and two mobile arm supports that constrain forearm motion to the horizontal plane (Fig. 1). The mobile arm supports were modified from commercially available units (North Coast Medical NC38022) that were originally designed to facilitate functional upper-limb activities in disabled individuals. The arm support bases were attached to an adjustable-height table. To assist the right arm, the subject faced the table; to assist the left arm, the subject faced the opposite direction. The robot end effector was attached to the trough of one mobile arm support near the center of mass of the forearm and moved the limb in predetermined trajectories. By directly controlling both forearm position and orientation, the configuration of the entire upper extremity during the movements was very consistent across subjects with possible differences due only to subject size and variations in shoulder protraction/retraction and elevation/depression.

A six-axis force/torque sensor (Assurance Technologies Gamma 130/10, 0.10 N resolution) measured the external forces applied to the limb. Position encoders (US Digital E2 optical encoder, 4096 counts/revolution) at the pivot points of the mobile arm supports measured their configurations. Position and force data were sampled at 105 Hz by a 486 PC and displayed on the computer monitor for the experimenter. A simple kinematic algorithm based upon the segment lengths and angles of the mobile arm support was used to calculate hand position (x, y) and forearm orientation (θ). Errors in the measurement of hand position were $<$0.3 cm. The native robot controller (Unimate Inc., VAL II programming language) implemented the low level proportional-integral-derivative servo control laws for the robot motors (1000 Hz update rate). The desired position of the hand (x, y) and orientation of the forearm (θ) were updated at 35 Hz via a 38.4-kbaud serial connection between the Unimate controller and the 486 PC. Location commands were recalled from trajectory files stored on the PC.

Several redundant safety features were incorporated into MIME. Since the weight of the limbs was supported by the mobile arm supports, we were able to use a relatively small and weak robot arm. The PUMA 260 is limited to 10 N of force at its end effector. Software and hardware stops kept the mobile arm supports in a restricted workspace (30×30 cm). The control algorithm automatically stopped the robot if velocities greater than $2\times$ the peak velocity of the programmed trajectory were encountered. A foot operated panic switch was located at the subject's feet. None of the subjects we tested expressed concern for safety when exercising with MIME.

For testing, subjects sat in a wheelchair and rested their forearms in the arm support troughs (Fig. 1). A chest strap limited torso movement and straps secured the forearms to the arm support troughs. The forearm was pronated and the hand grasped a 6.4-cm diameter ball. Six different 15 cm, point-to-point, reaching movements were tested, all in a horizontal plane approximately 8 cm above lap level (Fig. 4). These six different movement directions required six different upper-limb muscle coordination patterns, providing a reasonable representation of overall shoulder/elbow function. In order to determine "normal" kinematics for these movements, the robot was disconnected from the mobile arm support and three normal subjects attempted to perform each movement in 2.5 s, while being given feedback of their movement time after each trial. In Fig. 5, the 95% confidence envelope (average trajectory $\pm$ two standard deviations) for ten trials in the forward direction from a typical normal subject are overlaid with a modeled trajectory which used a straight line path from start to stop, and sinusoidal velocity profiles. It was found that for all movement directions, modeled trajectories were within the 95% confidence envelopes of the experimental trajectories. These six modeled forearm trajectories were stored in files on the PC and used to control all of the movements. While one would not normally expect sinusoidal trajectories in such slow movements, this might be attributable to inertia of the mobile arm support and a conscious effort on the part of the subjects to move smoothly.

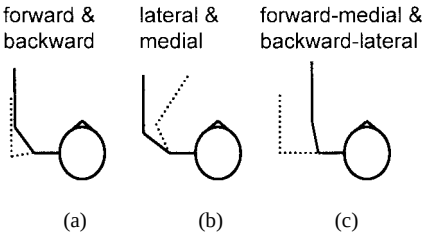


Fig. 4. Top view of the six movement directions. (a) Forward direction (from the dotted arm position to the solid arm position) and backward direction (reverse). (b) Lateral direction (dotted to solid) and medial direction (reverse). (c) Forward-medial direction (dotted to solid) and backward-lateral direction (reverse). In all movements the hand moves in a straight line 15 cm.

