Step-Tracking Movements of the Wrist. IV. Muscle Activity Associated With Movements in Different Directions

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Hoffman, Donna S. and Peter L. Strick. Step-tracking movements of the wrist. IV. Muscle activity associated with movements in different directions. *J. Neurophysiol.* 81: 319–333, 1999. We examined the patterns of muscle activity associated with multiple directions of step-tracking movements of the wrist in humans and monkeys. Human subjects made wrist movements to 12 different targets that required varying amounts of flexion-extension and radial-ulnar deviation. Wrist muscles displayed two patterns of electromyographic (EMG) modulation as movement direction changed: amplitude graded and temporally shifted. The amplitude-graded pattern was characterized by modulation of the quantity of muscle activity that occurred during two distinct time periods, an agonist burst interval that began before movement onset and an antagonist burst interval that began just after movement onset. The timing of muscle activity over the two intervals showed little variation with changes in movement direction. For some directions of movement, EMG activity was present over both time intervals, resulting in ''double bursts.'' Modulation of activity during the agonist burst interval was particularly systematic and was well fit by a cosine function. In contrast, the temporally shifted pattern was characterized by a gradual change in the timing of a single burst of muscle activity. The burst occurred at a time intermediate between the agonist and antagonist burst intervals. The temporally shifted pattern was seen less frequently than the amplitude-graded pattern and was present only in selected wrist muscles for specific directions of movement. Monkeys made wrist movements to 8–16 different targets that required varying amounts of flexion-extension and radial-ulnar deviation. These movements were performed more slowly than those of human subjects. The wrist muscles of the monkeys we examined displayed the amplitude-graded pattern of activity but not the temporally shifted pattern. Stimulation of individual wrist muscles in monkeys resulted in wrist movements that were markedly curved, particularly for the wrist extensors. These results indicate that step-tracking movements of the wrist are generated mainly by using the amplitude-graded pattern to modulate muscle activity. We propose that this pattern reflects a central process that decomposes an intended movement into an agonist, ''propulsive'' component and an antagonist, ''braking'' component. Separate bursts of muscle activity then are generated to control each component. On the other hand, we argue that the temporally shifted pattern may function to reduce the amount of movement curvature associated with the activation of wrist muscles.

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INTRODUCTION

A central question in systems neuroscience is how the nervous system generates the complex spatiotemporal commands needed to vary the speed, amplitude and direction of limb movement. We have chosen to investigate this issue by studying the generation of step-tracking movements of the wrist. Step-tracking movements are of interest because the muscle activity associated with them occurs in distinct bursts. Furthermore there is evidence that initial bursts in agonist and antagonist muscles are centrally generated (see Hoffman and Strick 1990 for discussion of this issue). Thus an examination of the timing and modulation of these bursts provides a ''window'' into the motor commands generated by the CNS. The wrist joint has the added complexity of rotating in two axes; this makes it particularly interesting for investigations into the neural control of movement direction.

In prior studies, we examined the modulations of agonist and antagonist muscle activity associated with step-tracking movements of different speeds and amplitudes and performed against different loads (Hoffman and Strick 1990, 1993). Here we will report on the muscle activity associated with movements in different directions. In preliminary experiments, we observed that wrist movements in different directions were produced by agonist and antagonist bursts that were graded in amplitude but relatively fixed in time (Hoffman and Strick 1986a). We proposed that during the programming of movement direction, the nervous system could perform a vector analysis, decomposing ''intended'' movement vectors into agonist and antagonist components for each muscle (pg. 290, Hoffman and Strick 1986a). In other experiments, we provided evidence that the two components are generated separately (Hoffman and Strick 1990, 1993; Waters and Strick 1981). However, they are ultimately summed so that, for some directions of movement, single muscles show ''double bursts,'' i.e., muscle activity over both the agonist and antagonist burst intervals. Thus our prior results suggested that the nervous system determines the direction of wrist movement by modulating the amplitude of two bursts of muscle activity whose time course is relatively fixed.

Recently, Flanders (1991) and Flanders et al. (1994, 1996) proposed an alternative hypothesis. These authors observed that individual shoulder and elbow muscles displayed a single burst of activity, the timing of which shifted gradually with the direction of a pointing movement. The burst could occur later than a normal agonist burst but earlier

than a normal antagonist burst. These authors proposed that modification of the timing of a single burst of activity in individual muscles is the primary mechanism used by the nervous system to specify movement direction.

To determine the prevalence of these two patterns of modulation, amplitude graded and temporally shifted, we examined muscle activity associated with step-tracking movements of the wrist in additional human subjects and in monkeys. We found that, for most movement directions, the wrist muscles of humans and monkeys displayed the amplitude-graded pattern of modulation. In fact, the amplitude modulations of the agonist burst (and, in some cases, the antagonist burst) for different directions of movement were well fit by a cosine function. Temporally shifted bursts were not observed in monkeys but occurred for movements to selected targets in human subjects. Thus although both patterns of modulation were seen in wrist muscles, the present results underscore the prevalence of the amplitude-graded pattern.

Brief reports of portions of this work have appeared previously (Hoffman and Strick 1987, 1997).

METHODS

Our results are based on an examination of patterns of muscle activity in six normal human subjects (2 males, 4 females, aged 24–43 yr) and in three nonhuman primates (*monkey A: Macaca mulatta*, 5.4 kg; *monkey B: M. nemestrina*, 5.8 kg; *monkey C: M. mulatta*, 4.2 kg). The same three monkeys also were used for the results reported in Hoffman and Strick (1993). The experiments were conducted according to National Institutes of Health guidelines and were approved by the relevant institutional committees overseeing human and animal experiments. All of the human subjects gave their informed consent. We first will describe the procedures for the human experiments and then describe those for the monkey studies.

Experimental setup and task

Each human subject sat in a chair that supported the forearm and elbow of the dominant (right) limb. The forelimb was held in the neutral position, midway between full pronation and full supination. The subject grasped the handle of a two-axis manipulandum. This device was described and illustrated in a prior study (Fig. 1 of Hoffman and Strick 1986b). It is a lightweight, low-friction device with a moment of inertia of $\sim$0.0025 kg $\times$ m^2 in the radial-ulnar direction. The handle of the manipulandum rotates in the vertical and horizontal axes. Two potentiometers are coupled to the device to measure the angles of the wrist in the planes of radial-ulnar deviation and flexion-extension.

Each subject sat $\sim$70 cm in front of a large screen oscilloscope (26 $\times$ 36 cm) that displayed a cursor and a target. The cursor was a small spot of light ($\sim$5 $\times$ 5 mm) that moved in proportion to the subject's wrist movements. One degree of wrist movement moved the cursor 4.5 mm on the screen. The target was an open square, the inside diameter of which equaled 2.5° of wrist movement. The location of the target on the screen was determined by a DEC PDP 11/03 computer.

Subjects were asked to perform a step-tracking task similar to that described in our previous publications (Hoffman and Strick 1986a,b, 1990). To initiate a trial, the subject placed the cursor inside the target, which was positioned at the center of the screen. This target location caused the wrist to be in the neutral position for the start of each trial. After a variable hold period, the target jumped to 1 of 12 different locations on the screen, arranged like the numerals on a clock (see Fig. 1, *left*). The subject was required to perform a ''step-tracking'' movement of the wrist; this placed the cursor in the new target location. Subjects were instructed to move as rapidly and accurately as possible to the new location. Different targets were presented in clockwise order, starting with target 12 and ending with the same target. Data were collected for 25 successive movements to each target. In different experiments, target locations could require a 5, 15, 20, or 25° change in wrist angle. In this report, we will focus on the patterns of muscle activity for the intermediate amplitudes of movement (15 or 20°). Subjects were allowed a small amount of practice ($\sim$5 trials to several of the targets) before the initiation of data collection.

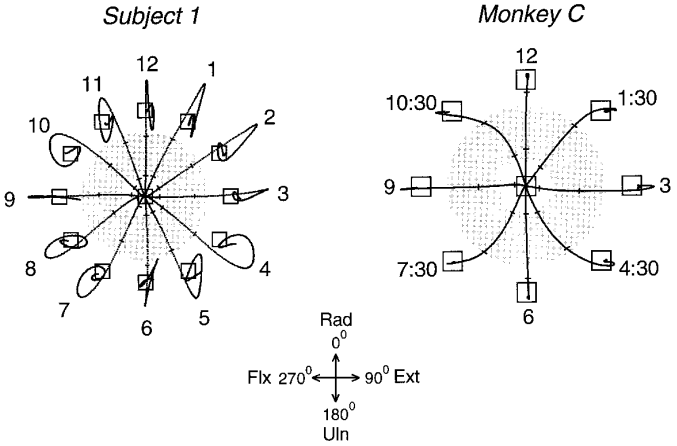

FIG. 1. Trajectories of wrist movements for humans and monkeys. *Left*: average trajectories of *subject 1*. Movements were performed as fast and as accurately as possible from the central target location to 12 different peripheral target locations, arranged like the numerals on a clock. Acquisition of the peripheral targets required a 15° change in the angle of the wrist joint. Each trace is the average of 20–25 movements to the same target location. Tick marks indicate 30 and 50 ms after movement onset. ■, 25–75% of the distance between the central and peripheral targets. *Right*: average trajectories of *monkey C*. Monkey was trained to make rapid movements (movement time <200 ms) from the central target location to 8 different peripheral targets. Acquisition of the targets required a 20° change in the angle of the wrist joint. Each trace is the average of 12–29 movements to the same target location. Tick marks indicate 50 and 100 ms after movement onset. ■, 25–75% of the distance between the central and peripheral targets. Ext, extension; Flx, flexion; Rad, radial deviation; Uln, ulnar deviation.

