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Control of Limb Dynamics in Normal Subjects and Patients Without Proprioception

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SUMMARY AND CONCLUSIONS

1. We recently showed that patients lacking proprioceptive input from their limbs have particular difficulty performing multijoint movements. In a pantomimed slicing gesture requiring sharp reversals in hand path direction, patients showed large hand path distortions at movement reversals because of failure to coordinate the timing of the separate reversals at the shoulder and elbow joints. We hypothesized that these reversal errors resulted from uncompensated effects of inertial interactions produced by changes in shoulder joint acceleration that were transferred to the elbow. We now test this hypothesis and examine the role of proprioceptive input by comparing the motor performance of five normal subjects with that of two patients with large-fiber sensory neuropathy.

2. Subjects were to trace each of six template lines presented randomly on a computer screen by straight overlapping out-and-back movements of the hand on a digitizing tablet. The lines originated from a common starting position but were in different directions and had different lengths. Directions and lengths were adjusted so that tracing movements would all require the same elbow excursion, whereas shoulder excursion would vary. The effects of varying interaction torques on elbow kinematics were then studied. The subject's dominant arm was supported in the horizontal plane by a low-inertia brace equipped with ball bearing joints and potentiometers under the elbow and shoulder. Hand position was monitored by a magnetic pen attached to the brace 1 cm above a digitizing tablet and could be displayed as a screen cursor. Vision of the subject's arm was blocked and the screen cursor was blanked at movement onset to prevent visual feedback during movement. Elbow joint torques were calculated from joint angle recordings and compared with electromyographic recordings of elbow joint musculature.

3. In control subjects, outward and inward paths were straight and overlapped the template lines regardless of their direction. As prescribed by the task, elbow kinematics remained the same across movement directions, whereas interaction torques varied substantially. The timing of the onsets of biceps activity and the offsets of triceps activity during elbow flexion varied systematically with direction-dependent changes in interaction torques. Controls exploited or dampened these interaction torques as needed to meet the kinematic demands of the task.

4. In contrast, the patients made characteristic errors at movement reversals that increased systematically across movement directions. These reversal errors resulted from improper timing of elbow and shoulder joint reversals. Instead of adapting biceps and triceps activity to direction-dependent changes in interaction torques, the patients cocontracted antagonists throughout the reversal phase. Although this may have increased joint stiffness, the strategy was not effective in controlling elbow dynamics: elbow joint acceleration varied directly with the amplitude of the interaction torques. Interaction torques, transferred to the elbow by upper arm deceleration, drove the elbow into flexion prematurely. This decoupled the normally synchronous reversals at the shoulder and elbow and resulted in large hand path distortions at movement reversals.

5. Our data indicate that interaction torques are normally controlled through feedforward mechanisms and that this control is severely impaired in patients deprived of proprioception because of sensory neuropathy. We therefore conclude that proprioceptive information plays an important role in interjoint coordination during multijoint movements. We hypothesize that information during movement serves to update an internal model of limb dynamics that is then used to program motor commands.

INTRODUCTION

Patients who are functionally deafferented by sensory neuropathy, and whose ability to detect the motions of their joints is impaired, also show marked degradation in the accuracy of movements aimed to visual targets. This degradation is particularly severe in the case of multijoint movements, manifesting itself both as increased trajectory variability and as errors that vary systematically with movement direction (Ghez et al. 1990; Gordon et al. 1995). For example, such patients make direction-dependent errors in the extents and directions of planar reaching movements (Ghez et al. 1990). These errors have been shown to result in part from a failure to program movements in accord with directional variations in the inertial resistance of the multijoint limb (Ghez et al. 1990, 1995; Gordon et al. 1987, 1990). Similarly, we have found that deafferented patients show prominent anomalies in the performance of unconstrained, three-dimensional movements (Sainburg et al. 1993b). Control subjects performed the pantomimed gesture of slicing a loaf of bread as a sequence of linear out-and-back motions of the hand that were largely confined to a single plane. In patients, however, the movements were neither linear nor planar and direction reversals from the outward to the return phase were dramatically distorted: instead of a sharp reversal, the hand path was rounded. This resulted from a failure to coordinate the normally synchronous reversals of individual shoulder and elbow joint movements.

Several considerations led us to suggest that, like the extent errors of reaching movements, these hand path anomalies also resulted from a failure to adapt motor commands to certain biomechanical properties of the limb. In particular, the clustering of these errors at the end of the outward phase of movement suggested that they resulted from a failure to control the forces that are transferred between limb segments

during multijoint movements. Hollerbach and Flash (1982) first demonstrated that failure to control interactions between limb segments might lead to movement errors. These authors showed through simulations that removal of the terms associated with these interactions from the equations of joint torque led to curved and inaccurate hand paths.

The dependence of the interaction torques at the elbow on shoulder joint velocity and acceleration and on the elbow joint angle[1] suggested that these particular interactions contributed to the reversal errors made by deafferented patients. Unfortunately, however, the complex three-dimensional nature of our task made it difficult to determine the contributions of uncompensated interaction torques and of other biomechanical factors to the deficits in interjoint coordination.

The purpose of the present study is to determine the role of intersegmental interactions and their control in reversal movements analogous to those examined earlier. We eliminated the effects of gravity by constraining movements to the horizontal plane and supporting the limb in a low-inertia, low-friction device and restricted movements to the shoulder and elbow. By using a task in which subjects were to move their hand along a prescribed path, serving as a template for the movements, we were able to adjust the required amplitude of shoulder and elbow excursions. We used templates of different directions and extents, varying the required shoulder excursions while maintaining the elbow excursions constant. Because the interaction torques at the elbow vary with shoulder movement, we were able to study how subjects regulate elbow kinematics when the interaction torques at the elbow varied.

We expected that to make accurate movements, subjects would need to account for directional variations in elbow joint interaction torques in their neural commands to muscles. The question therefore arose of the relative contributions of passive mechanical interactions and active muscle contractions to elbow kinematics. Bernstein (1967) hypothesized that control of such "reactive phenomena" would be accomplished by precise coordination of muscle timing, suggesting that these passive interactions could be specifically dampened or made use of to drive the movement. Alternatively, Bernstein suggested that such interactions might be controlled in a nonspecific manner by modulating joint stiffness via cocontraction of antagonist muscles. To address this question we first partitioned joint torques using a modification of the approach advocated by Smith and Zernicke (1987). This allowed us to compare the mechanical contribution of muscles and of interaction torques to the observed kinematics. Then, by recording myoelectric activity from elbow muscles, we assessed the contributions of cocontraction and reciprocal mechanisms to this control.

We now address the following questions. *1*) Do the interjoint coordination deficits and ensuing reversal errors in deafferented patients result from the action of uncompensated interaction torques at the elbow joint? *2*) Is the temporal sequence of muscle activities at a joint normally matched to variations in interaction torques, or are these interactions generally dampened by cocontraction? *3*) Do the electromyographic (EMG) patterns in deafferented patients reflect a strategy for controlling interaction torques that is qualitatively similar to that used by controls? Our findings indicate that control subjects adapted the timing of agonist and antagonist muscle activity to direction-dependent changes in elbow joint interaction torques. However, patients were unable to do this. As a result, they made direction-dependent errors at movement reversals that reflected the uncompensated action of elbow joint interaction torques. Preliminary accounts of this work have been published in abstract form (Sainburg et al. 1992) and presented at a symposium (Ghez and Sainburg 1994).

[1] These relations are true for planar movements of the arm (Hollerbach and Flash 1982) and for three-dimensional movements when the shoulder angle is defined relative to the projection of the upper arm in the moving plane passing through the shoulder, elbow, and wrist joints (Schneider and Zernicke 1990).

METHODS

Subjects

Subjects were five neurologically normal adults (3 males, 2 females, aged 27–53), and two patients (*MA,* 43-year-old right-handed female, and *CF,* 62-year-old left-handed male) with severe large fiber sensory neuropathies affecting both upper extremities as well as their trunks and lower extremities. In both patients, the etiology of the disease has not been determined and the disease has not progressed for several years. Both patients had complete loss of position, vibration, and discriminative touch sensation throughout their upper extremities, bilaterally. *Patient CF* was able to detect vibration just over the scapulae, medial to the glenohumeral joint. However, the sense of movement and position of the upper arm was absent. Pain, temperature, and coarse touch were preserved and sensory nerve conduction was slowed. These findings are consistent with selective loss of the large-diameter afferent fibers. Somatosensory evoked potentials from upper and lower extremities were absent. Muscle strength and EMG were normal. The degree and distribution of sensory loss was similar for both patients through the upper extremities; however, lower extremity involvement was more severe in *patient CF. MA* was ambulatory with use of a wide base stance, *CF* required a wheelchair for mobility. A more detailed description of the medical history and status of these patients is reported elsewhere (Sainburg et al. 1993b).

Apparatus

Figure 1 illustrates the experimental setup. Subjects sat facing a computer screen with their dominant arm supported over a digitizing tablet by a low-inertia brace equipped with ball bearing joints that were placed directly under the shoulder and elbow. A magnetic pen attached to the brace 1 cm above the digitizing tablet allowed the subject's hand position to be monitored and displayed as a screen cursor. The ball bearing joints were connected by thin steel rods that were rigidly attached to each limb segment with thermoplastic splinting material. The rod supporting the upper arm was adjustable to allow its length to be precisely matched to the upper arm of the subject. A thermoplastic splint was fitted to the subject's forearm and hand, immobilizing all joints distal to the elbow. The forearm was maintained in full supination and the subject's trunk and scapula were immobilized by a brace. Movements of the arm were thus restricted to the shoulder and elbow joints and to the horizontal plane. Two precision, single-turn, linear potentiometers (Beckman Instruments) were used to monitor the elbow and shoulder joint angles. Experiments were controlled using one computer (Apple Macintosh SE30), whereas data were collected and analyzed using another (Apple Macintosh II). Computer routines for data analysis were written in the IGOR (Wavemetrics) programming language.