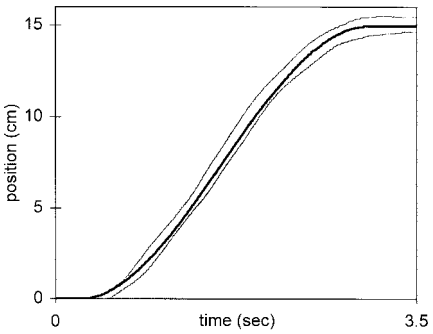


Fig. 5. The 95% confidence envelope (average trajectory $\pm$ two standard deviations) for ten trials in the forward direction from a typical normal subject (thin lines) overlaid with a modeled trajectory which used a straight line path from start to stop, and sinusoidal velocity profiles (thick line).

Six neurologically normal and 13 hemiparetic stroke subjects were tested. All stroke subjects had a diagnosis of cerebrovascular accident based upon the results of a head computed tomography or magnetic resonance imaging scan. Age, FM score, time since onset of stroke, and site of lesion characteristics of the 13 stroke subjects are summarized in Table I. Subjects were excluded if they had severe language or cognitive deficits which prevented them from following

TABLE I
STROKE SUBJECT CHARACTERISTICS

FM score	age	hemi side	months post	site of lesion
14	69	L	7	MCA
14	49	L	8	basal ganglia
15	61	L	45	brain stem
17	71	L	44	internal capsule
18	55	L	2	pontine nucleus
19	48	L	3	MCA
21	66	R	40	brain stem
39	64	L	34	carotid artery
44	71	L	8	internal capsule
52	47	L	1	MCA
58	62	L	15	MCA
62	63	L	1	internal capsule
65	68	R	41	brain stem

MCA: MIDDLE CEREBRAL ARTERY.

simple instructions. Eight of the stroke subjects were tested twice within the same week. All protocols were approved by the institutional human subjects committee. Informed consent was obtained from all subjects.

Each test session began with an upper extremity FM examination of motor function. The clinician subjectively scored reflex responsiveness and the ability to perform several simple upper-limb movements. Generally, FM scores of <20 indicate severe impairment, and scores approaching 60 indicate mild impairment (max score = 66, indicating no impairment). The validity and reliability of the FM have been established [2]–[6]. A single clinician performed all of the FM examinations. Subjects were then positioned in MIME as described above. For each movement direction, subjects were first instructed to remain passive as the robot moved the limb in the programmed trajectory. Data from five passive trials were collected. The data included the position and orientation of the forearm, and the external forces/torques applied to the arm. Next, the subjects were instructed to contribute voluntarily to movement by pushing or pulling "with approximately one pound of force."After each trial, subjects were given knowledge of their average force level in the direction of movement and were encouraged to increase/decrease their effort if their force levels were outside of a target window (target force $\pm$ 0.2* target force). Subjects were instructed to watch their moving limb, and to relax their contralateral limb throughout the trials. Data from ten active trials were collected before moving on to the next movement direction. Subjects performed the sequence first with their normal limb (despite the growing evidence of movement impairments ipsilateral to the lesion [21], [22], we will refer to this limb as the "normal" limb for convenience). The wheelchair was then turned to face the opposite direction and the sequence was performed by the paretic limb.

A quasistatic analysis was used to calculate several scalar metrics for each session. The following procedure was applied to each movement direction separately. First, the passive force/torque profiles $p_i^j(t)$ were averaged across all passive trials at each time point to produce an average force/torque profile. Subscripts refer to the trial number and superscripts refer to the vector component. The first three components $(j = 1,2,3)$ are the forces and the last three $(j = 4,5,6)$ are the torques

$$\overline{p^j}(t) = 0.2 \times \sum_{i=1}^{5} p_i^j(t). \tag{1}$$

For each active trial, the forces/torques voluntarily generated were calculated by subtracting $\overline{p^j}(t)$ from the active profile $a_i^j(t)$. These passive-biased profiles were then averaged over the movement duration and across trials to calculate the average force/torque components generated by the limb ($t_0 \equiv$ sample time at movement initiation, $t_f \equiv$ sample time at end of movement, $N \equiv$ number of samples between t_0 and t_f)

$$F^j = 0.1 \times \sum_{i=1}^{10} \frac{\sum_{t=t_0}^{t_f} \left[a_i^j(t) - \overline{p^j}(t)\right]}{N}. \tag{2}$$

The three force components from (2) form the vector $\vec{F} \equiv [F_x, F_y, F_z]^T$. The *force magnitude* (*Fmag*) was calculated for the paretic limb, the normal limb and for the paretic limb relative to the normal limb $|\vec{F}_{\text{paretic}}| - |\vec{F}_{\text{normal}}|$. The *force directional error* (*FDE*) was calculated for both limbs relative to the movement direction, and for the paretic limb relative to the normal limb

$$FDE = \arccos\left(\frac{\vec{F}_{\text{paretic}} \cdot \vec{F}_{\text{normal}}}{|\vec{F}_{\text{paretic}}||\vec{F}_{\text{normal}}|}\right). \tag{3}$$