Data acquisition

We obtained electromyographic (EMG) recordings from four wrist muscles: extensor carpi radialis longus (ECRL); extensor carpi radialis brevis (ECRB); extensor carpi ulnaris (ECU); and flexor carpi radialis (FCR) (see Table 1). EMG activity was recorded with surface electrodes (Liberty Mutual Myoelectrodes, Boston, MA) that have contact surfaces spaced 1.3 cm apart. The electrodes were positioned until they recorded large responses with wrist movement and minimal activity with finger movement. Three subjects were examined in two or more separate recording sessions to verify that the pattern of activity for single muscles was repeatable. One subject was examined using both surface electrodes and intramuscular fine-wire electrodes in ECRL and ECRB to verify the reliability of surface recordings from these muscles. The technical details for fine-wire electrodes are described below (see, *Experiments in nonhuman primates*). An analysis of activity in flexor carpi ulnaris is not included in this study because we were not convinced that surface recordings from this muscle provided a reliable indication of its activity.

Amplifiers built into the surface electrodes amplified the raw

TABLE 1. *Experimental subjects*

Subjects	ECRL	ECRB	ECU	FCR
Human				
1	S, I	S, I	S	S
2	S			
3	S	S	S	S
4	S	S	S	S
5	S		S	
6	S		S	
Monkeys				
A	I	I	I	I
B	I	I	I	I
C	I	I	I	I

S, surface electrodes; I, intramuscular electrodes; ECRL, extensor carpi radialis longus; ECRB, extensor carpi radialis brevis; ECU, extensor carpi ulnaris; FCR, flexor carpi radialis.

EMG signals by 2,666 or 2,800. These signals were monitored on a storage oscilloscope and were full-wave rectified and filtered (τ = 10 ms) (see Gottlieb and Agarwal 1970). Rectified and filtered EMG signals, along with the two position signals from the two-axis manipulandum, were digitized at 1.25 kHz and stored on a DEC PDP 11/34 computer.

Data analysis

All trials were examined individually and occasional odd trials (i.e., trials that were slow or were inaccurate in direction or amplitude) were eliminated from the database. The potentiometer and EMG signals were aligned on movement onset (defined by the computer as a 1.2° change in either x or y) and averaged for each direction of movement. Further analysis was performed using averaged data.

We calculated a ''combined'' *x-y* displacement signal from averages of the two potentiometer signals. Movement onset was redefined as the earliest change in wrist displacement in the combined signal, and the corresponding EMG records were realigned on this point. Displacement was smoothed (using a 9- or 13-point moving average) and differentiated to obtain velocity and acceleration. We measured the peak values of displacement, velocity, and acceleration for each movement direction and the time of occurrence of the peak values. Movement duration was defined as the time between movement onset and peak displacement.

The average of EMG during the hold period (*baseline activity*) was subtracted from each average EMG trace. EMG data then were rescaled in amplitude, setting the baseline activity during the hold period to 0% and the maximum peak amplitude for any of the 12 directions of movement to 100%. We measured the time of peak EMG to approximate overall burst timing. When an EMG burst consisted of two closely spaced peaks of nearly equal amplitude, we averaged the time of occurrence of the two peaks. To make contour plots (Figs. 8 and 9), we summed EMG data into 8-ms bins. We then used the method of kriging with a linear variogram in the computer program Surfer (Golden Software, Golden, CO) to interpolate between data points and generate a smooth contour plot. This method of weighted averaging maintains an accurate representation of the data points.

To examine the spatial tuning of activity from single muscles, we determined the movement direction with the largest agonist burst (*best agonist direction*) and the direction with the largest antagonist burst (*best antagonist direction*). We defined an *agonist burst interval* as the time when the largest agonist burst was >25% of its peak amplitude and an *antagonist burst interval* as the time when the largest antagonist burst was >25% of its peak amplitude (Hoffman and Strick 1986a). This method of defining temporal intervals for agonist and antagonist activity avoided the difficulty of determining the onset and endpoint of each burst. Finally, we integrated EMG activity for each movement direction separately during each of the two burst intervals. Occasionally, agonist and antagonist intervals overlapped slightly in time. When this occurred, we shortened the integration interval for the agonist burst and delayed the integration interval for the antagonist burst by equal amounts so that the intervals for agonist and antagonist activity were nonoverlapping.

We performed a cosine regression on the integrated EMG activity during the agonist burst interval and a separate cosine regression on the integrated EMG activity during the antagonist burst interval for each muscle, following the methodology of Georgopoulos et al. (1982). The regression equation was: $y = a + b*\sin x + c*\cos x$, where x = movement direction (in radians); a, b, and c = regression coefficients. The methodology of Georgopoulos et al. was modified to account for the fact that EMG activity was absent for some directions of movement. When EMG was absent, we decreased the weighting of 1 or 2 near zero values to 0.01, whereas the remaining points had a weighting of 1.0 in the calculation of the regression equation. The decreased weighting of points resulted in a cosine that more accurately fit the larger values of EMG. This adjustment of weights reduced the resulting R^2 values slightly. The peak of the cosine defined a ''preferred direction'' for each muscle's agonist or antagonist activity. The peak was calculated by determining $\theta = \tan^{-1} b/c$. Then peak = θ if $b > 0$ and $c > 0$; peak = θ + 180° if $c < 0$; peak = θ + 360° if $b < 0$ and $c > 0$. The preferred directions obtained in separate recordings from the same muscle in a single subject were averaged to obtain a single value for each muscle in a subject.

Experiments in nonhuman primates

Each monkey sat in a primate chair with its forearm supported and grasped the handle of a scaled-down version of the two-axis manipulandum described above. Monkeys naturally displayed considerable tonic activity in ECRL and ECRB when holding the manipulandum handle in the neutral position even though no weight was added to the handle. The task that the monkeys performed was similar to that in the human study. They initiated a trial by placing the cursor in the target, which was centered on the screen. The inside diameter of the target measured ~3.5° of wrist movement. After a variable hold period, the target was stepped from the central position to one of 8 or 16 different locations equally spaced around the central position (see Fig. 1, *right*). The monkey was required to place the cursor in the new target location with a movement time <200 ms to receive a small juice reward. The required change in wrist angle was 20°. Monkeys received considerable training in this task (800–1,500 trials per day for 2–7 yr) so that their performance was quite stable.

EMG recordings were obtained from the monkeys in one or two sessions per week over a 3- to 4-mo period. The same four wrist muscles as in the human experiments were sampled in the monkey (Table 1). In addition, we sampled flexor carpi ulnaris extensively in two monkeys from the present study and in a third monkey that was part of another study. In all cases, little activity was recorded from this muscle during the step-tracking task.

Each muscle was examined 2–10 times per animal to confirm that the pattern of EMG and its preferred direction were repeatable. EMG activity was recorded with pairs of single-stranded stainless steel, Teflon-coated wires (Medwire, Mt. Vernon, NY; 0.003-in diam) that were inserted percutaneously into one to two muscles per recording session. The tip of each wire was uninsulated for 1.5 mm, and the two wires were placed 4–5 mm apart within a muscle. We confirmed that both wires of a pair were inserted into the same muscle by observing the movement evoked when each single wire was stimulated (10–20 pulses at 50/s, 100–500 μA). During

EMG recording, the raw signals were amplified by 2,000 times and then were full-wave rectified and filtered as in the human experiments described earlier. The EMG signals and the two potentiometer signals were digitized by computer as in the human experiments. Data were processed as described above for the human experiments.

Agonist bursts in the monkey generally had a longer duration than those of the human. Therefore, we integrated agonist activity during a constant 100-ms interval beginning at EMG onset. This integration interval included as much of the agonist burst as possible while excluding antagonist activity when it was present. EMG activity was rescaled in amplitude as described for EMG activity of humans (see *Data analysis*). The preferred directions obtained in separate recordings from the same muscle in a monkey were averaged to obtain a single value for each muscle in a subject.

At the conclusion of every recording session, we attempted to determine the ''pulling direction'' of each muscle. This was accomplished by requiring the monkey to hold the manipulandum in the central position while we stimulated a single muscle through the recording electrodes. The muscle was stimulated with 250 μA to 1 mA for 10–20 pulses at 50/s. The current was adjusted to produce a 4–5° movement of the wrist. We recorded 8–10 trials of the resulting movement.

RESULTS

Our findings will be presented in three sections. In the first, we will briefly describe the kinematics of step-tracking movements of the wrist in the human and the monkey. Because we previously have reported our studies of the kinematics of movements in a single plane (Hoffman and Strick 1986b), this section will emphasize the special features of movements in different directions. The next section will describe how the spatiotemporal patterns of EMG activity are modulated to produce different directions of wrist movement in humans. The final section will compare and contrast the EMG patterns observed in monkeys with those of humans.

Kinematics

When human subjects performed step-tracking movements in different directions, the initial 50–70 ms of movement occurred in a nearly straight line and was well directed toward the target (Fig. 1, *left*, ■). All movements overshot the targets considerably and then curved back toward the target. The step-tracking movements of human subjects were quite rapid. Radial and ulnar deviation movements to the 15° targets had mean durations of ~80 ms; flexion and extension to the 15° targets had mean durations of ~110 ms. Mean peak tangential velocities for these movements were 550°/s (radial + ulnar deviation) and 440°/s (flexion + extension). The corresponding peak tangential accelerations were 18,900 and 13,400°/s^2.

In contrast, the wrist trajectories of monkeys were usually straight in the radial-ulnar and flexion-extension directions, but movements in some diagonal directions were curved after the initial 50–70 ms of movement (Fig. 1, *right*, ■). Step-tracking movements of monkeys overshot the targets by far less than the movements of humans. The movements of monkeys were rapid, but movement durations were approximately twice those of humans and peak velocities and accelerations were less than half those of humans. Movements of 20° performed by monkeys had mean durations of ~200 ms. Mean peak tangential velocities for these movements were 218°/s (radial + ulnar deviation) and 155°/s (flexion + extension). The corresponding peak tangential accelerations were 4,300 and 2,400°/s^2.

Patterns of wrist muscle activity in humans

Each of the four prime movers of the wrist joint (ECRL, ECRB, ECU, FCR) displayed a well-defined *agonist* burst for movements close to the muscle's ''pulling direction'' (Fig. 2, *A*, target 12; *C*, target 12) (see also Hoffman and Strick 1990). Agonist bursts generally began 30–50 ms before movement onset, peaked before the peak of acceleration, and had a duration of 75–100 ms. However, in some subjects, the agonist bursts recorded from extensor muscles were prolonged (e.g., Fig. 3*D*) (see also Hoffman and Strick 1993).