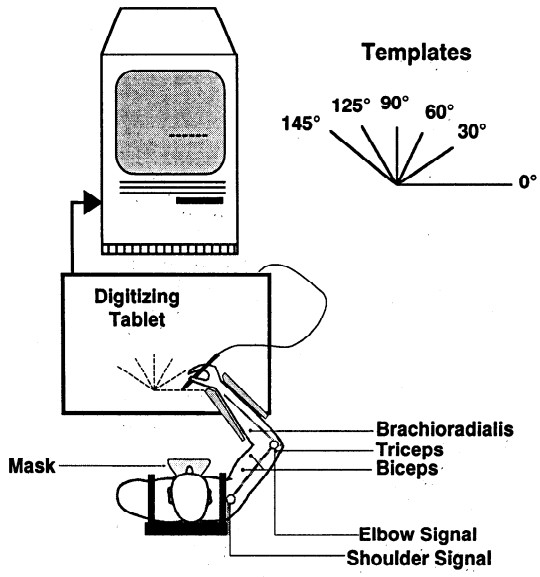

FIG. 1. Experimental setup. The subject's arm was supported in a brace with ball bearing joints under the shoulder and elbow that were attached to precision potentiometers. Triceps brachii, biceps brachii, and brachioradialis electromyogram (EMG) were recorded with bipolar surface electrodes. The position of a magnetic pen that was attached to the brace, 2 cm above a digitizing tablet, was used to monitor hand position. The position of the pen could be displayed on the computer screen as a cursor. A mask was used to block vision of the arm while allowing vision of the screen. One of six template lines, shown at *right,* were displayed in random order on the screen.

Task

Subjects were to trace a template line presented on the screen, using a single overlapping out-and-back movement of the hand. The lines were presented one at a time in pseudorandom order, such that no line was repeated in any two consecutive trials. As illustrated in Fig. 1, the lines projected in each of six directions from a common starting position. At the presentation of a tone, subjects were to trace the template line at a comfortable speed as long as the movement was completed within the sampling window (2.5 s). They were instructed to focus on making their movements straight and to retrace forward and backward motions. Vision of the hand and arm was blocked during and between movement trials and the cursor was blanked at movement onset. This assured that subjects could not use visual feedback to correct their movements. Movement paths were displayed on the computer screen at the end of every third trial. This form of knowledge of results sustained subjects' motivation and maintained stable performance. Each experimental session comprised 30 trials, with rests of approximately 10 s between trials. These relatively long intertrial intervals prevented subjects from becoming fatigued. Before the initiation of data collection, all subjects practiced for 15 trials with full vision of their limbs. This was done to familiarize subjects with the apparatus and experimental protocol.

To vary the amplitude of elbow joint interaction torques systematically, subjects were presented with templates lines that required movements with varying amounts of shoulder joint excursion. These lines were oriented toward the following directions with respect to the frontal plane of the subject: 0°, 30°, 60°, 90°, 125°, and 145°. In right-handed subjects, the 0° target line was directed to the right (3 o'clock direction) from an origin in front of the subject's right shoulder. Other target lines originated in the same location but were directed in progressively counterclockwise directions. In left-handed subjects this was reversed to maintain among different subjects the same geometric relationship between the joint angles and the required hand paths. In these subjects, who included *patient CF,* right and left were inverted in hand path plots. The length of each template line was calculated such that the elbow joint angular excursion required to make an accurate movement was similar, whereas the required shoulder joint angular excursion increased systematically across target directions. Figure 2 illustrates this by typical limb trajectories performed by a control subject toward 0° (*left*) and 125° (*right*). Stick figure representations of the upper arm and the forearm/hand segment are drawn every 40 ms to the end of the outward portion of the trajectory. As seen in Fig. 2, although the excursions at the elbow joint were similar for both movements, the shoulder excursions were quite different.

EMG data

EMG activity was recorded from three muscles—the biceps brachii, a flexor of the elbow (and to a lesser degree of the shoulder); the brachioradialis, a flexor of the elbow; and the triceps brachii, an extensor of the elbow (and to a lesser degree of the shoulder). The electrode position was determined according to two criteria: *1*) maximum EMG activity during isolated flexor or extensor movements of the elbow joint and *2*) electrical silence or minimal EMG activity during isolated movements of the shoulder joint. The possibility of crosstalk was minimized by demonstrating that electrical silence was maintained during activation of antagonistic muscles. EMG was recorded with active, bipolar, stainless steel surface electrodes (Liberty Mutual MY0111) with a bandpass of 45–550 Hz. The electrode contacts were 3 mm diam and spaced 13 mm apart. The EMG signals were digitized at 1,000 Hz via a BioPacq MP-100 A-D converter. During recording, the EMG signals were displayed simultaneously on a computer screen and on an oscilloscope to verify digitized recordings. The EMG signals were full-wave rectified and stored on disk for later analysis.

The times of onset and termination of muscle activity were determined in relation to the initiation of flexor acceleration at the elbow (acceleration zero crossing). Onset and offset were identified using an interactive computer graphics program written within IGOR. The algorithm placed a cursor at the bin at which the average EMG (within a moving 30-ms window) exceeded a critical amplitude, defined as 4 times the SD of the lowest average activity recorded over any 50-ms window during that trial. The program allowed for visual determination of whether the chosen onset was appropriate and manual correction when needed. The termination of triceps activity was determined by a similar method. However, the algorithm worked backward in time from the second zero crossing of elbow angular acceleration (see also Karst and Hasan 1991b).

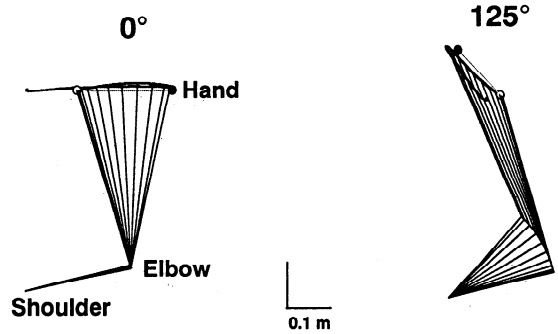

FIG. 2. Shoulder excursion varies with target direction. Shoulder, elbow, and hand coordinates of movements performed toward 0° and toward 125°. Stick figures of upper arm and forearm are drawn every 40 ms during the outward portion of the movement. Template lines are also shown, the beginning and end of which are marked with open and closed circles for clarity. These circles were not present in the displays presented to subjects.

The forearm was splinted in 90° supination to minimize activity in brachioradialis and thus reduce the distribution of flexor muscle activity across multiple muscles. We thus expected to maximize the agreement between our recorded biceps EMG and our calculated muscle torques. In all subjects, brachioradialis either remained silent or exhibited the same pattern of activity as recorded in biceps brachii.

Kinematic data

Potentiometer signals were digitized at 1,000 Hz, low-pass filtered at 12 Hz, and differentiated twice to yield angular velocity and angular acceleration values for the elbow (ϕ) and shoulder (θ) joints. The 180° complement of the inside angle between the upper arm and forearm was taken as the elbow angle. The data were edited to remove irrelevant portions using two critical values to identify the initiation and end of the movement. Because the task was designed to control elbow joint kinematics, movement initiation was taken at a point 50 ms before elbow acceleration reached 1% of its maximal value. The final point was taken at 50 ms after 1% of the peak elbow joint deceleration. This ensured that the full elbow angular trajectory was included in each trial.

Hand paths were calculated from joint angle data by using the measured lengths of the upper arm and forearm/hand segments, respectively. The latter was measured as the distance from the axis of the elbow joint to the position of the magnetic pen. The angular data were transformed to a Cartesian coordinate system with the origin at the shoulder, the x axis along the lateral dimensions and the Y axis along the anterior posterior dimensions.

We defined the reversal phase as beginning at the peak in tangential hand velocity within the outward phase of movement and ending at the peak in tangential velocity during the return phase. The area of the hand path lying within this reversal phase was used as a measure of the accuracy with which the subjects aligned the outward and inward portions of the movement path. This was determined, using *Eq. 1,* by computing the area between two vectors. Vector 1 (V1) was calculated from the hand position at the first peak in tangential hand velocity ($i = 0$) during the outward motion to the next hand path point. Vector 2 (V2) was then calculated from the hand position at $i = 0$ to the hand path point at $i = 1$. This procedure was applied for every consecutive pair of points until the peak in tangential velocity of the return movement ($i = n$). Then all areas were summed to yield the total area within the reversal phase of motion, as shown in *Eq. 1.*

$$\text{Path Area} = \sum_{i=0}^{n} [(\text{V1}_i \, \text{V2}_i) \sin \theta]/2$$

$$\text{Angle } \theta = \text{the angle between V1 and V2} \qquad (1)$$

Anthropometric data

The upper arm and forearm/hand segment lengths were measured. Segment mass, center of mass, and inertia were then calculated on the basis of the subject's weight and segment lengths using the regression equations compiled by Winter (1990). The moment of inertia of the segment of the apparatus supporting the forearm was determined by constructing a vertical pendulum using this segment of the apparatus. The oscillations of the pendulum were recorded by connecting a potentiometer to the ball bearing pivot of the pendulum. The moment of inertia of the apparatus was calculated using the frequency of the pendulum, averaged over the first second of movement. The mass, center of mass, and inertia of the forearm-hand segments for all subjects, and for the corresponding part of the apparatus are shown in Table 1.

TABLE 1. *Estimates of mass and inertial characteristics of the forearm/hand segment*

Subject	Mass, kg	Center of Mass, m	Moment of Inertia, kg m^2
RLS	1.74	0.18	0.026
CG	1.92	0.21	0.028
SS	1.43	0.19	0.025
LN	1.15	0.19	0.020
MFG	1.08	0.15	0.012
MA	1.21	0.20	0.022
CF	1.50	0.15	0.016
Device (distal segment)	0.65	0.20	0.009

Calculation of interaction torques

Using our recorded joint angles and the morphometric data shown above, we computed the joint torques acting at the elbow and partitioned the terms into three categories: *1*) the *self torque,* representing the inertial resistance of the forearm to elbow acceleration; *2*) the *interaction torque,* representing the torques imposed by movement of the shoulder joint; *3*) the *generalized muscle torque,* representing the torques resulting from active muscle contraction as well as from the passive resistance due to deformation of muscles and connective tissue. The latter is a residual term computed, based on D'Alembert's principle, from the equilibrium equation shown below

$$R_2 - A\ddot{\phi} - (A + BL_1 \cos \phi)\ddot{\theta} - (BL_1 \sin \phi)\dot{\theta}^2 = 0 \qquad (2)$$

The first term, R_2, in *Eq. 2* is the generalized muscle torque; the second term, $-A\ddot{\phi}$, is the self torque and can be seen to vary inversely with elbow joint acceleration.[2] The interaction torque, $-(A+BL_1 \cos\phi)\ddot{\theta} - (B L_1 \sin\phi)\dot{\theta}^2)$, includes a combination of terms that vary with the square of shoulder angular velocity and with shoulder angular acceleration (See Table 2).