Let $\Delta_i^j(t)$ be defined as the forearm displacement vector from the previous sample time to sample time t. The generated force/torque profiles and $\Delta_i^j(t)$ were used to calculate the *positive work* (W_i) done by the limb during each trial. The potential work (Θ_i) was defined as the work that would have been produced during a trial if, at each instant, the *Fmag* was directed precisely in the movement direction, and the torque magnitude was oriented precisely in the direction of rotation. The *work efficiency* (η_i) was calculated by normalizing the positive work done over each trial by the potential work for that trial

$$\begin{aligned} W_i &= \sum_{t=t_0}^{t_f} \left\{ \sum_{j=1}^{6} \max\left\{ \left[a_i^j(t) - \overline{p^j}(t)\right] \times \Delta_i^j(t), 0 \right\} \right\} \\ \Theta_i &= \sum_{t=t_0}^{t_f} \left\{ \sqrt{\sum_{j=1}^{3} \left[a_i^j(t) - \overline{p}^j(t)\right]^2} \times \sqrt{\sum_{j=1}^{3} \left[\Delta_i^j(t)\right]^2} \right. \\ &\qquad \left. + \sqrt{\sum_{j=4}^{6} \left[a_i^j(t) - \overline{p^j}(t)\right]^2} \times \sqrt{\sum_{j=4}^{6} \left[\Delta_i^j(t)\right]^2} \right\} \\ \eta_i &= \frac{W_i}{\Theta_i}. \end{aligned} \tag{4}$$

Average values for positive work (W) and work efficiency (η) were then obtained by averaging across trials. To summarize, each movement direction yielded several descriptive metrics: *Fmag* (paretic, normal, paretic-normal), *FDE* (paretic, normal, paretic-normal), W (paretic, normal), η (paretic, normal). Each

of these metrics was then averaged across the six movement directions to yield scalars representative of the entire session. These values will be referred to as *passive-biased* metrics. While analyses of individual movement directions are of interest, averaging across movement directions was necessary before comparison with FM scores, which reflect overall limb ability. The entire procedure was also done without biasing the active profiles with the passive profiles. These values will be referred to as *unbiased* metrics. The procedure was also done with passive forces alone to produce *passive* metrics. All metrics were tested for correlation with the FM. Sensitivity was measured by the slope of the linear regression between the metric and FM scores. Repeatability was determined by testing subjects twice in the same week.

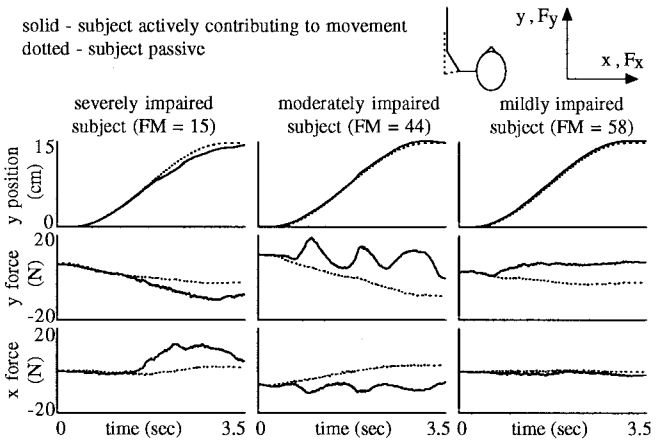


Fig. 6. Typical trials from three left-hemiparetic subjects when assisted by MIME. The top row shows the y movement of the left forearm for a passive (dotted lines) and an active trial (solid lines). The middle and bottom rows show the forward (y) and medially (x) directed force profiles from the left arm during these movements. The active force profile minus the passive profile is the voluntary generated force.