Each of the prime movers of the wrist also displayed a well-defined *antagonist* burst for movements in a direction nearly opposite to the muscle's pulling direction (Fig. 2, *A*, target 6; *C*, target 6). Antagonist bursts began 10–45 ms after movement onset, peaked just after the peak of velocity, and had a duration of 70–100 ms. To aid in characterizing patterns of muscle activity, we defined an *agonist burst interval* and an *antagonist burst interval* for each EMG recording (see METHODS) (Hoffman and Strick 1986a). On average, the agonist burst interval began ~20 ms before movement onset (Fig. 3, *A* and *D*). The antagonist burst interval began ~35 ms after movement onset (Fig. 3, *B* and *E*). The end of the agonist burst interval usually coincided with the start of the antagonist burst interval.

Modulations with changes in movement direction

When subjects generated wrist movements in directions that differed from the ''best'' agonist or antagonist direction for a muscle, the activity was modulated in either of two spatiotemporal patterns, termed *amplitude graded* and *temporally shifted.* The key feature of the amplitude-graded pattern (Hoffman and Strick 1986a) was the presence of two bursts of muscle activity: one during the agonist burst interval and the other during the antagonist burst interval (Fig. 2*A*, target 9). The ''double bursts'' apparent in averages of EMG activity also were visible in single trials (Fig. 4*C*) and thus were not an artifact of averaging.

A second feature of the amplitude-graded pattern was that muscle activity during the agonist and antagonist burst intervals displayed marked modulation. The amplitude of muscle activity during the agonist burst interval was graded from a maximum for movements in the best agonist direction (Fig. 3, *A* and *D*) to a minimum for movements in the best antagonist direction (Fig. 3, *B* and *E*). Quantitative analysis showed a systematic reduction in the amplitude of the agonist burst as movement direction rotated away from the best agonist direction. Activity during the agonist burst interval followed a cosine function ($R^2 = 0.84–0.97$) for all muscles sampled in all subjects (Fig. 5, *A* and *B*). Each of the wrist muscles we examined displayed at least one instance of an exceptional fit ($R^2 = 0.96–0.97$) with a cosine function.

Individual wrist muscles had distinct preferred directions (Table 2). The preferred directions for ECRL, ECU, and

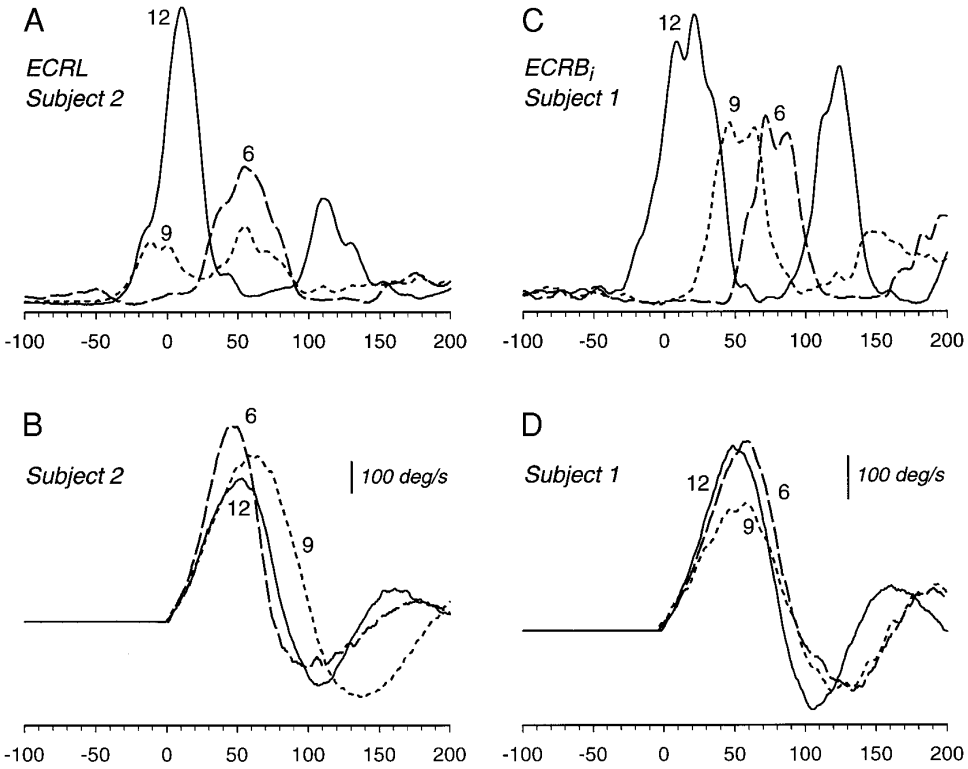

FIG. 2. Two patterns of electromyographic (EMG) modulation associated with wrist movements in different directions. *A*: amplitude-graded pattern. An agonist burst occurred for movements to target 12 (solid line). An antagonist burst occurred for movements in the opposite direction (target 6, long dashed line). Two bursts of activity, one at the time of an agonist burst and the other at the time of an antagonist burst, occurred for movements in an orthogonal direction (target 9, short dashed line). ECRL, extensor carpi radialis longus (surface recording). *B*: tangential velocity traces for the movements associated with the EMGs shown in *A*. *C*: temporally shifted pattern. An agonist burst occurred for movements to target 12 (solid line). An antagonist burst occurred for movements in the opposite direction (target 6, long dashed line). A single burst of activity that was shifted in time, compared with agonist and antagonist bursts, occurred for movements in an orthogonal direction (target 9, short dashed line). ECRB$_i$, extensor carpi radialis brevis (intramuscular recording). *D*: tangential velocity traces for the movements associated with the EMGs shown in *C*. All EMG traces were full-wave rectified, filtered (τ = 10 ms), and averaged (n = 20–25). Time scale is in ms; 0 = onset of movement.

FCR showed little variability among subjects and recording sessions and were distributed evenly (i.e., were separated by 120°). The preferred direction of ECRB was less consistent and varied by ≤60° between subjects. All muscles had prominent agonist activity (≥25% of the integrated EMG present for the best agonist direction) for movements to six or more targets (i.e., at least one-half of the targets). Thus even though individual muscles displayed a specific preferred direction, muscle activity was broadly tuned during the agonist burst interval (Fig. 6).

In general, there was considerable coactivation of wrist muscles (Fig. 6). Two or more wrist muscles were active during the agonist burst interval for every direction of wrist movement. For example, a wrist extensor (ECRL) and a wrist flexor (FCR) were coactivated when subjects moved to targets 9–11. All three wrist extensors (ECRL, ECRB, ECU) were coactivated when subjects moved to targets 1–4. Thus coactivation included muscles that normally are considered as synergists (e.g., ECRL and ECU) as well as those that normally are considered as antagonists (e.g., ECRL and FCR).

Activity over the antagonist burst interval also was graded from a maximum for movements in the best antagonist direction (Fig. 3, *B* and *E*) to a minimum for movements in the best agonist direction (Fig. 3, *A* and *D*). However, amplitude modulations during this interval were less systematic than those during the agonist burst interval. Only 9 of 17 EMG recordings displayed cosine tuning of activity during the antagonist burst interval with an $R^2 \geq 0.54$. Of these nine recordings, six displayed a preferred direction of antagonist activity that was not directly opposite to (i.e., differed by <170° from) the same muscle's preferred direction of agonist activity (Table 2). Restricting comparisons of preferred directions to the six recordings for which antagonist activity was well fit by a cosine function (i.e., $R^2 > 0.8$), four of these had preferred directions of antagonist and agonist activity that were not directly opposite to each other. These observations suggest that activity during the antagonist burst interval is tuned separately from activity during the agonist burst interval. The remaining 8 of 17 EMG recordings had a nonsignificant R^2 and showed little variation in the amplitude of EMG during the antagonist burst interval associated with changes in movement direction.

A final, but essential, feature of the amplitude-graded pattern was a relative constancy in the timing of agonist and antagonist bursts. The constancy was present despite large changes in burst amplitude, as described earlier. For example, the time of peak agonist activity in ECRL and ECU generally varied by only 5–10 ms over a 150–180° range of target directions (Fig. 7, *A*, targets 10–3; *D*, targets 2–6). We observed a slightly greater variation in the timing of the antagonist burst (~20 ms; Fig. 7*A*, targets 4–8). Overall, the peaks of each burst in a double burst (Figs. 2*A* and 8 and 9, Db) occurred at nearly the same times as the peaks of the agonist and antagonist bursts when these bursts were present in isolation (Figs. 8 and 9, Ag and Ant). Because the amplitude of each burst in a double burst varied with movement direction, the agonist component was larger than the antagonist for some directions of movement, whereas the antagonist component was larger for other movement directions. When the largest EMG component shifted from one burst interval to the other, the time of peak EMG showed a step change of ≥50 ms (Fig. 7, *A*, targets 3–4, 9–10; *B*, targets 4–5; *C*, targets 3–4; and *D*, targets 1–2). On the basis of all of our observations on the amplitude-graded pattern, we conclude that one strategy used by