As in the partitioning scheme used by Smith, Zernicke, and coworkers (Hoy and Zernicke 1985, 1986; Koshland and Smith 1989a; Schneider and Zernicke 1989, 1990; Schneider et al. 1989; Smith and Zernicke 1987), this separates the potential causes of motion at a particular joint, the generalized muscle and interaction torques, from the reaction to movement or the self torque. Here it is possible to determine the action of either the muscle or interaction torque at a joint by comparing the sign of the torque component to that of the self torque. For example, if the generalized muscle torque is opposite in sign to the self torque, its action is to accelerate the angular motion of the joint. However, if the sign of the muscle torque is the same as that of the self torque, its action is to counter the acceleration caused by the interaction torque. By comparing the relative magnitudes and waveforms of these torques, it is possible to assess the contribution of each component to the resulting changes in joint kinematics. Other differences between the partitioning scheme used here and that used by Smith and Zernicke (1987) are considered in the discussion.

Statistics

The Mann-Whitney U test was used to test the significance of the difference between data sets. This nonparametric test was chosen because of the small sample sizes when comparisons were made between subgroups of data within a single subject's movements. This test was also used for analysis of larger sample sizes to eliminate the assumptions regarding population distributions required

[2] The separation of a distinctive "self torque" including all inertial terms that vary with the acceleration of the joint in question was introduced by S. Cooper (personal communication).

TABLE 2. *Symbols for equation of elbow joint motion*

Symbol	Description
I	Limb segment Inertia
m	Limb segment mass
r	Distance from proximal joint to limb segment center of mass
L	Limb segment length
θ	Shoulder joint angle
ϕ	Elbow joint angle
I_d	Device inertia (forearm segment)
m_d	Device mass (forearm segment)
r_d	Distance from elbow to device center of mass
A	$I_2 + m_2 r_2^2 + I_d + m_d r_d^2$
B	$m_2 r_2 + m_d r_d$

Subscripts 1 and 2 denote upper arm and forearm segments, respectively.

in parametric tests. We used a simple linear regression analysis to assess the relationships between data sets. To reduce the probability of type I errors, we divided the α value (0.05) by the number of tests (55). Thus we used an α value of 0.001, which provided a stringent and conservative test for our analyses.

RESULTS

Deafferented patients make errors at movement reversals

Figure 3*A* shows examples of reversal movements made by two control subjects (*left*), *MFG* and *CG*, and both deafferented patients (*right*), *MA* and *CF*. The hand paths consisted of an outward phase in which the movement was directed distally along the template and a return phase in which the movement was directed back toward the starting point.

Although the subjects could not see their hands or the screen cursor during movement, the initial direction of movement varied with that of the templates in both patients and control subjects. As seen in these examples, the differences in trajectories between patients and controls became striking at movement reversals. In control subjects, direction reversals were sharp and the return phase of the hand path closely paralleled the outward phase. In the patients, movement reversals were severely distorted, showing large medially directed curves, and return directions no longer matched those of the templates. As can be appreciated in Fig. 3*A*, these reversal errors were dependent on the direction of hand movement, becoming greater for movements made toward targets in more counterclockwise directions.

We used two measures to characterize hand path accuracy: the angular deviation in the hand path between the outward and return phases, and the area circumscribed by the path during the reversal phase. The former indicates the change in direction of the hand path over the course of movement reversals and the latter resolved the degree of sharpness in the reversal. Figure 3*B* illustrates our measure of hand path angular deviation. The angle of the hand path was calculated at the beginning and at the end of the reversal phase. These two measures were taken as the angle of the vectors (V1 and V2) originating at the start location and ending at the hand locations at the peaks in tangential velocity defining the beginning and the end of the reversal phase, respectively. The difference in these angles ($\Delta\theta$) indicates the change in direction of the hand path during the reversal phase. A difference of 0° indicates that the path completely reversed direction, whereas larger values indicate incomplete reversals. Positive values indicate a counter-clockwise deviation from the direction of outward movement, whereas negative values indicate a clockwise deviation.

Figure 3*B* shows the mean ± SE of the angular deviation in the hand path during the reversal phase. Data have been averaged across all 30 trials of movement made by all control subjects together and each deafferented patient separately. Because the hand paths of controls closely paralleled the template lines throughout the movement, the angular deviation was also small. In the patients, however, a large counterclockwise deviation in the hand path angle occurred during the reversal phase. The mean hand path deviation during the reversal phase was only 7 ± 0.6°, mean ± SE, for control subjects, compared with 53 ± 6.5°, mean ± SE for *CF* and 143° ± 15°, mean ± SE, for *MA*.

We next determined the dependence of path distortions during reversals on movement direction by computing the area circumscribed by the hand path during the reversal phase of movement (i.e., between the peaks in hand velocity in the outward and return phases of motion). Figure 4*A* shows hand paths (*left*) and tangential velocities (*right*) for a movement in the 125° direction performed by a control subject (*MFG, top*) and by a patient (*CF, bottom*). The peaks in tangential velocity on the way out and on the way back are identified by open and filled arrowheads, respectively, and the areas forming the reversal errors are shown in gray. The relationship of this error to the direction of movement prescribed by the template is illustrated for the two patients and all controls in the box-and-whisker plots of Fig. 4*B*. To increase the number of trials used for this comparison, the data for pairs of templates in adjacent directions were grouped. Reversal areas are seen to be substantially larger in the patients than in controls ($U = 202$, $P \leq 0.001$) and increase progressively across direction groups. Thus, in patients, the reversal areas of the 125° and 145° movements were substantially larger than those of the 0° and 30° movements (*MA*, $U = 4$, $P \leq 0.001$: *CF*, $U = 3$, $P \leq 0.001$).

Reversal errors reflect deficits in interjoint coordination

In a previous study of three-dimensional movements, we showed that for hand paths to reverse direction sharply, the shoulder and elbow joints must reverse direction in close synchrony (Sainburg et al. 1993b). We therefore sought to determine whether the reversal errors in the patients observed here might also result from improper timing of the movement reversals at the shoulder and elbow. Because in the present study the relative contribution of shoulder and elbow motions to the hand paths varied with template direction, we first needed to identify those trials that incorporated substantial motion at both joints. We thus measured peak joint excursion as the difference between the initial joint angle and the peak extensor angle for movements initiated toward extension, or the peak flexor angle for movements initiated toward flexion. Figure 5 displays the ratio of peak shoulder to elbow excursion, group averaged (mean ± SE) for all movements made toward individual templates by each patient and by all control subjects. As expected, in all subjects the shoulder/elbow excursion ratio increased monoton-

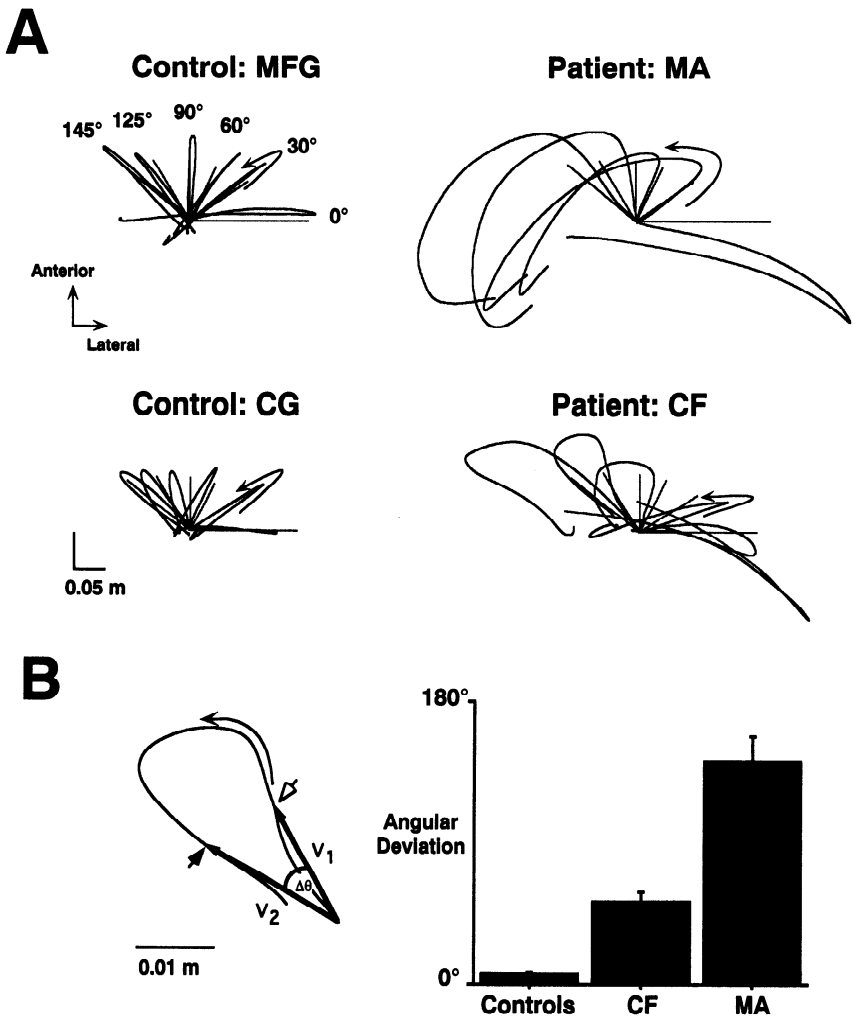

FIG. 3. Hand paths and reversal errors in controls and deafferented patients. *A*: representative hand paths from 2 controls (*MFG* and *CG*) and both patients (*MA* and *CF*) are shown drawn over the template lines (gray) toward each of 6 directions. The direction of movement is marked by the curved arrow for the 30° movement. *B*: our measure of angular deviation during the reversal phase. *Left*: hand path from a movement made toward 125° by patient CF is shown. The 2 vectors used to measure hand path angle are drawn from start position to the position of the hand at the peak in tangential velocity during the outward phase [open arrow, vector 1 (V1)] and the return phase [closed arrow, vector 2 (V2)]. The angle between these vectors ($\Delta\theta$) is our measure of angular deviation during the reversal phase (see text and Fig. 4 for criteria used for marking the reversal phase). *Right*: mean ± SE hand path angular deviation for all trials made by all controls (together) and each patient (separate).

ically as the angle of the template increased. This reflects a progressive increase in required shoulder flexion. Because, in movements with smaller excursion ratios the hand path was predominately dependent on motion at only one joint, we selected for analysis only movements toward directions in which all subjects had mean excursion ratios of >0.25 (nonshaded region). As shown in Fig. 5, this included the 60°, 90°, 125°, and 145° directions.