III. Results

MIME successfully assisted the paretic arm movements in all but one of the subjects tested (described below). The kinematics showed only minor differences across the other 12 subjects. Confidence intervals were calculated for the hand position 1.8 s after movement initiation (approximately 2/3 through the movement) in all forward movement active trials from all hemiparetic subjects, except the one outlier. The hand position in 95% of trials fell within a 2.6-mm-diameter circle. The ranges for the backward, lateral, medial, forward-medial and backward-lateral movements were 1.5, 2.5, 2.3, 2.0, and 1.9 mm, respectively. Recalculation at 1.4 and 2.8 s after movement initiation (midpoint and final location) yielded comparable or smaller ranges. However, individual trials occasionally showed large deviations from the programmed trajectory in one subject with extreme spasticity and flexor tone. Even under passive conditions, this subject applied forces to the robot that were large enough to overpower the wrist joint motors, causing the controller to cease movement. Nevertheless, the protocol was completed by this subject after the experimenter stabilized the robot wrist during the movements with his hand. This yielded movement trajectories within the ranges listed above. This subject's data was also included in the analysis.

Performance of stroke subjects reflected their recovery level. In Fig. 6, three stroke subjects (representative of severe, moderate and mild impairment) are contrasted in terms of interaction forces during typical passive and active trials in the forward movement direction. Since there was movement only in the forward $(+y)$ direction, neurologically normal subjects typically generated force only in this direction, with little or no medial $(+x)$ or lateral $(-x)$ force. In contrast, the severely impaired subject $(\mathrm{FM} = 15)$ resisted movement, generating force in the opposite direction of movement $(-y)$. There was also considerable medial $(+x)$ force, but movement in that direction was prevented by the robot. The moderately impaired subject $(\mathrm{FM} = 44)$ appropriately generated force in the forward $(+y)$ direction, but had difficulty maintaining a constant level throughout the movement and generated large lateral $(-x)$ forces. Similar to control subjects, the mildly impaired subject $(\mathrm{FM} = 58)$ generated a smooth constant force in the forward $(+y)$ direction with no medial or lateral force.

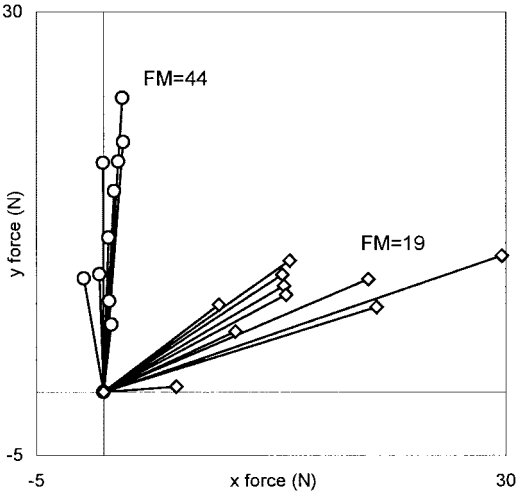


Fig. 7. The across-trial variability in force production typically observed during the active movements. Time-averaged force vectors (passive-biased) in the horizontal plane for all ten trials in the forward direction are overlaid for two stroke subjects (circles and diamonds).

Fig. 7 illustrates the across-trial variability in force production typically observed during the active movements. Time-averaged force vectors (passive-biased) in the horizontal plane for all ten trials in the forward direction are overlaid for two stroke subjects. The across-trial *Fmag* standard deviation ranged from 1.9 to 11.4 N for the stroke subjects, with a mean of 4.9 N. Thus, the average subject had a *Fmag* standard deviation that was greater than the target force level (one pound). Despite this large variability in force amplitudes, the directionality was fairly consistent, independent of amplitude. Force direction standard deviations ranged from 3.2 to 15.1°, with a mean of 10.8°. When considering that the range of possible errors spans 360°, the average subject had a directional standard deviation of only 3% of this full range.