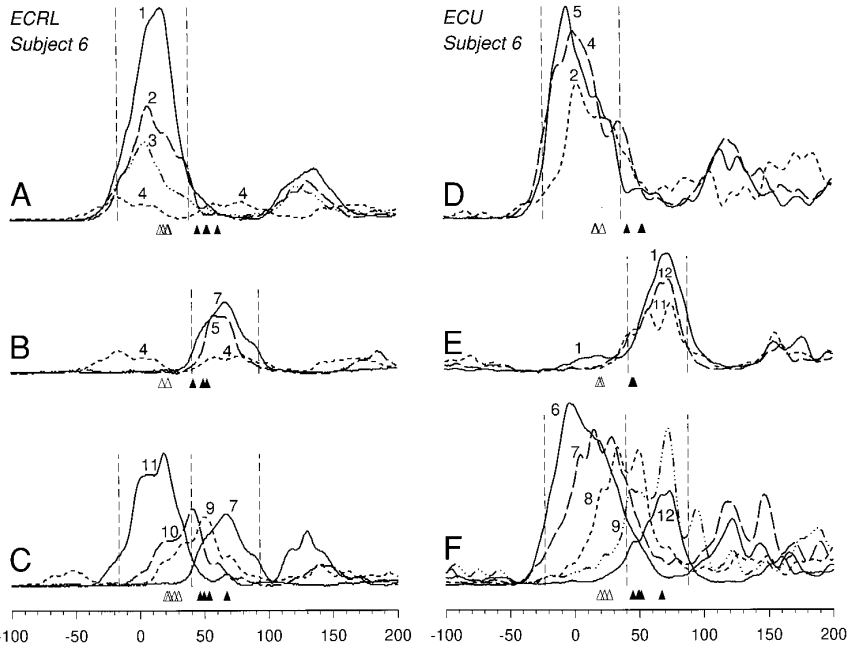

FIG. 3. Patterns of EMG modulation for 2 different muscles in a single subject. *A*: amplitude grading of the agonist burst in ECRL for movements to targets 1 (solid line), 2 (long dashed line), 3 (dot-dashed line), and 4 (short dashed line). Vertical dashed lines indicate the beginning and end of the agonist burst interval for this EMG recording. Note: 2 small bursts, 1 during the agonist burst interval and 1 during the antagonist burst interval, occurred for movements to target 4. *B*: amplitude grading of the antagonist burst in ECRL for movements to targets 7 (solid line), 5 (long dashed line), and 4 (short dashed line). Vertical dashed lines indicate the beginning and end of the antagonist burst interval. *C*: temporally shifted burst in ECRL for movements to targets 10 (long dashed line) and 9 (short dashed line). An agonist burst occurred for movements to target 11 (solid line), and an antagonist burst occurred for movements to target 7 (solid line). Vertical dashed lines indicate the agonist and antagonist burst intervals. *D*: amplitude grading of the agonist burst in ECU for movements to targets 5 (solid line), 4 (long dashed line), and 2 (short dashed line). *E*: amplitude grading of the antagonist burst in ECU for movements to targets 1 (solid line), 12 (long dashed line), and 11 (short dashed line). Two bursts, 1 during the agonist burst interval and 1 during the antagonist burst interval, occurred for movements to target 1. *F*: temporally shifted burst in ECU for movements to target 8 (short dashed line). An agonist burst occurred for movements to targets 6 (solid line) and 7 (long dashed line), and an antagonist burst occurred for movements to targets 12 (solid line) and 9 (dot-dashed line). ECU, extensor carpi ulnaris. All EMG traces were full-wave rectified, filtered (τ = 10 ms), and averaged (n = 20–25). Open triangles: times of peak acceleration. Closed triangles: times of peak velocity. Time scale is in ms; 0 = onset of movement.

the nervous system to determine movement direction is to adjust the *amplitude*, but not the timing, of agonist and antagonist bursts in multiple muscles at a single joint.

Temporally shifted

In some instances, when movements differed from the best agonist and best antagonist directions, we observed a second pattern of muscle activity, termed *''temporally shifted''* (Fig. 2*C*, target 9). This pattern was characterized by a single burst of activity, the peak of which lagged that of a normal agonist burst but led that of a normal antagonist burst. We defined a temporally shifted burst as a burst with a peak that occurred in the interval *after* peak acceleration and *before* peak velocity.[1] This interval is indicated by the cross-hatching in Fig. 7. Single bursts in this interval displayed a gradual and systematic shift in their timing from just after the agonist burst interval to just before the antagonist burst interval (Figs. 3, *C* and *F*; and 7, *B*, targets 8–11; *C*, targets 9–12; and *D*, targets 6–9). Temporally shifted bursts have been described previously in studies of shoulder and elbow muscle activity during pointing movements (Flanders et al. 1994, 1996).

Temporally shifted bursts displayed more modest changes in burst amplitude than amplitude-graded bursts and ranged in size between the largest agonist and the largest antagonist bursts (Fig. 3, *C* and *F*). The duration of a temporally shifted burst was approximately the same (~70–80 ms) as the duration of a single agonist or antagonist burst (Figs. 2*C* and 8 and 9, Sh). This feature should be contrasted to the clear increase in burst duration (to ~130 ms, Fig. 2*A*) associated with the occurrence of double bursts in the amplitude-graded pattern (Figs. 8 and 9, Db).

When temporally shifted bursts were present in wrist extensor muscles, they occurred only for movements that required some wrist flexion and only for movements that were nearly perpendicular to the preferred direction of an individual muscle. Specifically, we observed temporally shifted bursts in *ECU* for movements to targets 7 and/or 8 (Figs. 7*D* and 9*B*), in *ECRL* for movements to targets 9 and/or 10 (Figs. 7*B* and 8*B*), and in *ECRB* for movements to targets 9, 10 and/or 11 (Figs. 7*C* and 9*C*). No consistent pattern was apparent in the occurrence of temporally shifted bursts in FCR.

[1] In general, peak acceleration occurred ~25 ms after movement onset and peak velocity occurred ~50 ms after movement onset.

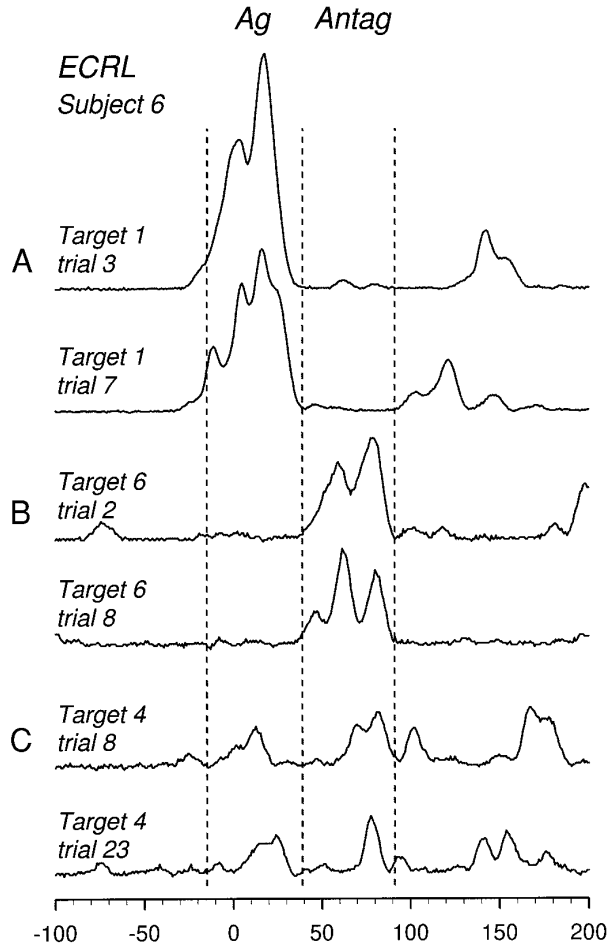

FIG. 4. EMG patterns on single trials. *A*: agonist burst for 2 movements to target 1 (best agonist direction). *B*: antagonist burst for 2 movements to target 6 (best antagonist direction). *C*: 2 small bursts, 1 during the agonist burst interval and 1 during the antagonist burst interval, for 2 movements to target 4. Vertical dashed lines indicate the agonist and antagonist burst intervals. EMGs were full-wave rectified and filtered (τ = 10 ms). ECRL, extensor carpi radialis longus. Time scale is in ms; 0 = onset of movement. Vertical scale in *B* and *C* is twice that in *A*.

If one considers 10 directions of movement (i.e., excluding the best agonist and antagonist directions) for 17 EMG recordings (Table 1), temporally shifted bursts were present for ~18% of the directions of wrist movement. In three subjects who were studied extensively, the occurrence of temporally shifted bursts varied from a maximum of 30% of the movement directions (*subject 4*) to a minimum of 10% of the movement directions (*subject 3*). Temporally shifted bursts were not found in the ECRL recording of *subject 2* (Figs. 7*A* and 8*A*) or in the FCR recording of *subject 3* (Fig. 9*A*). Thus overall, the temporally shifted pattern was less prevalent than the amplitude-graded pattern.

Patterns of wrist muscle activity in monkeys

The basic patterns of EMG activity associated with wrist movements of monkeys differed from those of humans in three respects. First, agonist bursts in the wrist extensor muscles of monkeys were prolonged and sometimes lasted 300 ms (Hoffman and Strick 1993, 1995). Second, antagonist bursts were small and were observed consistently only in *monkey C*. Third, ECRL and ECRB displayed considerable tonic activity (~30% of the maximum agonist burst) during the hold period and had a complex pattern of activity during movement (Fig. 11). These features of the activity of wrist muscles in monkeys made it more difficult to identify double bursts and temporally shifted bursts.

We focused our analysis of muscle activity on *monkey C* because this animal had relatively brief agonist bursts and consistently displayed antagonist bursts. As a consequence, we could determine whether this monkey displayed the amplitude-graded or temporally shifted pattern of activity. In *monkey C*, we found clear examples of muscle activity that was characteristic of the amplitude-graded pattern observed in humans. Variations in movement direction were associated with modulations in the amplitude, but not the timing, of agonist and antagonist bursts (Fig. 10, *A* and *B*). Distinct double bursts, with activity over the agonist and antagonist burst intervals, were present in ECU when *monkey C* performed wrist movements in an axis orthogonal to the best agonist and antagonist directions (Fig. 10*C*). When agonist and antagonist bursts were clearly identifiable in *monkeys A* and *B*, EMG recordings in these monkeys also provided evidence for amplitude grading of these bursts.