Figure 6 shows representative movements toward the 125° target by a control subject (*MFG, left*) and by a patient (*MA, right*). It can be seen that the movement is initiated by simultaneous shoulder flexion and elbow extension, which act to propel the hand outward over the template line. The sharp reversal in the hand path of the control subject (Fig. 6*B, left*) was produced by nearly synchronous direction reversals at the elbow and shoulder. In the patient (Fig. 6*B, right*), the beginning of the reversal error in the hand path corresponds to the peak elbow extension angle (bold stick figure at open arrowhead), which precedes the reversal in shoulder joint motion (bold stick figure at filled arrowhead in Fig. 6*A*) by 116 ms. Thus the elbow was beginning to accelerate into flexion while the shoulder was still flexing. The concurrent flexion at shoulder and elbow resulted in the large medial deflection of the hand, shown in Fig. 6*A* (*right*) as the portion of the trajectory between the two bolded stick figures.

The coupling interval was measured from the time the elbow angular velocity crossed zero (open arrow), as it went from extension to flexion, to the time the shoulder velocity crossed zero (filled arrow), going from flexion to extension. Figure 6*C* shows the distributions of coupling intervals for controls (*left*) and patients (*right*). It can be seen that in control subjects, direction reversals at the shoulder and elbow were tightly coupled, varying little from the median value of −8 ms. By contrast, in patients, the distribution of coupling intervals was very broad with a median value of −84 ms. Thus controls accurately retraced the target lines and sharply reversed the direction of hand motion by synchronizing individual joint reversals. Hand path reversals were distorted in the patients because motions at the shoulder and elbow joints were not coordinated.

Interjoint coordination deficits result from failure to take account of directional variations in interaction torques at the elbow joint during movement reversals

Why should the abnormalities in the patients' hand paths and interjoint coordination be particularly prominent at

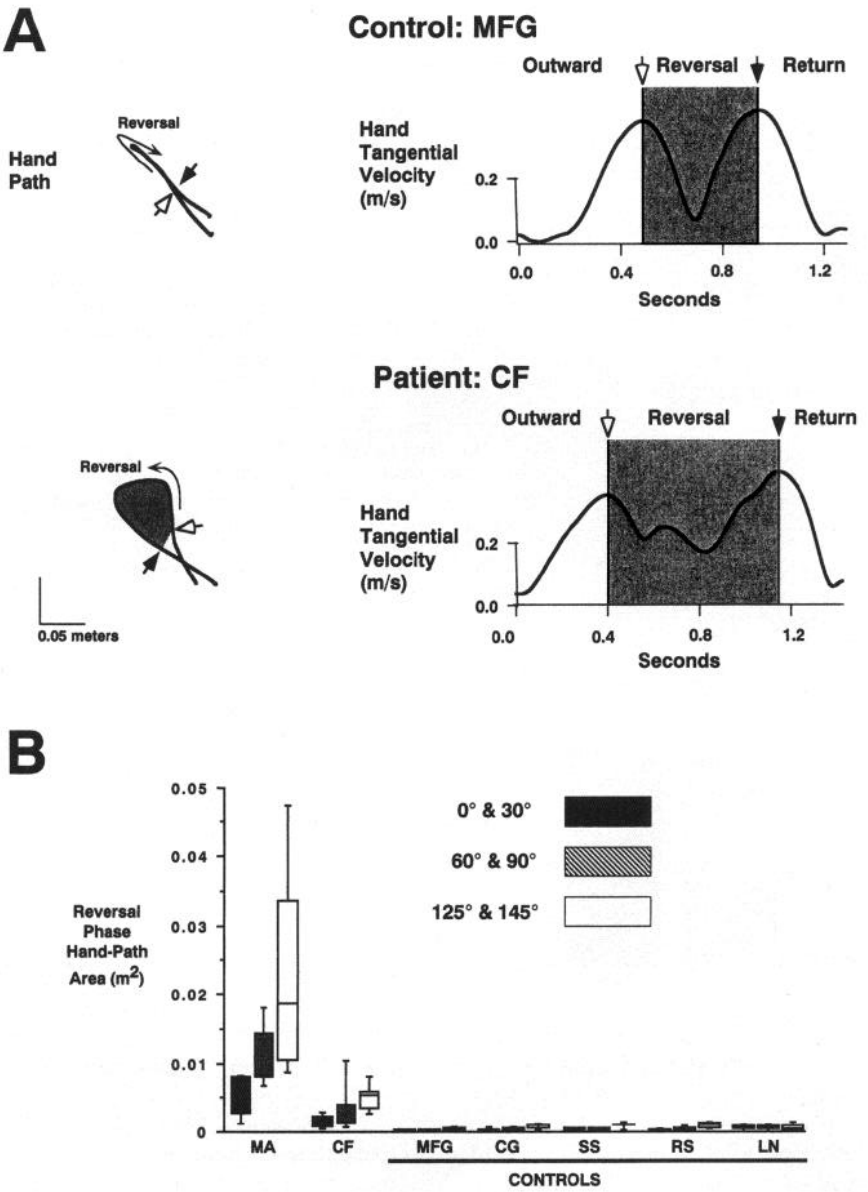

FIG. 4. Variation in reversal errors with movement direction. *A*: on the *left*, sample paths from *control MFG* and *patient CF* are shown with hand path areas circumscribed during the reversal phase (gray). Curved arrow: direction of movement. On the *left*, the corresponding tangential velocity profiles have been separated into outward, reversal, and inward phases that are delimited by the initial (open arrow) and final (closed arrow) peaks in tangential velocity. The positions of the hand that correspond to these critical points are marked by open and closed arrows on the hand paths at the *left*. The area circumscribed by the path within this phase is shaded. *B*: median (horizontal line), interquartile ranges (bar), and the full positive and negative range (whiskers) of hand path areas are shown for each subject separately. The data for pairs of templates were grouped as shown.

movement reversals? During movement reversals, the shoulder and elbow joints undergo large angular accelerations as joint motions decelerate in one direction and accelerate toward the opposite direction. As discussed in the INTRODUCTION, acceleration of any one joint produces torques at all other joints of the limb. However, limb inertia is greater at proximal than at distal joints and acts to resist acceleration. As a result, the effects of interaction torques should be greater at distal than at proximal joints. Based on *Eq. 2,* we expected that the large angular accelerations at the shoulder during movement reversals might produce significant flexor interaction torques at the elbow. We therefore hypothesized that while controls might be able to compensate for this effect, patients might not. The flexor interaction torque at the elbow might lead to the premature flexion that gave rise to the patients' reversal errors.

To test this hypothesis, we computed the torques acting at the elbow joint. Using a modification of the scheme proposed by Smith, Zernicke, and colleagues (Hoy and Zernicke 1986; Schneider and Zernicke 1990; Schneider et al. 1989; Smith and Zernicke 1987), we subdivided the torque at the elbow into three components—self torque, interaction torque, and generalized muscle torque (see METHODS for more detail). The interaction torque at the elbow is dependent on the acceleration and velocity of the shoulder joint. Because shoulder excursion increased progressively across movement directions, we expected that the interaction torque would become progressively larger in movements aimed at targets in successively more counterclockwise directions. Figure 7 shows ensemble averages of shoulder and elbow angles, elbow joint torques, and EMG records for movements made over the 0° template and the 125° template by a control subject (*RLS*). The interval of flexor acceleration, which encompasses the hand path reversal, is shaded. As prescribed by the task, the elbow angular trajectory is similar in both movements. In

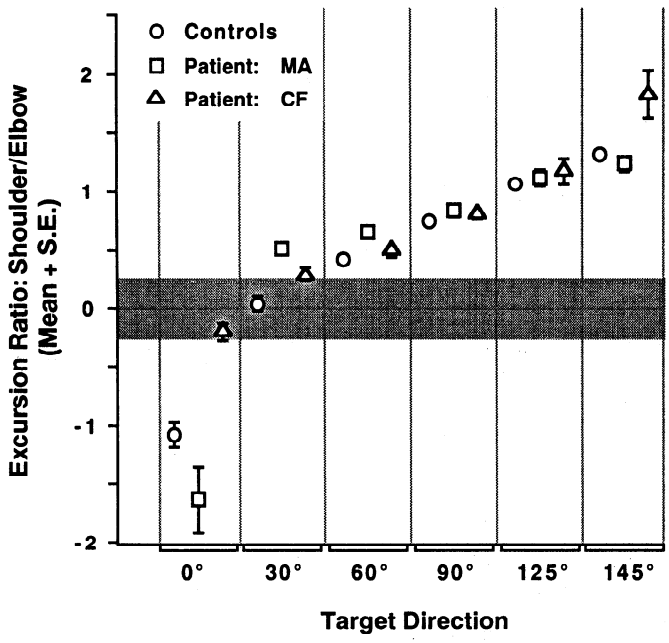

FIG. 5. Variation in shoulder to elbow excursion ratio across movement direction. The mean ± SE excursion ratio for trials made toward each template by all controls (together) and each patient is shown. Positive values indicate elbow flexion with shoulder flexion: negative values indicate elbow flexion with shoulder extension. Because the task required similar elbow excursions for all movements, the variation in excursion ratio primarily reflects changes in shoulder excursion. For our analysis of interjoint coupling, only movements toward directions in which all subjects had mean excursion ratios of greater than 0.25 were considered. The gray area marks excursion ratio values of ±0.25.

contrast, shoulder joint motion was small in the 0° movement and large in the 125° movement.

The elbow torques responsible for these movements are plotted under the angular trajectories; biceps brachii (flexor) and triceps brachii (extensor) EMG records are shown at the *bottom* of Fig. 7. It should be noted that the self torques (Self, thin line), which vary with elbow acceleration, have similar profiles in the two movements, whereas the interaction (Inter, bold line) and generalized muscle torques (Mus, - - -) are markedly different. In the 0° movement, very little shoulder joint excursion occurred, resulting in a very small elbow interaction torque. Thus the waveform of the generalized muscle torque is similar to, but of opposite sign to the self torque. In agreement with this, elbow flexor EMG activity (biceps) begins and extensor activity terminates just before the onset of elbow flexor acceleration (shaded region).