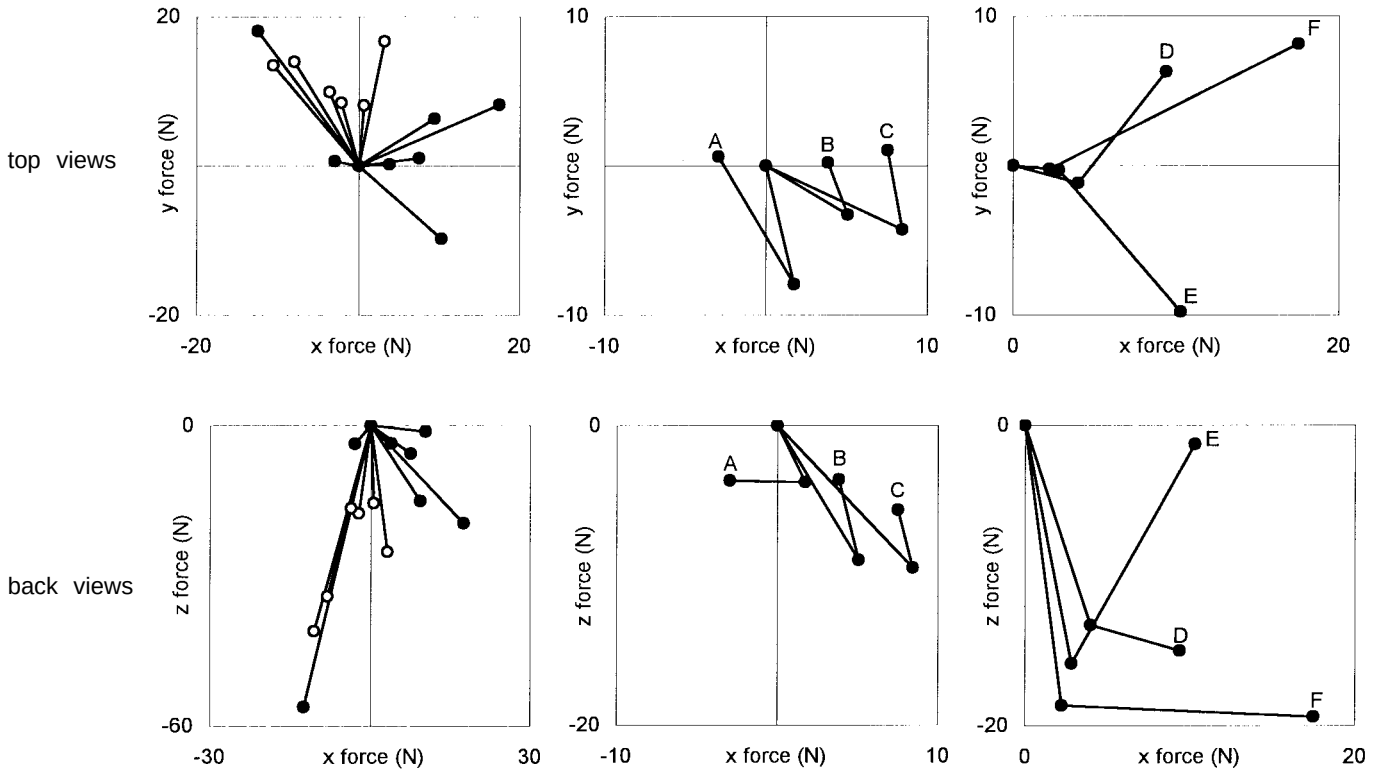


Fig. 8. (left column) Top and back two-dimensional (2-D) projections of the average force vector (unbiased method) generated during the forward movement by the paretic limbs. Subjects are divided into severely impaired ($\mathrm{FM} < 22$, solid circles) and moderate-mildly impaired ($\mathrm{FM} > 38$, open circles). These vectors represent the vector sum of passive and voluntary generated force vectors. Each line represents the average of ten active trials from a single subject. (middle and right columns) The passive and voluntary generated force vectors are separated for six severely impaired subjects (A–F). The straight line segments originating from the origin represent the passive vectors, and the voluntary generated force vectors are drawn with their origin at the tip of the passive vectors.

Both passive and voluntary generated forces contributed to abnormal performance during the active trials. The average force vector (unbiased method) from each paretic limb during the forward movement is plotted in Fig. 8 (left column). These vectors represent the vector sum of passive and voluntary generated force vectors. Subjects are divided into severely impaired subjects ($\mathrm{FM} < 22$, solid circles) and moderate-mildly impaired subjects ($\mathrm{FM} > 38$, open circles). The moderate-mildly impaired subjects typically generated force in the direction of movement $(+y)$ and in the downward $(-z)$ direction, indicating they allowed the arm support to carry the weight of their limb (as per instructions). In contrast, several of the severely impaired subjects tended to direct forces in the medial $(+x)$ direction, and to produce smaller downward $(-z)$ forces, indicating they were lifting their limbs out of the support. In the middle and right columns of Fig. 8, the passive and voluntary generated force vectors are separated for six severely impaired subjects (A–F). The straight line segments originating from the origin represent the passive vectors, and the voluntary generated force vectors are drawn with their origin at the tip of the passive vectors. Three subjects were hindered by passive resistance forces in the backward $(-y)$ direction (middle column, subjects A–C). All three of these subjects voluntarily generated some force in the direction of movement $(+y)$, but the magnitudes were not large enough to overcome passive forces, and none reached the target force levels, perhaps due to weakness. In contrast, three other severely impaired subjects had little passive resistance in the $(-y)$ direction (right column, subjects D–F). Two of these three were able to reach the target force level during active trials, but in all three subjects, the directionality of the voluntary generated forces had large errors. The medial $(+x)$ bias in the severely impaired subjects was due to passive forces in some subjects (B and C), but due mostly to voluntary generated *FDE*'s in others (D–F). The small $-z$ forces in the severely impaired subjects were due to passive forces in subject A, due to voluntary generated forces in subject E, and due to a combination of passive and voluntary generated forces in subjects B and C. In summary, both passive and voluntary generated force abnormalities contributed in varying amounts to abnormal performance during the active movements.