As noted above, the pattern of activity in ECRL and ECRB in monkeys was complex. For movements in the best agonist direction, these muscles exhibited agonist bursts that were prolonged compared with the bursts in human wrist muscles (compare Fig. 11, *A* and *D*, with 2, *A* and *C*). For movements in the best antagonist direction, these muscles exhibited a pronounced suppression of tonic activity before movement onset, followed by a small antagonist burst (Fig. 11, *B* and *E*). This pattern was similar to that found in human wrist muscles (Fig. 2, *A* and *C*), except that the suppression of tonic activity was more prominent in monkeys due to the presence of substantial activity during the hold period.

For movements orthogonal to the best agonist and antagonist directions, a shortened and reduced early suppression of EMG activity was followed by a burst of activity during the agonist burst interval and activity in the antagonist burst interval (Fig. 11, *C* and *F*). This pattern differed from the typical double burst seen in human studies. However, we believe that the mechanisms for generating the human and monkey patterns of activity for these directions of movement are potentially the same. We previously defined a double burst as the combination of agonist and antagonist bursts in one muscle (p. 290 in Hoffman and Strick 1986a). In the monkey, the concept of double bursts needs to be modified to include the summation of three elements: the early antagonist suppression (e.g., Fig. 11, *B* and *E*) as well as the agonist burst and the antagonist burst. The complete shape of the antagonist suppression is unknown because the absence of EMG activity may not indicate the full extent of motoneuron hyperpolarization that occurs. However, if the early suppression possessed an exponential decay, then the sum of the suppression with an agonist burst might account for the delayed onset of the first burst of EMG activity seen in Fig. 11, *C* and *F*.

Quantitative analysis of muscle activity during the agonist burst interval for 16 directions of wrist movement in two monkeys (*A* and *C*) showed that activity during this interval

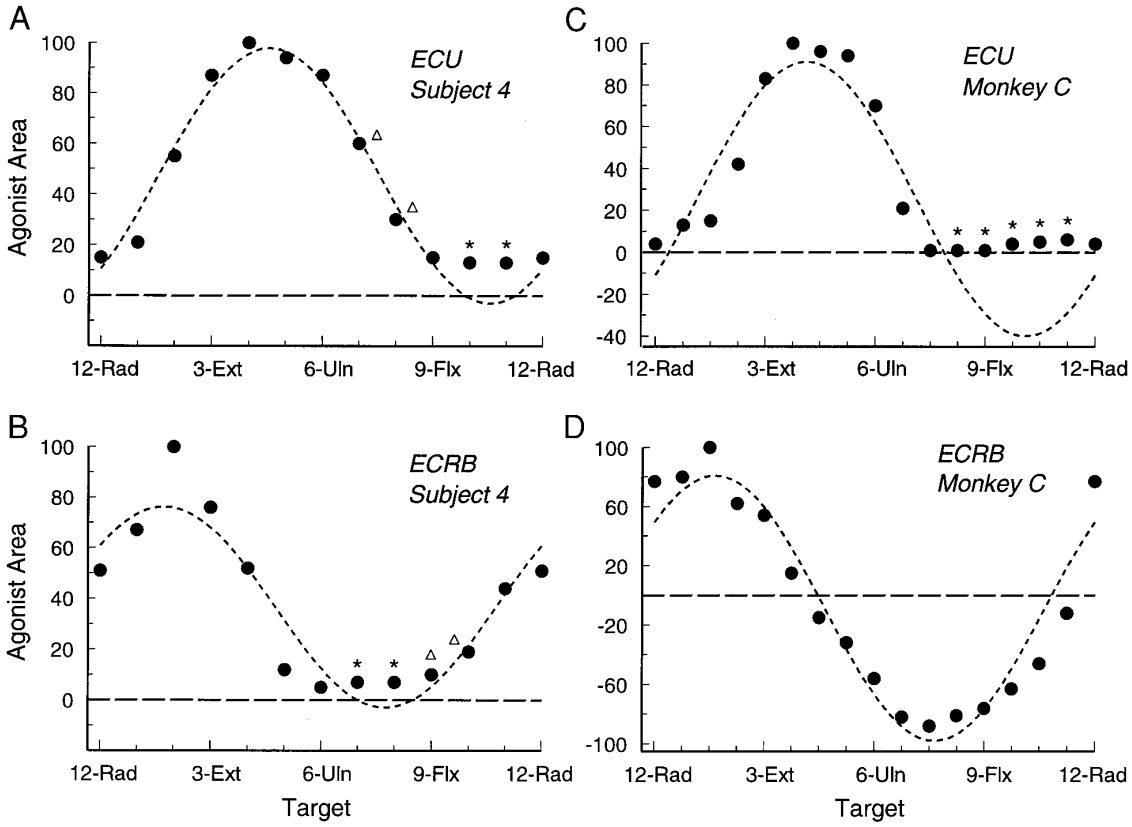

FIG. 5. Cosine tuning of agonist burst in human and monkey wrist muscles. *A*: best cosine fit to the amplitude of the agonist burst in a human subject, $R^2 = 0.97$. *B*: poorest cosine fit to the amplitude of the agonist burst in a human subject, $R^2 = 0.84$. ECRB, extensor carpi radialis brevis. *C*: cosine tuning of ECU in *monkey C*, $R^2 = 0.94$. *D*: cosine tuning of ECRB in *monkey C*, $R^2 = 0.93$. *, points that were given a weighting of 0.01 in the regression (see METHODS). △, bursts that were temporally shifted. Abscissa: numbers indicate target number. Ordinate: 0 = EMG level during the hold period; 100 = maximum integrated EMG for any direction of movement; negative = decrease in EMG below level during the hold period.

followed a cosine function for all four wrist muscles in both monkeys ($R^2 > 0.88$, e.g., Fig. 5, *C* and *D*). Each of the wrist muscles examined displayed at least one instance of an exceptional fit to a cosine function ($R^2 = 0.94–0.98$). As noted above, ECRL and ECRB displayed marked tonic activity during the hold period. During the agonist burst interval, these muscles exhibited systematic increases and decreases in activity that varied as a continuous, cosine-tuned function of direction (Figs. 5*D* and 12).

The preferred directions of individual muscles in monkeys were similar to those of humans (Table 2, compare filled arrows in Figs. 6 and 12). However, in the monkey, there was a large gap of ~160° between the preferred directions for ECU and FCR. It is interesting to note that the preferred directions for the early suppression in ECRL and ECRB occurred in this gap.

Wrist muscles in monkeys were less broadly tuned than those in humans (compare Figs. 6 and 12). For example, only ECRB was significantly activated when *monkey C* performed movements to the target at 1:30 (Fig. 12). It is clear that some directions of wrist movement in the monkey were generated by combining activation of one wrist muscle with the suppression of activity in another muscle. For example, movements to the targets at 4:30 and 6 were generated by activation of ECU and suppression of ECRL and/or ECRB (Fig. 12). Movements to the targets at 7:30, 9, and 10:30

TABLE 2. *Preferred directions*

	ECRL	ECRB	ECU	FCR
Human				
Agonist	14 ± 6.3 (6)	42 ± 28.8 (3)	135 ± 7.4 (5)	253 ± 8.8 (3)
Antagonist	216 ± 22.1 (5)	183 (1)*	331 ± 6.8 (2)*	98 (1)*
Monkey				
Agonist	354 ± 12.0 (2)	36 ± 18.8 (2)	118 ± 8.1 (2)	283 ± 3.3 (2)
Stimulation	1 ± 1.7 (3)	13 ± 4.5 (3)	170 ± 1.8 (3)	299 ± 12.3 (3)

Numbers are means ± SD in deg, 0 = radial deviation (see Fig. 1); *n* values are in parentheses. * Preferred direction was calculated for data that were fit by a cosine regression with $R^2 > 0.5$.

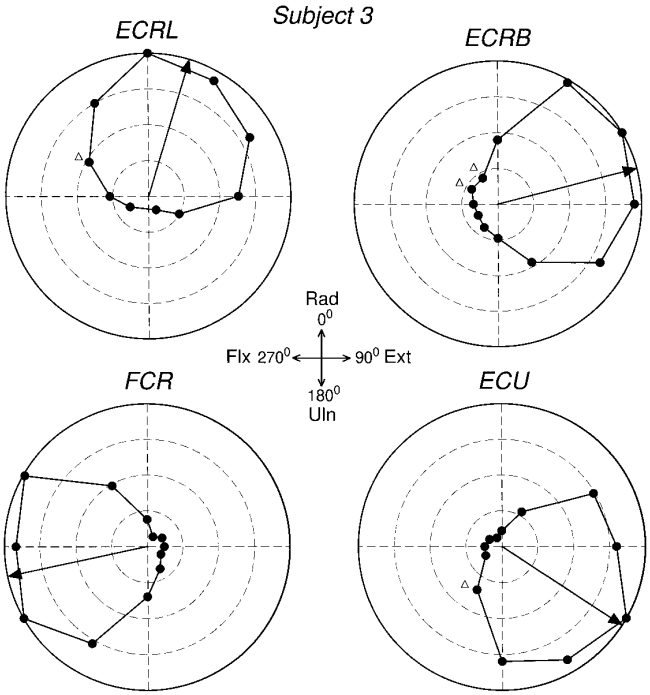

FIG. 6. Directional tuning of the agonist burst in 4 wrist muscles of *subject 3*. ●, integral of EMG activity during the agonist burst interval for 12 different directions of movement. →, preferred direction for each muscle. Preferred direction was determined from the cosine fit for each recording. Outer circle indicates the maximum integrated EMG activity observed for any of the 12 directions of movement. △, bursts that were temporally shifted. FCR, flexor carpi radialis.

were generated by activation of FCR and suppression of ECRL and/or ECRB (Fig. 12).

We did not observe clear examples of the temporally shifted pattern of EMG activity in the three monkeys that we examined. The presence of substantial amounts of tonic activity before movement onset, the occurrence of prolonged agonist bursts, and the early suppression of EMG activity for some directions of movement may have obscured the presence of a temporally shifted pattern of activity, particularly in ECRL and ECRB. However, these difficulties were largely not present in recordings from ECU in monkeys. Still, we did not observe the temporally shifted pattern in ECU of monkeys, even though this pattern was clearly evident in the same muscle of human subjects. Thus we believe that the absence of the temporally shifted pattern in monkeys reflects a limit in the movement strategies available to or utilized by monkeys to control step-tracking movements.