In contrast, in the 125° movement shoulder joint motion was substantial and gave rise to a large interaction torque at the elbow joint. During elbow flexor acceleration (shaded region) it acted to flex the elbow and its magnitude exceeded that of either the self torque or the generalized muscle torque. The muscle torque was now in the extensor direction and acted to dampen the driving action of the interaction torque. Accordingly, the initiation of elbow flexor acceleration was accompanied by triceps EMG. Biceps EMG began some 200 ms later, about at the peak in flexor acceleration. Because the net effect of muscle action was still extensor, it is likely that biceps activation served to counter remaining contractile and elastic forces in the triceps.

Figure 8 shows ensemble averages of *patient MA*'s shoulder and elbow angular trajectories, elbow torque, and EMG for all five movement trials aimed over the 0° (*left*) and the 125° (*right*) templates. As in the control, shoulder excursion varied substantially between the two directions. However, instead of remaining constant, elbow excursion also increased significantly. The differences in the elbow self torque, which varies with elbow angular acceleration, reflects the different elbow angular trajectories in the two directions. The large difference in shoulder joint excursions leads to similar differences in interaction torque amplitude, which is small at 0° and large at 125°.

In the patient, the generalized muscle torques are not matched to the directional differences in interaction torques. In the 0° movement, the interaction torque is small and, like the self torque, extensor during flexor acceleration. Both act to resist the movement imposed by the generalized muscle torque. As was the case for the control, elbow flexion is driven by flexor muscle action. Also as in the control, at 125° the large excursion at the shoulder joint gives rise to a large flexor interaction torque (thick line) at the elbow during elbow flexor acceleration (shaded). However, instead of countering the interaction torque at the onset of flexor acceleration, the generalized muscle torque is also flexor. This adds to the effect of the interaction torque and results in premature and excessive flexor acceleration of the elbow (and a correspondingly large self torque). The patient's failure to adapt muscle actions to directional variations in interaction torque is also apparent in the EMGs on the *bottom* of Fig. 8: very little change in the EMG pattern between the two directions can be seen. In both movements, flexor activity (biceps) is initiated just before the onset of elbow flexor acceleration and extensor activity (triceps) continues throughout the flexor acceleration phase.

In addition to errors at movement reversals, the failure of the patients to maintain constant elbow excursions across directions may also have resulted from a failure to control elbow joint interaction torques. As can be seen in Figs. 7 and 8, the magnitude of the interaction torque during the initial, outward phase of movement varied significantly across directions for the patient and control subject. In the 0° movements, the interaction torque is initially close to 0 and does not increase substantially until the flexor acceleration phase of motion. However, in the 125° movements, the interaction torque becomes large in the extensor direction during the outward phase of movement. In the latter case, the generalized muscle torque remains small during the outward phase. It is therefore primarily the extensor interaction torque that initially drives the elbow joint into extension during the 125° movement. Failure to regulate the effects of this torque in the outward phase may account for the hypermetria of elbow excursions that we observed in movements toward more counterclockwise targets.

The directional differences in trajectory control between controls and patients noted for the 0° and 125° directions were characteristic of other movement directions as well: in controls, systematic directional variations in interaction torque were associated with compensatory variations in generalized muscle torque, whereas this was not apparent in either of the two patients. Figure 9 plots the peak values of elbow joint accelerations against the peaks of the interaction

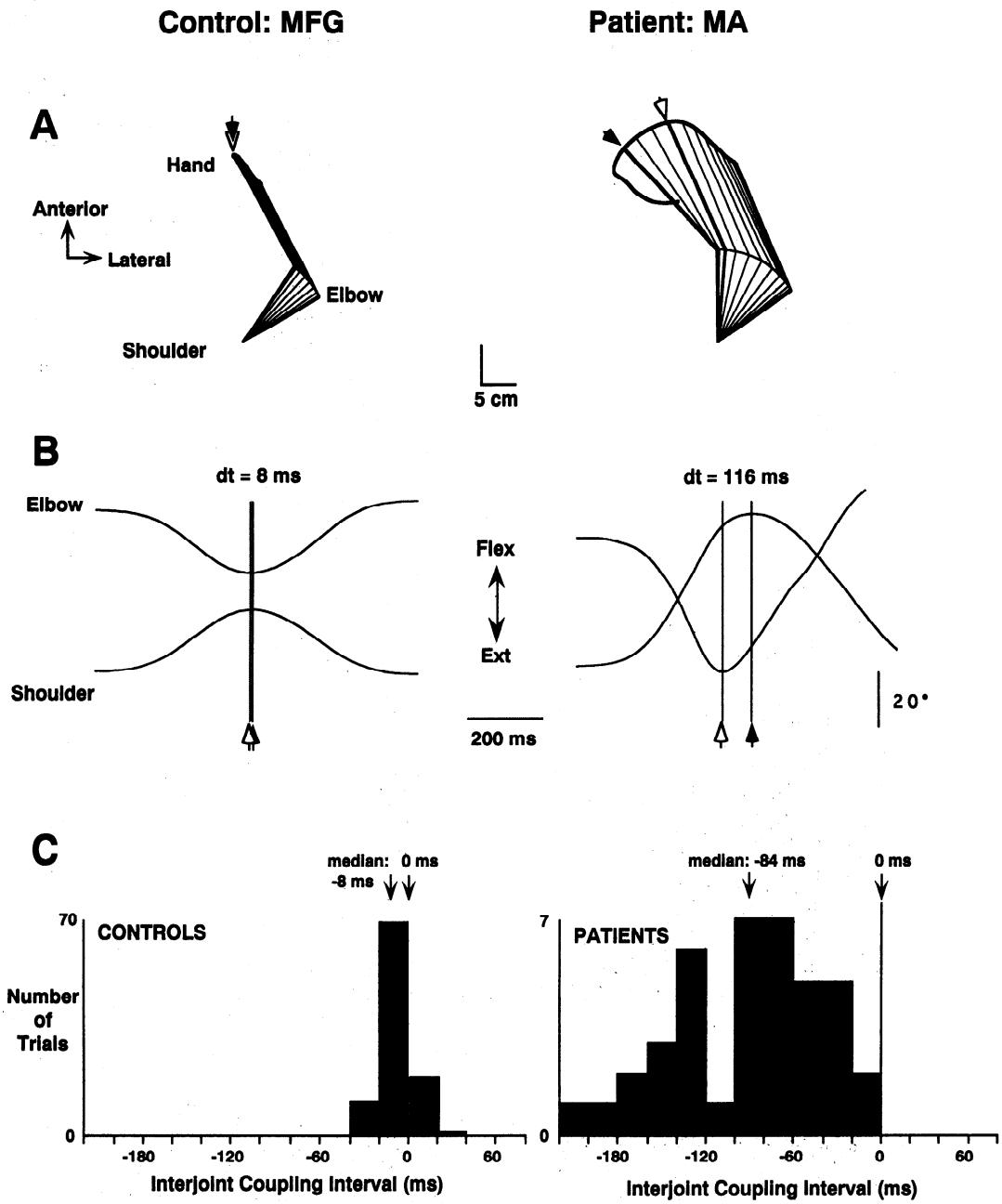

FIG. 6. Reversal errors result from decoupling between individual shoulder and elbow reversals. Representative movements performed by *control MFG* and *patient MA* are shown in *A* and *B*. *A*: stick figures of the upper arm and forearm drawn every 40 ms until the peak in shoulder angle was achieved. *B*: shoulder and elbow angles corresponding to the movements shown in *A*. Open arrowhead: peak (extension) elbow angle. Filled arrowhead: peak shoulder angle. Corresponding positions of the limb are marked by open and closed arrows and by bolded stick figures on the trajectory plots in *A*. The time between these values was our measure of interjoint coupling. *C*: histograms show the range of interjoint coupling intervals in 20-ms bins for all control subjects (*left*) and both patients (*right*).

torques, within the reversal phase of movement, for all directions in two representative controls and the two patients. As illustrated by these examples and by the statistics in Table 3, there was no significant correlation between the amplitude of interaction torque and flexor acceleration for the movements made by any control subject.[3] It can be seen that control subjects were able to modify the effects of elbow joint interaction torques during movement reversals appropriately to maintain elbow joint accelerations relatively constant regardless of movement direction. In the patients, however, 86%–91% of the variance in elbow angular acceleration could be accounted for by peak interaction torque. Because the patients were unable to adapt their muscle commands to take account of the direction-dependent variations in interaction torque, elbow joint kinematics were effectively enslaved to these passive biomechanical interactions.

[3] Only the trials along templates greater than 0° were included. This is because the interaction torque was often in the extensor direction (negative) during the reversal phase of the 0° movements which would have yielded a bimodal distribution.

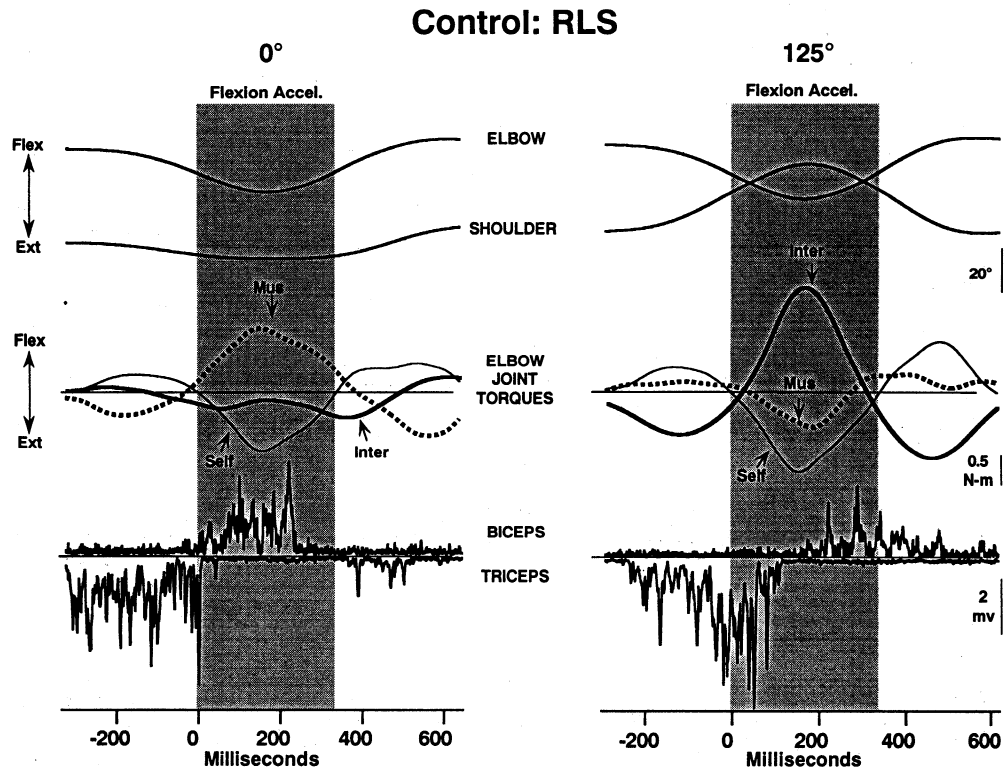

FIG. 7. Elbow kinematics, dynamics, and EMG patterns for movements in 2 directions in *control RLS*. Ensemble averages of joint angles (*top*), elbow joint torques (*middle*), and EMG (*bottom*) are shown for movements made along 0° (*left*) and 125° (*right*) templates. Data have been aligned to the 0 cross in elbow joint flexor acceleration, from extensor to flexor acceleration (0 on abscissa), and averaged across all 5 trials made toward each direction. The interval of elbow flexor acceleration, encompassing the reversal phase of the hand path, is shaded gray.