Variations in performance across subjects during the passive trials were examined with correlation analysis. Metrics were derived from the passive forces, as described in Section II. In the paretic limb, the *Fmag* did not vary with FM score whether calculated in absolute units or relative to *Fmag* in the contralateral limb $(p > 0.2)$. However, the *FDE* relative to the movement direction, and relative to the force direction in the opposite limb during the same movement, both decreased with increasing FM score $(p < 0.01)$. There was also a greater resistance to movement in the severely impaired subjects. Subjects with lower FM scores generated greater levels of negative work than subjects with higher FM scores $(p <$

TABLE II
CORRELATION, SENSITIVITY, AND REPEATABILITY OF THE UNBIASED AND PASSIVE-BIASED METRICS FOR THE PARETIC LIMB, THE NORMAL LIMB, AND FOR THE PARETIC LIMB RELATIVE TO THE NORMAL LIMB. CORRELATION (C) WAS DETERMINED BY THE METRIC VERSUS FM r VALUE. SENSITIVITY (S) WAS MEASURED BY THE SLOPE OF THE METRIC VERSUS FM REGRESSION. REPEATABILITY (R) WAS MEASURED BY THE TEST–RETEST r VALUE. VALUES THAT WERE SIGNIFICANT AT THE 0.01 LEVEL ARE REPORTED (NS = NOT SIGNIFICANT, NA = NOT APPLICABLE, FMP = FUGL–MEYER POINT)

		passive-biased analysis			unbiased analysis		
metric		paretic	normal	paretic - normal	paretic	normal	paretic - normal
force direction error	C =	-0.72	NS	-0.87	NS	NS	-0.87
	S =	-0.56 °/fmp	NS	-0.90 °/fmp	NS	NS	-0.97 °/fmp
	R =	0.69	0.46	0.39	0.73	0.54	0.64
force magnitude	C =	NS	NS	-0.80	NS	NS	NS
	S =	NS	NS	-0.18 N/fmp	NS	NS	NS
	R =	0.57	0.49	NS	0.74	0.59	0.54
work positive	C =	NS	NS		NS	NS	
	S =	NS	NS	NA	NS	NS	NA
	R =	0.77	0.53		0.81	0.66	
work efficiency	C =	0.81	NS		0.81	NS	
	S =	0.62 %/fmp	NS	NA	0.64 %/fmp	NS	NA
	R =	0.79	0.58		0.83	0.63	

0.005). None of these metrics in the contralateral limb varied with FM score ($p > 0.5$).

For the active trials, passive-biased metrics were evaluated in terms of correlation with the FM (metric versus FM r value), sensitivity (metric versus FM linear regression slope) and repeatability (test–retest r value [23]) (Table II). None of the normal limb metrics in the stroke subjects correlated with the FM, and there was no group difference between the normal limbs of stroke subjects and the limbs of neurologically normal subjects ($p > 0.2$); no ipsilateral abnormalities were detected. After calculating the voluntary generated forces, *FDE* of the paretic limb relative to the movement direction ($p < 0.005$) and relative to the contralateral limb ($p < 0.001$) dropped significantly with increasing FM (Fig. 9). The *Fmag* relative to the contralateral limb also dropped with increasing FM ($p < 0.001$). While positive work did not vary across subjects, the work efficiency (η) of the paretic limb increased significantly with the FM ($p < 0.001$) (Fig. 9). The sensitivity of these metrics was relatively low: *FDE* (*paretic-normal*) decreased 0.90°/FM point, and η (*paretic*) increased 0.62% per FM point. *FDE* and η had significant test–retest r values ($p < 0.01$). Repeatability was also tested with a two-tailed paired t-test. There were no significant differences between the first and second session values across subjects for *FDE* and η ($p > 0.3$). However, *Fmag* repeatability was poor compared to the other metrics; the test–retest r value was not significant ($p = 0.056$). The unbiased metrics are also summarized in Table II. Two of the significant results are missing from the unbiased metrics. In these two cases, external force abnormalities due to abnormal muscle activity were obscured by other forces in the unbiased metrics, but were revealed when these other forces were biased out in the passive-biased metrics.