Electrical stimulation of muscle in monkey

We stimulated each wrist muscle to determine its ''pulling direction.'' Stimulation was tested while the monkey held the manipulandum in the central hold position. This required tonic activity in ECRL and ECRB. However, this muscle tension was the same as that needed for normal performance of the step-tracking task. Overall, the movements produced by muscle stimulation were markedly curved (Fig. 13). The initial evoked movement was followed by a marked deviation of the wrist toward extension or flexion, depending on whether the muscle was a flexor or extensor. Because a variety of factors could produce the later curved portion of

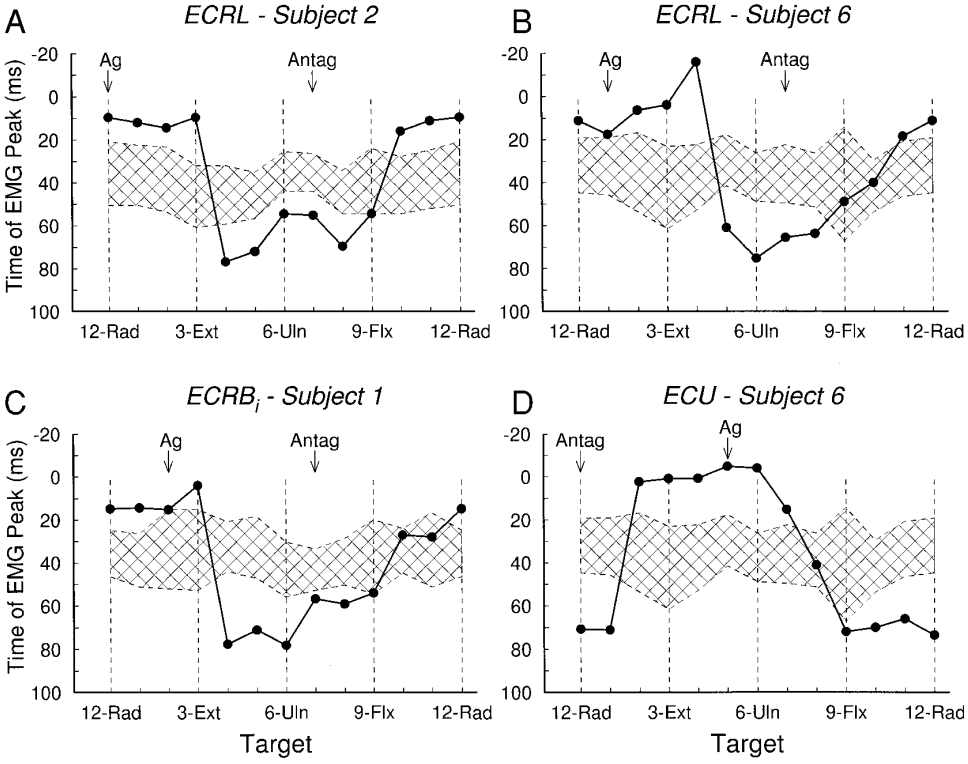

FIG. 7. Analysis of temporal shift. *A*: absence of temporal shift in ECRL. *B*: temporal shift in ECRL for movements to targets 9 and 10. *C*: temporal shift in ECRB for movements to targets 10 and 11. *D*: temporal shift in ECU for movements to target 8. Hatched area in each panel indicates the interval between peak acceleration and peak velocity. Ag (arrow) and Antag (arrow) indicate the best agonist and antagonist directions for each muscle. Abscissa: numbers indicate target number. Ordinate is in ms; 0 = onset of movement.

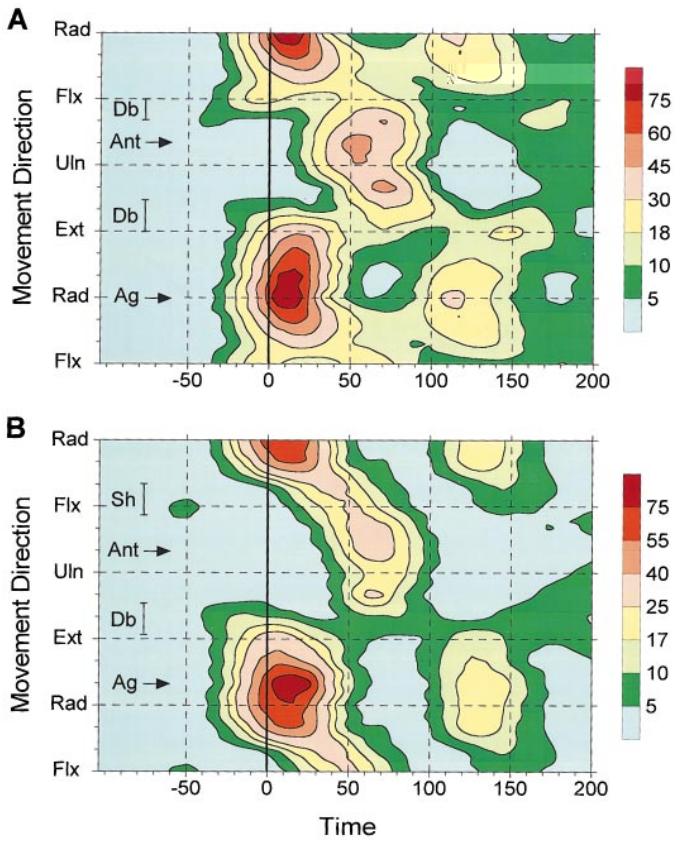

FIG. 8. Temporal shift and double bursts in ECRL. *A*: amplitude-graded pattern in *subject 2* (see also Fig. 7*A*). Red area at time = 0–25 indicates the occurrence of agonist bursts (Ag). Several orange peaks at time = 50–75 indicate the occurrence of antagonist bursts (Ant). Note that a sudden transition occurs between the 2 areas, with double bursts (Db) in the transitional area. *B*: amplitude-graded and temporally shifted patterns in *subject 6* (see also Figs. 3, *A–C*, and 7*B*). Note that a sudden transition occurs between agonist and antagonist bursts near wrist extension and is marked by double bursts (Db). Gradual transition occurs between agonist and antagonist bursts for wrist flexion and is marked by temporally shifted bursts (Sh). Ag (arrow) and Antag (arrow) indicate the best agonist and antagonist directions for each muscle. Db (bracket) indicates movement directions with double bursts. Sh (bracket) indicates movement directions with temporally shifted bursts. Time scale is in ms; 0 = onset of movement. EMG scale: 100 = maximum EMG observed, 0 = EMG level during hold period.

the evoked movement, we defined the pulling direction of a muscle as the direction of the initial 40–50 ms of evoked movement.

In general, the pulling directions of individual wrist muscles were very repeatable both within and between monkeys (Table 2). However, stimulation of FCR resulted in an initial direction of movement that varied by ≤30° in different sessions in the same monkey. This variation appeared to depend on the specific location of the EMG electrodes within FCR. The initial pulling direction was close to radial deviation for two muscles (ECRB, ECRL), close to ulnar deviation for two muscles (ECU, FCU), and 30° away from flexion for one muscle (FCR). No muscle had an initial pulling direction toward wrist extension.

Table 2 provides a comparison of the pulling directions of the wrist muscles with the preferred directions of the same muscles. On average, the preferred directions of ECRL, ECRB, and FCR closely matched their pulling directions. Only ECU exhibited a consistently large (50°) difference between its preferred direction and its pulling direction. In all monkeys, the preferred direction of ECU was shifted toward extension and away from its initial pulling direction of ulnar deviation. In *monkey C*, the preferred direction of ECRB also was shifted toward extension and away from its

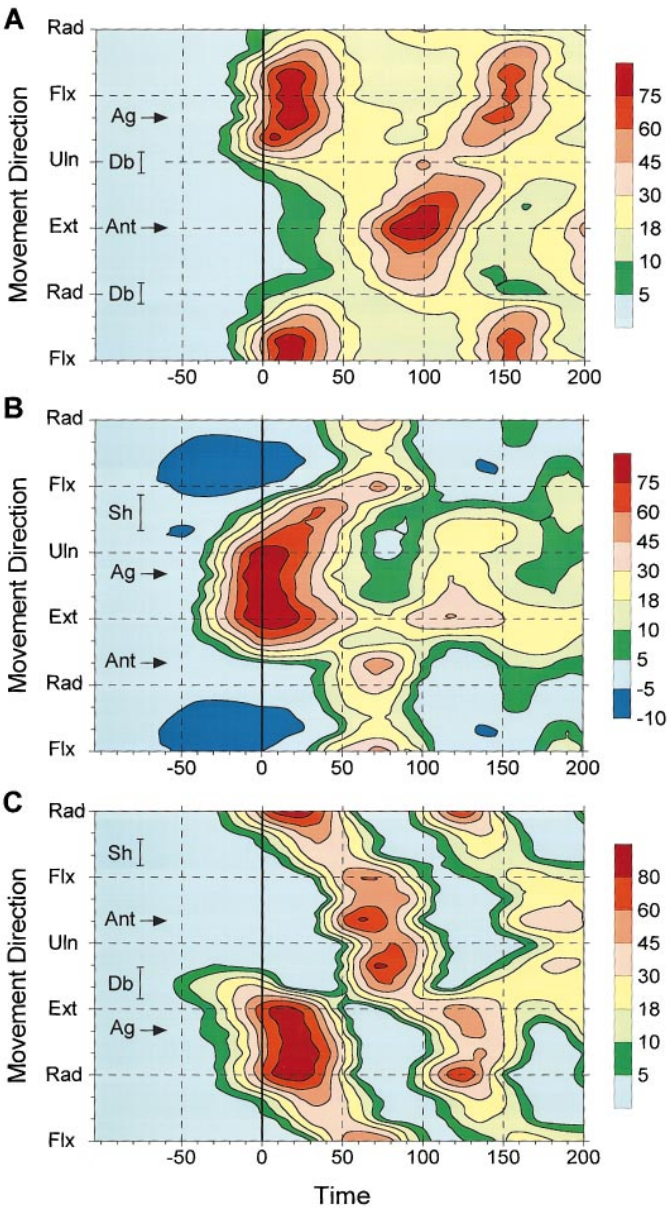

FIG. 9. Temporal shift and double bursts in several wrist muscles. *A*: amplitude-graded pattern in flexor carpi radialis of *subject 3.* Note that a sudden transition occurs between agonist and antagonist bursts and is marked by double bursts (Db). *B*: amplitude-graded and temporally shifted patterns in extensor carpi ulnaris of *subject 6* (see also Figs. 3, *D–F*, and 7*D*). Dark blue area indicates that muscle activity occurring during the hold period was suppressed for some directions of movement. Note that a gradual transition occurs between agonist and antagonist bursts for combined wrist flexion + ulnar deviation and is marked by temporally shifted bursts (Sh). *C*: amplitude-graded and temporally shifted patterns in extensor carpi radialis brevis of *subject 1* (see also Fig. 7*C*). Note the occurrence of double bursts for movements near wrist extension and the occurrence of temporally shifted bursts for movements near wrist flexion.