Deafferentiation disrupts normal directional variations in muscle activation patterns

Figure 10 shows raster plots of elbow angular acceleration and EMG records of biceps and triceps brachii for 30 movement trials from a session performed by *control subject CG* and by *patient MA*. Both are aligned on the onset of elbow flexor acceleration (vertical line) for a 600-ms interval that includes the reversal phases of all movements. Amplitude is represented by color scale illustrated by the code bar shown to the right of each raster. Red indicates flexor acceleration (*top*) and flexor EMG (biceps, *middle*), whereas blue depicts extensor acceleration (*top*) and extensor EMG (triceps, *bottom*). The data are sorted along the ordinate axis by target direction. Each group of five trials is labeled according to the orientation of the template line.

Because subjects had little practice in this task before data collection, variability is apparent in the elbow accelerations between individual trials toward any one direction. However, controls did not show systematic variations in elbow acceleration across movement directions. On the other hand, the timing and amplitude of EMG activation did vary with direction. In movements toward 0°, biceps activity began and triceps activity ended just before the initiation of elbow flexor acceleration (time 0). The initiation of biceps and termination of triceps activity occurred progressively later with the movements in successively more counterclockwise directions. Thus at 145° flexor acceleration occurred some 200 ms before biceps activation and was accompanied by triceps activity for some 150 ms. As discussed earlier, it was the interaction torque produced by the deceleration of shoulder flexion, rather than flexor muscle activity, that drove the elbow joint into flexion.

In the patients, this orderly relationship of muscle timing to movement direction did not occur and systematic directional increases in elbow acceleration were present. As in the example on the *right* of Fig. 10, the increase in elbow acceleration amplitude was most pronounced in the reversal phase, and resulted from the uncompensated effect of interaction torques.

Figure 11 shows the median and interquartile range of biceps onset time (hatched bars) and triceps termination time (open bars) for movements in different directions made by all control subjects and both patients. All data are aligned to the initial cross zero, from extension to flexion, in elbow angular acceleration. Coactivation is represented as the time between the median biceps onset time and triceps termination time (black bars). Biceps onset and triceps termination varied systematically with movement direction for movements made by all control subjects. Very little coactivation of flexors and extensors occurred. Instead, triceps activity tended to terminate before the onset of biceps activity for all movement directions, and thus regardless of the magnitude of the interaction torque. However, the patients coactivated flexors and extensors throughout much of the reversal phase of movement. For all movement directions, both patients initiated biceps activity before or within some 50 ms of the onset of flexor acceleration, whereas triceps activity

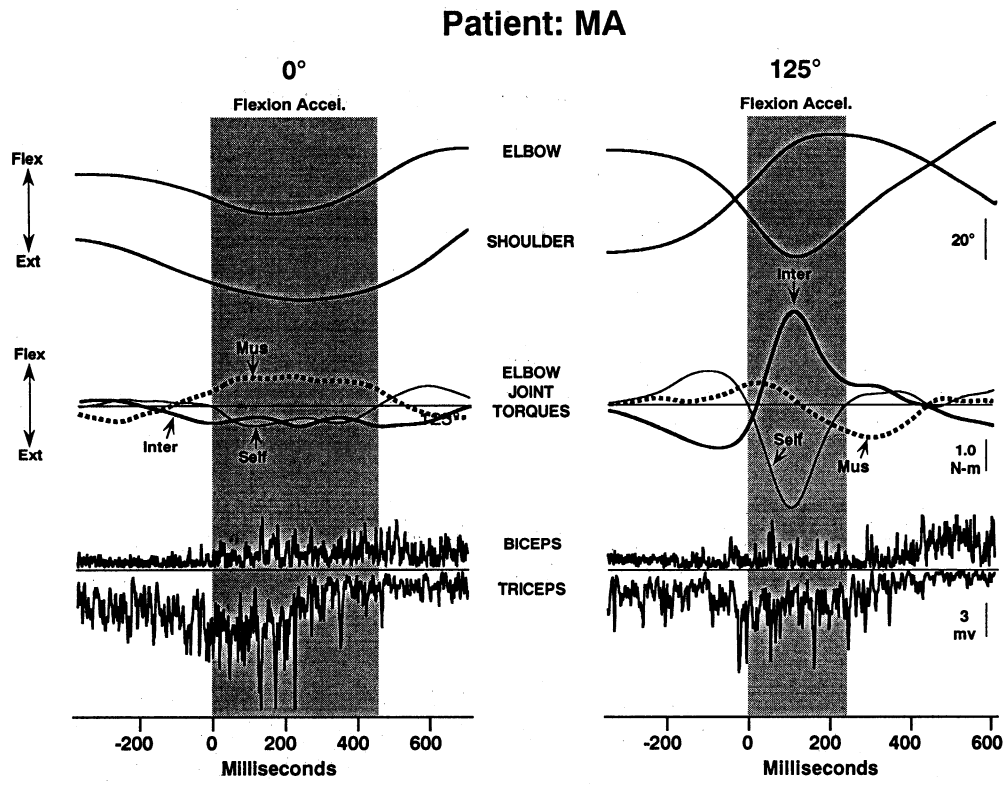

FIG. 8. Elbow kinematics, dynamics, and EMG patterns for movements in 2 directions in *patient MA*. Ensemble averages of joint angles (*top*), elbow joint torques (*middle*), and EMG (*bottom*) are shown for movements made toward the 0° (*left*) and the 125° (*right*) templates. Refer to Fig. 7 legend for details.

continued throughout the flexor acceleration phase. This coactivation was possibly an attempt to regulate elbow joint motion by increasing joint stiffness. However, this strategy was not successful in preventing interaction torques from significantly degrading trajectory control.

As demonstrated for the movements of control subjects, the interaction torque at the elbow joint systematically increased, whereas elbow joint kinematics remained relatively constant across movement directions. We therefore asked whether the directional variations in EMG timing seen for control subjects were related to variations in elbow interaction torques. As shown in Table 4, from 38% to 69% of the variance in biceps onset time (measured relative to the initiation of elbow flexor acceleration) was accounted for by peak elbow joint interaction torque. Similarly, as shown in Table 5, from 50% to 65% of the variance in triceps termination time was accounted for by peak elbow interaction torque for four of the five control subjects. However, in the case of *LN*, the timing of triceps varied neither with movement direction nor interaction torque amplitude. This indicates that, for this subject, modulation of biceps was adequate to control elbow kinematics. Also as shown in Table 4, neither the onset of biceps nor the termination of triceps activity was related to peak elbow joint acceleration for movements made by controls. Thus the timing of agonist (biceps) onset was scaled to the amplitude of the interaction torques but was not related to the amplitude of joint acceleration. In addition, when control subjects used the antagonist (triceps) to dampen elbow joint interaction torques, the antagonist was also scaled to the ampli-

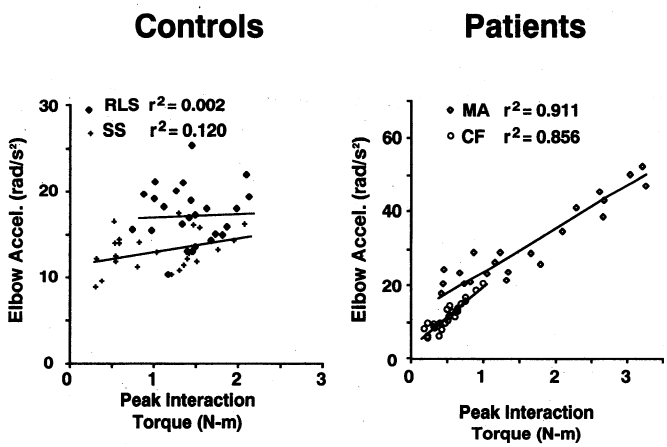

FIG. 9. Relationship of elbow joint acceleration to elbow interaction torque in controls and patients. Peak elbow angular acceleration is plotted against peak interaction torque, within the reversal phase, for movements performed by 2 controls (*RLS* and *SS*), and by both patients (*MA* and *CF*). Regression lines are drawn for each subject separately.