To test for the possibility of learning over the course of ten trials, all of the metrics were calculated only considering the first five trials, and then only considering the last five trials. In some subjects, and in some movement directions, there was a significant increase in *Fmag* in the last five trials relative to the first five (t-test, $p < 0.05$). However, none of the other metrics showed any differences. Learning did not affect our major results: all of the significant results presented above remained significant, and the nonsignificant results remained nonsignificant, independent of whether all trials, the first five trials, or the last five trials were considered.

IV. DISCUSSION

The sensitivity of our metrics was low despite the fact that the subjects spanned nearly the entire range of FM scores. This was partially due to a ceiling effect; there was not a significant difference between the mildly impaired and normal subjects (Fig. 9). This ceiling effect is also consistent with the result that no ipsilateral abnormalities were observed. This might have been expected since the task was designed to measure only one aspect of lost motor function, impaired shoulder/elbow motor performance at low force levels during slow movements. Although the *FDE*'s we observed are likely to persist at higher force levels (other studies have reported scaling of the spatial pattern of muscle activity up to 50% of maximal voluntary contraction in stroke subjects [24]), increased sensitivity might have resulted from requiring subjects to generate larger forces. This would emphasize overall weakness, which many studies have demonstrated is a major aspect of lost motor function [25]–[28]. Increasing the movement speed and the number of movements tested, or testing the same movements as in the FM would likely also increase sensitivity. Given the large variability across stroke subjects, a more definitive measure of sensitivity would entail repeated testing of recovering subjects.

The low sensitivity in our metrics would indicate that, at present, they have limited usefulness for quantifying "improvement" in the FM sense. From theoretical considerations, it might be unreasonable to expect to find a motor performance metric that parallels FM scores perfectly. Since the FM scale is based upon the chronological progression of recovery typically observed in stroke patients, and not solely upon improved quality of movement, one might actually expect performance of many tasks to get worse before improving. For example,

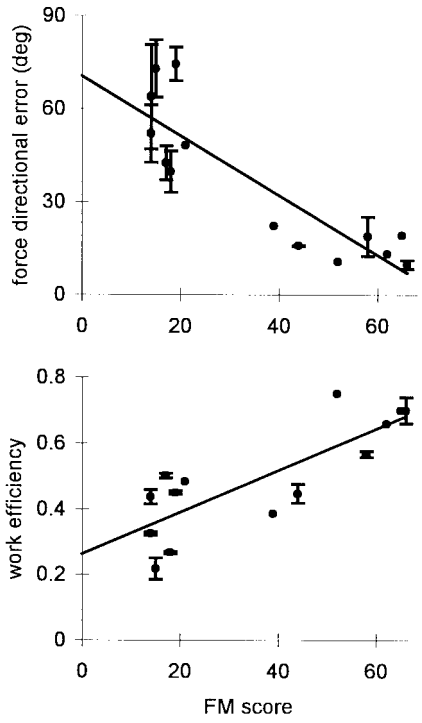


Fig. 9. *FDE* (paretic-normal) and work efficiency (paretic) versus FM score for 13 stroke subjects and one composite normal $(\mathrm{FM} = 66)$. Both of these are passive-biased metrics. For the eight stroke subjects who had two sessions within one week, the top error bar represents performance in one session and the bottom bar represents performance in the other session.

moving from a flaccid limb to a spastic or synergistic limb might result in larger misdirected forces in certain movements, even though according to the FM scale, there has been "improvement."

The six most severely impaired subjects had a small range of FM scores (14–19), but large ranges in the *FDE* and η metrics (Fig. 9). One possible explanation is that since the FM is a cumulative score, subjects with the same FM might have significant differences in shoulder and elbow motor function that are exhibited in our metrics. In fact, this spread in the data, coupled with the significant test–retest repeatability in all subjects, suggests that these metrics have the power to quantify differences between subjects, and changes within subjects that would not be measurable with the FM scale in the early stages of recovery. This capability at the low end of the scale would be especially useful given the apparent lack of measurement tools for severely impaired subjects (see literature review in Section I).