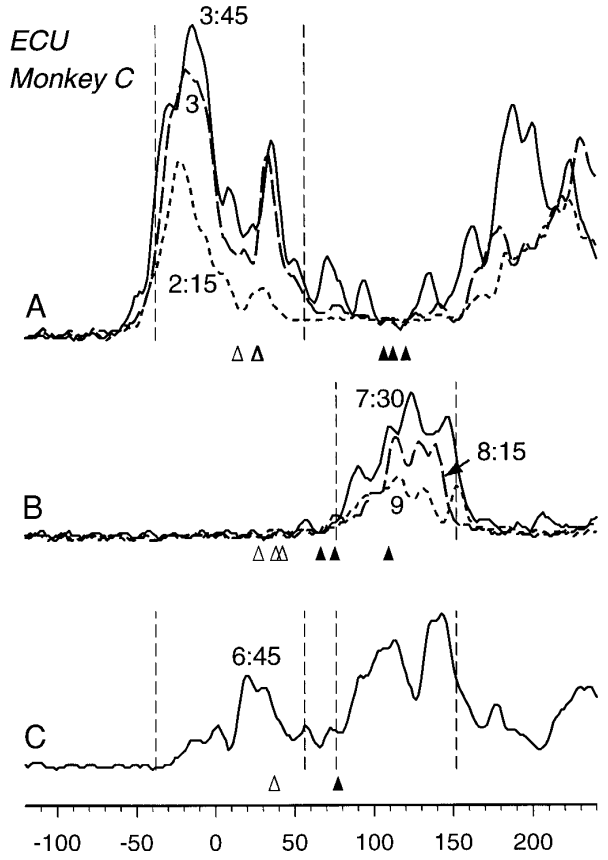

FIG. 10. Amplitude-graded pattern in *monkey C*. *A*: amplitude grading of the agonist burst for movements to target locations 3:45 (solid line), 3 (long dashed line), and 2:15 (short dashed line). Vertical dashed lines indicate the beginning and end of the agonist burst interval. *B*: amplitude grading of the antagonist burst for movements to target locations 7:30 (solid line), 8:15 (long dashed line), and 9 (short dashed line). Vertical dashed lines indicate the beginning and end of the antagonist burst interval. *C*: double burst for movements to target location 6:45. EMG traces were full-wave rectified, filtered (τ = 10 ms), and averaged (n = 12–29). Open triangles: times of peak acceleration. Closed triangles: times of peak velocity. Time scale is in ms; 0 = onset of movement.

initial pulling direction of radial deviation (Fig. 12). We believe that these instances of disparity between preferred direction and pulling direction result because the absence of a muscle that pulls toward ''pure'' extension requires the increased involvement of ECU and ECRB to generate this direction of movement. Thus the preferred directions of these muscles are rotated away from their pulling direction toward the direction of pure extension.

DISCUSSION

In humans, movement direction is controlled by modulating muscle activity in two distinct patterns, termed *amplitude graded* and *temporally shifted*. Our results indicate that the amplitude-graded pattern is the one used most frequently by humans to generate step-tracking movements of the wrist. The amplitude-graded pattern is characterized by modulation of the quantity of muscle activity during two distinct time periods, corresponding to agonist and antagonist burst intervals. As a result, for some directions of movement, EMG activity appears as double bursts because it is present during both time intervals. Examples of double bursts have been observed previously not only in wrist muscles (Hoffman and Strick 1986a) but also in muscles that move the elbow (Fig. 9 in Sergio and Ostry 1995; Fig. 8*A*, 4 and 6, in Wadman et al. 1980) and shoulder (Fig. 5, directions 6 and 8 in Pellegrini and Flanders 1996). Thus the amplitude-graded pattern of muscle activity appears to be an essential feature of arm movements at proximal as well as distal joints.

We believe that the amplitude-graded pattern and the appearance of double bursts provide insights into the neural processes used to determine the direction of a step-tracking movement. Our hypothesis is that, on a muscle by muscle basis, an intended movement is decomposed into an agonist, ''propulsive'' component, and an antagonist, ''braking'' component (Hoffman and Strick 1986a). These components are graded individually in amplitude but are relatively fixed in time. Double bursts are created when separate agonist and antagonist components for each muscle are summed at the level of the motoneuron pool. Our hypothesis implies that the signals for agonist and antagonist bursts are, at some level of the nervous system, independently generated (see also Sergio and Ostry 1995).

Our study is unique in examining separately the tuning of activity during the agonist and antagonist burst intervals (cf. Buchanan et al. 1986; Flanders and Soechting 1990; Flanders et al. 1996). We found that the agonist burst was

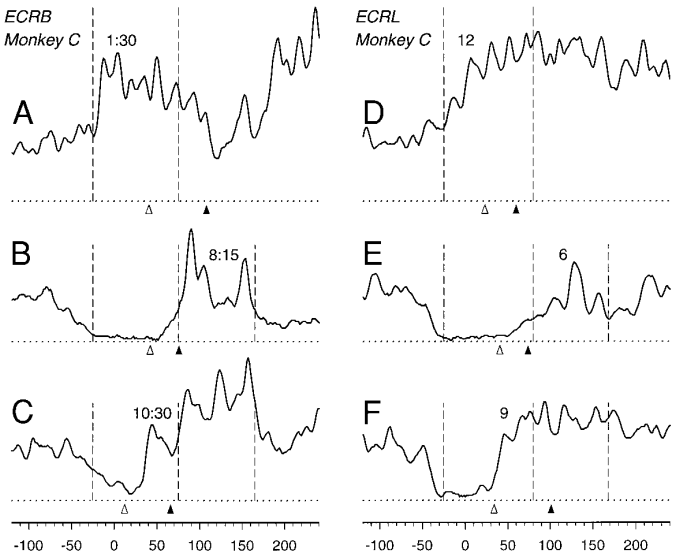

FIG. 11. Complex pattern of activity in ECRL and ECRB of *monkey C*. *A*: agonist burst for movements in the best agonist direction. Vertical dashed lines indicate the agonist burst interval. *B*: suppression of tonic activity, followed by an antagonist burst, for movements in the best antagonist direction. Vertical dashed lines indicate the agonist and antagonist burst intervals. *C*: complex pattern of activity for movements orthogonal to the best agonist direction. Vertical dashed lines indicate the agonist and antagonist burst intervals. *D*: agonist burst for movements in the best agonist direction. Vertical dashed lines indicate the agonist burst interval. *E*: suppression of tonic activity, followed by a small antagonist burst, for movements in the best antagonist direction. Vertical dashed lines indicate the agonist and antagonist burst intervals. *F*: complex pattern of activity for movements orthogonal to the best agonist and antagonist directions. Vertical dashed lines indicate the agonist and antagonist burst intervals. All EMG traces were full-wave rectified, filtered (τ = 10 ms), and averaged (n = 10–18). Open triangles: times of peak acceleration. Closed triangles: times of peak velocity. Time scale is in ms; 0 = onset of movement.

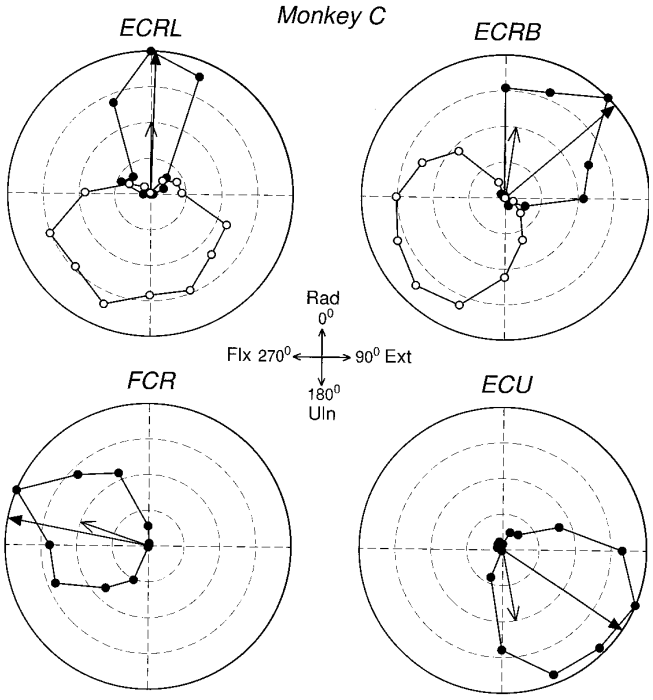

FIG. 12. Directional tuning of muscle activity in *monkey C.* Closed circles plot increases in muscle activity during the agonist burst interval. Open circles plot decreases in muscle activity during the agonist burst interval. Closed arrows point toward the muscle's preferred direction, which was determined from the cosine fit. Open arrows point toward the muscle's ''pulling direction,'' i.e., the initial direction of movement resulting from intramuscular stimulation. Outer circle indicates the maximum integrated EMG activity observed for any of the 16 directions of movement.

modulated according to a cosine function. In some cases, the antagonist component also was cosine tuned. Surprisingly, in most instances, the preferred directions for agonist and antagonist activity differed by $<170°$ and therefore, were not directly opposite to each other (see also Flanders et al. 1996). This result provides further support for the proposal that the neural processes that determine the properties of antagonist muscle activity are partially separate from those that determine agonist activity (Hoffman and Strick 1986a, 1990).