TABLE 3. *In deafferentation, elbow acceleration varies with interaction torque amplitude—correlation Statistics*

Subject	R^2	*P*-value
RLS	0.002	0.8352
CG	0.12	0.0891
SS	0.062	0.2296
LN	0.053	0.2665
MFG	0.011	0.6245
MA	0.911	≤0.001
CF	0.856	≤0.001

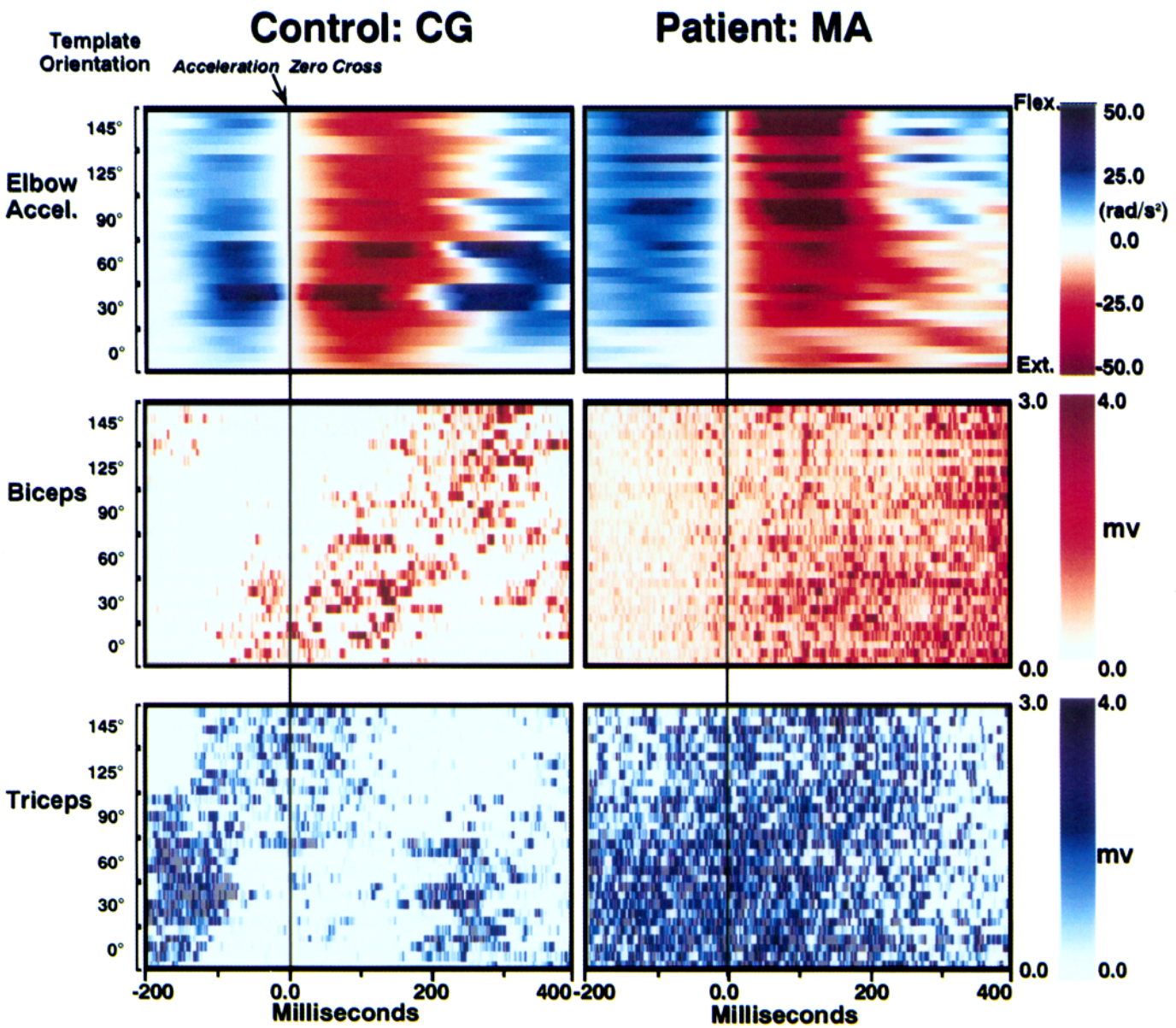

FIG. 10. EMG activation patterns across directions in controls and patients. Raster plots for all 30 trials performed within a session by *control CG* and *patient MA* are shown. The amplitude in elbow joint acceleration, biceps brachii EMG, and triceps brachii EMG is shown by the bar scales to the *right*. The amplitudes in flexor acceleration (*top*) and flexor EMG (*middle*) are represented by red shading, whereas the corresponding extensor values are scaled in blue. For EMG data, the scales for *control CG* (shown on the *left* of the calibration bar) and *patient MA* (shown to *right* of color calibration bar) are different. All data have been synchronized to the initiation of elbow flexor acceleration (vertical line) and are shown for a 600-ms interval, 200 ms before and 400 ms after this point, encompassing the full reversal phase of movement. On the ordinate, data have been grouped into template directions and sorted by movement direction.

tude of the interaction torques, but not to joint acceleration. Thus when interaction torques became large during movements they were utilized to drive elbow motion. At the same time, elbow joint muscles functioned to control the effects of the mechanical interactions between limb segments, rather than to directly accelerate the limb.

In contrast to controls, EMG activity was not related to interaction torque amplitude for the movements made by deafferented patients (see Tables 4 and 5). The patients were unable to adapt the timing of their muscle actions to the amplitude of elbow joint interaction torques. Consequently, as shown in Fig. 9, the amplitude of elbow joint acceleration during movement reversals was completely dependent on the amplitude of this interaction torque. In those directions in which this torque was large, elbow joint accelerations became correspondingly large, and the elbow joint was driven into flexion prematurely. Instead of reversing direction simultaneously, elbow and shoulder joint motions thus became desynchronized, producing the large medial curvatures in the hand paths that we referred to as reversal errors.

DISCUSSION

In our previous study of unconstrained three-dimensional movements we found that patients lacking proprioceptive

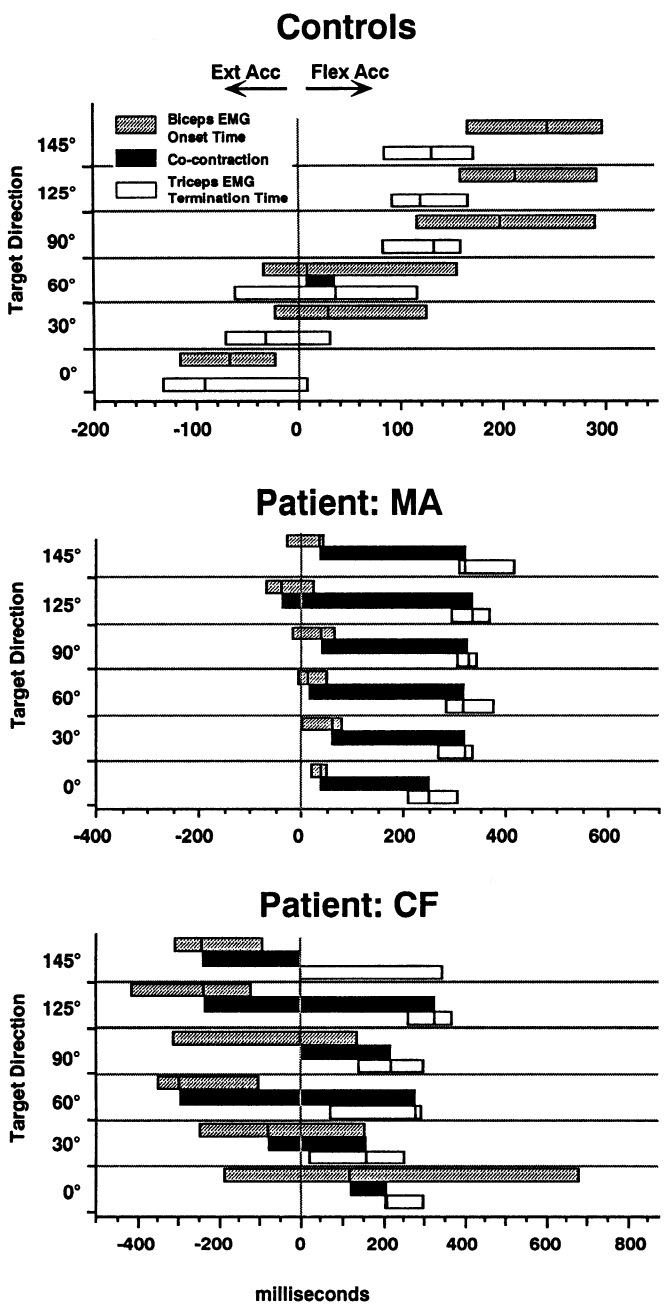

FIG. 11. Instead of reciprocal patterns of muscle activity that vary across directions, patients show excessive cocontraction across all directions. The median (vertical bar) and first 25th percentile for biceps onset time (crosshatch) and triceps termination time (white) are shown for all movements, grouped into template directions (ordinate) for all controls (*top*) and patients (*MA* and *CF*). Cocontraction (black) is represented as the positive time interval between the median biceps onset time and triceps termination time.

sensation show large trajectory anomalies at movement reversals (Sainburg et al. 1993b). We hypothesized that these reversal errors resulted from the occurrence of interaction torques produced by shoulder motion. We now test this hypothesis by examining the production of controlled planar hand paths requiring similar elbow kinematic trajectories but different shoulder motions. This allowed us to determine the effect of systematic variations in interaction torques on elbow kinematics. In addition, by using a planar task we eliminated a significant factor that complicates comparisons of three dimensional movements in controls and patients, namely, the varying effects of gravity that result from group differences in planarity of motion (Sainburg et al. 1993b). Normal subjects, as instructed, were able to produce overlapping hand paths for all directions of movement, synchronizing the reversals of elbow and shoulder. Analysis of joint torques and EMG recordings showed that subjects with intact sensation varied their patterns of muscle contraction with the direction of movement to match the varying patterns of interaction torques. Patients did not do so and this failure produced the observed interjoint coordination deficits and hand path errors.

TABLE 4.

	Biceps Onset Vs. Peak Interaction Torque (Elbow)		Biceps Onset Vs. Peak Elbow Joint Acceleration	
Subject	R^2	*P* Value	R^2	*P* Value
RLS	0.67	≤0.001	0.026	0.391
CG	0.43	≤0.001	0.047	0.25
SS	0.38	≤0.001	0.132	0.05
LN	0.40	≤0.001	0.0	0.97
MFG	0.69	≤0.001	0.15	0.03
MA	0.01	0.580	0.029	0.375
CF	0.001	0.890	0.12	0.069

In the following paragraphs we first consider the method we used to partition joint torques. We then discuss the implications of our findings for normal limb control and the role of proprioception in this control.

Partitioning of joint torques

As can be appreciated from an examination of *Eq. 2*, the formula for computing joint torques in the multijointed limb is a complex expression with many terms. This expression can more readily be understood, however, if its terms are combined into a smaller number of quantities whose meaning can be recognized more readily. Hollerbach and Flash (1982), recognizing the importance of intersegmental interactions, subdivided the terms of the torque equation into velocity- and acceleration-dependent elements. Another approach was taken by Smith, Zernicke and colleagues (Hoy and Zernicke 1985, 1986; Koshland and Smith 1989a; Schneider and Zernicke 1989; Schneider et al. 1989; Smith and Zernicke 1987) who sought to identify the relative con-

TABLE 5.