MIME can augment kinematic studies with the ability to detect changes in the attempted performance of a movement pattern, even in the early recovery stages before voluntary movement returns. As a patient recovers from a flaccid limb to trace levels of activation in a few muscles, MIME would be able to detect this change, even if there was not enough activity to initiate movement. For example, MIME was able to measure voluntary effort from subjects who were unable to initiate movement in the desired direction without assistance (subjects A, B, C, and E in Fig. 8). Thus, the MIME paradigm has essentially no floor effect, defined as the ability to measure changes at the low end of the scale (although determining which changes are "improvements" may not be possible). MIME can then retest a subject in the same movement pattern as recovery progresses, irrespective of the subject's changing level of motor function. Measurements of shoulder and elbow strength [25], [26] and coordination during isometric tasks [24], [29] can also detect performance changes in the early recovery stages. However, MIME has the potential to measure strength and coordination impairments during movement. The ability to generate force during movement may have greater functional importance than isometric tasks. MIME can also separate external force abnormalities due to muscle activity from abnormalities due to altered mechanical properties of the limb, both of which will change dramatically during the recovery period. This represents another advantage of the MIME paradigm over kinematic studies. For example, improved movement kinematics might be due to improved muscle activity and/or due to decreased passive tissue stiffness.

The abnormalities in passive forces could have been due to several mechanisms. The increased negative work in severely impaired subjects indicates hypertonia, which could be caused by hyperreflexia, abnormal descending commands or altered mechanical properties of the limb. The addition of EMG measures can help tease out the contributions of each mechanism. For example, force abnormalities due to altered mechanical properties of the limb can be measured by eliminating muscle activity during the movement with biofeedback or nerve block. The abnormalities in voluntary generated forces could be due to either paresis, inability to grade contractions, or incoordination. For example, the large directional errors observed in the severely impaired subjects could be due to paresis in selected agonists, while other muscles were activated at normal levels. Another possibility is that the agonists can be activated to normal levels, but only in conjunction with other inappropriate muscles, which would be considered a coordination impairment. Stroke subjects often show inappropriate coactivations in stereotypical flexor and extensor synergies [30], [31], and these synergies have been quantified at the muscle level during isometrics tasks [24], [29].

To summarize, preliminary data from 13 stroke subjects demonstrated the existence of performance metrics derived from the interaction forces during passive and active-assisted movements that correlate with FM scores. The more severely impaired subjects produced higher levels of negative work during passive movements. The metric that was most descriptive of the voluntary effort abnormality was the inability to generate a consistent force in the direction of movement, while eliminating forces in nonmovement directions. Sensitivity was low, indicating the metrics we derived would be poor indicators of improvement as defined by the FM scale. However, we expect increased sensitivity if more movements, faster movements, and higher force levels were tested. There was considerable spread in FDE and η across several severely impaired subjects with similar FM scores. This fact, coupled with the significant test–retest repeatability in all subjects, suggests MIME measurements can measure differences between subjects and changes within subjects during the recovery period following

the stroke or during therapeutic interventions that might be missed by the FM exam. Our data also indicate that external force abnormalities due to abnormal muscle activity can be separated from abnormalities related to altered mechanical properties of the limb, both of which will change dramatically during the recovery period.

The use of the robotic arm as the actuator allowed us to quickly prototype a research tool to test initial hypotheses. As a clinical tool, however, the cost of a robotic device and the time required to evaluate a subject compares poorly with other evaluation methods currently available. However, once the value of such measurements has been established and the salient elements of the device identified, MIME can be redesigned at much lower cost. A user-friendly interface would allow subjects to evaluate themselves without supervision, eliminating the disadvantage of the extra time required for the exam. In previous studies, therapists have expressed a general acceptance of robotic devices [32], [33].

There is growing interest in the potential therapeutic benefits of producing movement patterns in paretic limbs assisted by mechanical systems such as MIME [34]–[40]. An important pilot clinical trial has shown improved upper-limb motor function in a test group of acute stroke subjects who received robot assisted practice of 2-D planar arm movements in addition to their regular therapy [41]. Greater improvements might result from practice with a system flexible enough to handle as many functional movement patterns as possible. To this end, MIME has been redesigned with a larger, stronger robot (PUMA 560) capable of 3-D, faster movements, and clinical trials are underway for evaluating its therapeutic potential. We acknowledge that the cost and complexity of MIME are serious disadvantages when considering it as a clinical therapeutic device. However, once the movement patterns and modes of assistance that are most effective in restoring upper-limb function are identified, simpler devices can be developed.

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