	Triceps Termination Vs. Peak Interaction Torque (Elbow)		Triceps Termination Vs. Peak Elbow Joint Acceleration	
Subject	R^2	*P* Value	R^2	*P* Value
RLS	0.64	≤0.001	0.057	0.205
CG	0.50	≤0.001	0.001	0.872
SS	0.55	≤0.001	0.210	0.011
LN	0.05	0.262	0.108	0.077
MFG	0.65	≤0.001	0.066	0.169
MA	0.15	0.036	0.072	0.160
CF	0.02	0.522	0.0	0.967

tribution of intersegmental interactions and of muscles to the generation of torque. They reduced the terms of the torque equation into four categories: interaction, generalized muscle, gravitational, and net torque. Although the partitioning of torques used here is closely related to the one devised by Smith and coworkers, it differs in two important respects: *1*) our computations are carried out in joint rather than absolute coordinates, so that torques are considered in reference to joint rather than to segment angles, and *2*) we have introduced a quantity termed "self torque" that represents the inertial resistance acting on the joint.[4] In addition, because our movements in the current study were performed in the horizontal plane, the force of gravity did not contribute to the torque at the joints and thus was not taken into account.

We partitioned our torque expressions in relation to anatomic joint angles. This intrinsic reference frame seemed likely to be better related to that of muscles and proprioceptive feedback signals than an extrinsic reference frame. Using joint coordinates also allowed us to isolate an expression that varies only with the acceleration of the joint under consideration. We used the term self torque for this reason, defining it as the joint angular acceleration multiplied by the effective limb inertia distal to the joint. Thus, if there is no acceleration at the joint, the self torque is equal to 0. If the other joints in the limb were immobilized during a movement, the self torque would be equal and opposite to the muscle torque. In a multijoint movement, however, it is directly inverse to the summed muscle and interaction torques. This term therefore provides an index by which to examine the relative actions of the generalized muscle and interaction torques.

Normal control of the hand trajectory

Although the computed generalized muscle torque provides information about the net effects of neural output, it does not distinguish between active muscle contraction and passive effects of soft tissue deformation. Moreover, it does not indicate whether motion at the joint results from contraction of a particular agonist muscle group or from variations in joint stiffness produced by cocontraction. However, some insights into the latter question can be obtained from myoelectric recordings which, by themselves, do not indicate the origin of the torque moving the joint. As pointed out by Hasan (Hasan 1991; Hasan and Stuart 1988), inverse dynamics computations and EMG recordings provide complementary information about the biomechanical function of muscle contraction and together can provide valuable information about the relationship between neural output and limb kinematics. The observations reported here indicate that intact subjects were able to produce accurate hand trajectories by precisely timing muscle actions so as to specifically counter or utilize joint interaction torques. This was most clear at movement reversals, when interaction torques became large and when accuracy demanded precise control over the timing of movement reversals at each joint.

As noted insightfully by Bernstein, more than 40 years ago (see Bernstein 1967), in multijoint movements muscle activation is not only needed to accelerate the limb but is critical for controlling the effects of reactive phenomena such as interaction torques. Bernstein hypothesized that the high levels of coordination needed to perform movements with skill would, in addition, exploit intersegmental interactions. To achieve this, the nervous system would have to produce precisely timed patterns of muscle activity. Our findings that EMG bursts in normal subjects are largely reciprocal and temporally adapted to directional variations in interaction torque indicate that subjects naturally utilize or dampen these torques as needed to satisfy the kinematic demands of the task. This provides strong support for Bernstein's hypothesis.

Such precise adaptation of neural output to mechanical interactions implies that the CNS must have accurate information about the motion of individual joints. This information could be used either "on line" to counter the perturbing effects of joint interaction torques or in a predictive capacity so that neural output is adapted to impending mechanical events. Because of the delays of neural transmission and muscle contraction, it is difficult to see how on-line feedback could prevent interaction torques from producing significant errors (Hasan 1991; Hasan and Stuart 1988; Hollerbach and Flash 1982). Because these torques are large when limb position is changing rapidly, corrective responses would occur when they were no longer appropriate. In addition, whether interaction torques assist or oppose movement depends on the intended trajectory. Indeed, kinematic plans are highly adaptable and the relationships between interaction torques and limb kinematics must be matched to the myriad demands of varied behavioral tasks. Thus if reflex actions contribute to the coordination of neural output with peripheral mechanics, delays, thresholds and gains must be strictly controlled by descending input. By gating segmental connections of proprioceptive and other afferent systems, descending control signals can influence both the reflex actions and the effective impedance at the joint (Lacquaniti et al. 1991).

These considerations make it likely that feedforward mechanisms, possibly based on an internal model of limb dynamics, should be of prime importance in controlling intersegmental dynamics. However, the dependence of this control on proprioceptive input suggests that the accuracy or fidelity of this model may rely on proprioceptive signals. In support of this hypothesis, Aoki recently showed that anticipatory postural adjustments in upper arm musculature were adapted to the interaction torques produced at the elbow by impending wrist movements (Aoki 1991). The anticipatory response of upper arm muscles varied according to the mechanical consequences of the intended motion, not with the prime mover at the wrist. On the other hand, Karst and Hasan (Karst and Hasan 1990; Karst and Hasan 1991a,b) recently showed that during planar reaching movements, the sign of muscle activity at movement onset could not be predicted by the torques required to make straight lines toward the target. Instead, the authors proposed rules based on

[4] Smith, Zernicke, and colleagues (Hoy and Zernicke 1985, 1986; Koshland and Smith 1989a,b; Schneider and Zernicke 1990; Schneider et al. 1989; Smith and Zernicke 1987) define a "net" torque as the inertia of the segment multiplied by the segment angular acceleration. This may at first appear similar to our self torque; however, the "net" torque varies with the angular accelerations of *all the joints proximal to the segment* and thus includes interjoint effects. In addition, the net torque is dependent only on the inertia of the limb segment, as isolated from the more distal segments, and thus does not represent the effective inertia carried by the particular joint.

geometric parameters that could better predict initial muscle activities. These findings suggest that intersegmental dynamics were not predicted before movement onset. However, in that study, the subjects were not instructed to make straight movements. Therefore, neither the initial path direction nor the initial kinetic requirement was explicitly dictated by the task. In contrast, the present study focused on how limb dynamics are controlled when the kinematic and dynamic requirements of the task are systematically varied. Our findings indicate that anticipatory control of interaction torques occurs during the course of movement. Due to their high dynamic sensitivity (Mathews, 1981), it is plausible that the information provided by muscle spindles could be used by the nervous system to derive inertial characteristics of the limb. This information could then be used to control intersegmental dynamics.

Role of proprioception in hand path control

The large differences we find in hand path control between subjects with intact sensation and patients with large-fiber sensory neuropathy indicate that proprioception plays a critical role in the control of interjoint dynamics. Deafferented patients failed to coordinate their muscle actions at the elbow with the occurrence of joint interaction torques. Instead, they cocontracted flexors and extensors, possibly in an attempt to regulate elbow joint movement through modulating joint stiffness. However, this was not a successful strategy. It should be noted that the effectiveness of cocontraction in increasing joint stiffness may be reduced in deafferented patients. This is because loss of stretch reflexes may alter the functional stiffness of muscles. Levin and coworkers (1994) showed that cocontraction of antagonists does not appear to produce the same degree of joint stiffness in deafferented patients as in intact subjects. Nevertheless, the coactivation strategy used by patients appears to reflect use of a significantly different strategy and form of descending control than used normally. Intact subjects timed the activities of their muscles precisely according to the magnitude of the interaction torques, rather than coactivating antagonist muscles in an attempt to dampen these interactions. As noted earlier, this may reflect descending control over reflex gating mechanisms that are not available to the patients.

These results confirm and extend the observations of Smith and Zernicke (1987) and their collaborators (Koshland and Smith 1989a,b; Sabin and Smith 1984) in establishing a role for proprioceptors in controlling limb interaction torques. These authors have shown that during the paw shake response in intact and in spinalized cats, muscle activity at the ankle functions largely to accelerate the paw, whereas muscle activity at the knee functions largely to counterbalance interaction forces produced by ankle and hip movement (Koshland and Smith 1989a,b; Sabin and Smith 1984). The activity in muscles that counteract large interaction torques during the paw shake was significantly altered by hind limb deafferentation or by immobilization of the joints that give rise to those torques (Koshland and Smith 1989a,b; Sabin and Smith 1984; Smith and Zernicke 1987). In contrast, activity in muscles that function primarily to accelerate joint motions were minimally affected by deafferentation or immobilization. Thus, during the paw shake response, the timing of muscle activities at a given limb segment was shown to depend on information generated by the movement of the other segments in the limb. These findings establish that spinal actions of muscle proprioceptors are responsible for coordinating muscle actions with interaction torques during the paw shake. It is easy to imagine how such stereotypic connections could support the relatively predictable dynamic interactions between limb segments that occur during this fixed response. However, it is unlikely that such connections could account for the fine coordination observed here during voluntary movements. Because the role of interaction torques in either assisting or countering joint motion varies with the diverse demands of different tasks, such coordination is more likely to be supported by feedforward commands that act to gate the activity of spinal interneurons and motor neurons.

We conclude that proprioception is normally important for controlling intersegmental dynamics. Our results suggest that this control occurs through feedforward mechanisms through which descending commands may modulate the activity of segmental circuits. During the course of movement, feedforward commands based on a proprioceptively updated internal model of the limb could allow the precise coordination between muscle actions and interaction torques observed in control subjects. In fact, we have shown elsewhere (Ghez et al. 1990; Gordon et al. 1995) that deafferented patients were able to use visual information to improve the feedforward control of movement and improve accuracy of reaching: direction and extent errors in planar movements were reduced for a brief period of time after patients viewed their limb during movement. Thus visual information acquired before movements allowed patients to program subsequent movements, made toward different directions, more accurately. The nature of the feedforward mechanisms normally used to control interjoint dynamics remains to be resolved and is the subject of ongoing research in our laboratory. In pilot experiments we have obtained comparable results examining reversal movements after patients had practiced moving in a different direction while viewing their limb (Ghez and Sainburg 1994; Sainburg et al. 1993a). We are currently examining how normal subjects and patients learn novel patterns of interjoint dynamics, and the extent to which the dynamic models used for such control can be generalized across task parameters.

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