The second pattern of muscle activity, temporally shifted, is characterized by a gradual and systematic delay in the timing of a single burst of muscle activity relative to movement onset. Several studies have provided clear examples of temporally shifted bursts in shoulder and elbow muscles with changes in the direction of reaching movements (Flanders 1991; Flanders et al. 1994, 1996; Karst and Hasan 1991; Wadman et al. 1980). Our results demonstrate that this pattern also occurs in distal muscles. The presence of this pattern of activity in both proximal and distal muscles and the size of temporally shifted bursts (i.e., intermediate between agonist and antagonist bursts in the same muscle) highlight the functional importance of this pattern of muscle activity.

Despite its apparent functional importance, the temporally shifted pattern was used infrequently by human subjects to generate wrist movements and was not observed in the three monkeys examined in this study. Furthermore, temporally shifted bursts in wrist muscles were restricted to particular directions of movement. This also appears to be the case for shoulder muscles (see Figs. 6, right, MD, and 7, BI, in Flanders et al. 1996). These observations suggest that the temporally shifted pattern is not simply an alternative to the amplitude-graded pattern. Rather, we believe that the temporally shifted pattern serves a unique function that is required for a specific set of movement conditions.

Our hypothesis is that the temporally shifted pattern functions to reduce the amount of movement curvature that would occur with contraction of particular combinations of agonist muscles. This proposal is based on the following observations. First, temporally shifted bursts in wrist muscles followed agonist bursts by ~25–50 ms. The force resulting from these delayed EMG bursts would influence the trajectory of a movement not at its onset but later during the initial portion of its path. Second, temporally shifted bursts were present only for movements that were approximately perpendicular to the pulling direction of a muscle. These bursts were spatially inappropriate to apply force in the specific direction of the intended movement. Third, electrical stimulation of single wrist muscles in monkeys produced curved trajectories (Fig. 13). The origins and insertions of wrist muscles in humans are similar to those of monkeys (Hartman and Straus 1969). As a consequence, the anatomic arrangement of single wrist muscles in humans also should produce curved movement trajectories. Finally, in comparing our human and monkey results, there appeared to be an association between the presence or absence of temporally shifted bursts and the extent to which the initial movement trajectories were curved. Human subjects displayed temporally shifted bursts and could make wrist movements that displayed little curvature during the initial portion of the movement trajectory (Fig. 1, *left,* ■). In contrast, monkeys did not display clear examples of temporally shifted bursts, and the trajectories of their movements began to curve ~50

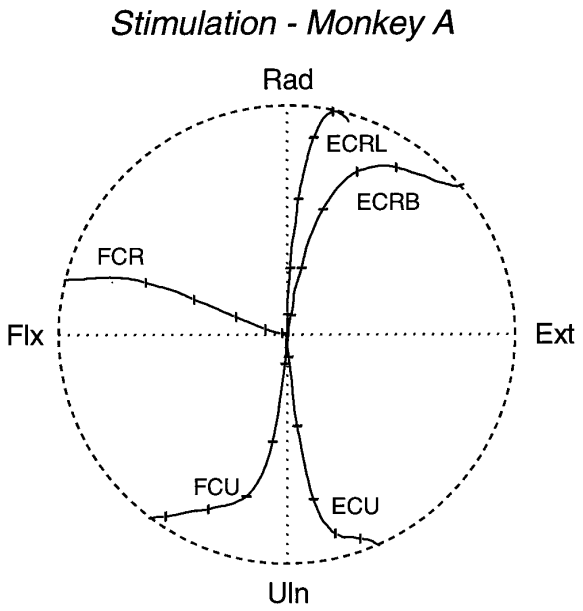

FIG. 13. Wrist movements evoked by intramuscular stimulation in *monkey A.* Each trace represents the average of 8 muscle stimulations (600 μA to 2 mA, 10 pulses at 50/s). Time period illustrated is from movement onset to 105–145 ms later. Tick marks indicate 20-ms intervals, $\leq$100 ms after movement onset. Movement amplitude has been rescaled so that the maximum movement amplitude is the same for all muscles.

ms after movement onset for some directions of movement (Fig. 1, *right,* ■). With specific and extended training, monkeys might be induced to generate wrist movements that were more similar to those of humans. If so, we predict that a straightening of movement trajectories in monkeys would be associated with the emergence of temporally shifted bursts.

On the other hand, one should use caution in interpreting this comparison between monkey and human movements. The movements of human subjects were faster and had more overshoot than the movements of the monkeys. Thus the different patterns of muscle activity demonstrated by monkeys and humans also may reflect the use of different behavioral strategies to perform the step-tracking task.

Our hypothesis about the function of the temporally shifted pattern for wrist movements is similar to that of Hollerbach and Atkeson (1987) concerning the control of reaching movements. They proposed that straight reaching movements are achieved by ''staggered joint interpolation,'' i.e., by staggering the onset of shoulder and elbow joint movements. This would be accomplished by adjusting the time of onset of EMG activity at these two joints. Wadman et al. (1980) also proposed that the time shift between activation of elbow and shoulder muscles was an important factor in determining trajectory shape. Our proposal contrasts with the suggestion of Pellegrini and Flanders (1996) that asynchronous patterns of muscle activity may contribute to hand path curvature during reaching movements. Thus we believe that the exceptional skill of human subjects in generating relatively straight trajectories, whether at the shoulder or the wrist, may depend on their ability to adjust accurately not only the amplitude but also the timing of EMG bursts.

Central generation of muscle activity

How does the CNS generate the complex spatiotemporal patterns of muscle activity that are necessary for determining the direction of wrist movement? Although many central sites may participate in this process, it is likely that the primary motor cortex (M1) plays a key role. Lesions or inactivation of M1 lead to disruption of the normal patterns of muscle activity associated with limb movements (e.g., Hoffman and Strick 1995; Matsumura et al. 1991). Specifically, we have shown that removal of the arm area in M1 results in a misdirection of step-tracking movements of the wrist due to changes in the spatial and temporal pattern of muscle activity about the wrist joint (Hoffman and Strick 1995).

There are apparent similarities between the spatial patterns of activity in wrist muscles and of single neurons in M1 for movements in different directions. These correspondences are present, despite the fact that single neurons were recorded in the elbow-shoulder region of M1 during reaching movements. For example, both wrist muscles and single neurons in M1 are broadly tuned for movement direction. In fact, the amplitude of muscle activity during the agonist burst interval, like the frequency of firing of M1 neurons, is cosine-tuned with movement direction (e.g., Caminiti et al. 1990; Fu et al. 1995; Georgopoulos et al. 1982; Kalaska et al. 1989; Schwartz et al. 1988). The similarities between neuron and muscle activity suggest that grading of the amplitude of agonist activity in wrist muscles may be due, in part, to the directional properties of M1 neurons.

One feature of the activity of wrist muscles is strikingly different from the findings for M1 neurons related to reaching movements. The preferred directions of wrist muscles in the monkey displayed an uneven distribution, characterized by a large gap (160°) between the preferred directions of ECU and FCR. In contrast, the preferred directions of M1 neurons during reaching movements are reported to be uniformly distributed in space (e.g., Schwartz et al. 1988; see, however, Scott 1997; Scott and Kalaska 1997). A uniform distribution is a key requirement of the population vector hypothesis. According to this hypothesis, each M1 neuron active for a particular movement makes a vectorial contribution to movement direction based on its preferred direction (Georgopoulos et al. 1988). The magnitude of the contribution is proportional to the cell's discharge. It will be important to determine the distribution of preferred directions for wrist-related neurons in M1 and to compare this with the distribution of preferred directions for wrist muscles. The differences between these two distributions will provide information about the nature of the transformation that occurs between M1 and motor output from the spinal cord. Any similarities in the two distributions would suggest that the representation of motor output in M1 is not as abstract as the population vector hypothesis suggests (see also Scott 1997; Scott and Kalaska 1997).

Although many studies have considered the role of M1 in determining the spatial parameters of motor output, the involvement of M1 in generating temporal patterns of muscle activity has received little attention (cf. Sergio and Kalaska 1998). Yet our results emphasize that muscle activity occurs in distinct temporal bursts during step-tracking movements. The agonist and antagonist bursts in wrist muscles occur over separate time intervals and appear to be generated independently (see also Hoffman and Strick 1986a, 1990, 1993). The demonstration that, under certain circumstances, the activity of wrist muscles in human subjects shows a temporal shift and occurs over an interval between the agonist and antagonist bursts would make the generation of spatiotemporal patterns of muscle activity even more complex in humans.

In a related study, we found that monkeys with M1 lesions failed to generate distinct agonist or antagonist bursts and failed to display the marked decrease in antagonist activity that normally precedes the agonist burst. These observations emphasize the importance of M1 in producing the temporal patterns of muscle activity associated with step-tracking movements. If M1 neurons contribute to the generation of both agonist and antagonist bursts, separate neurons could produce temporal patterning by having a broad range of onset times in relation to the initiation of movement. This result has been seen in many prior studies of neuron activity in M1 (e.g., Cheney and Fetz 1980; Evarts 1974; Murphy et al. 1982; Smith et al. 1975; Wannier et al. 1991). Furthermore, M1 neurons with preferred directions close to the intended movement direction (agonist-related) should become active much earlier than neurons with preferred directions opposite to the intended movement direction (antagonist-related) (Georgopoulos et al. 1982). As a consequence, the population vector for neurons in M1, when measured over small time intervals, would point first in the direction

of net agonist activity, then would rotate to the direction of net antagonist activity, and finally would settle in the direction of net activity required to hold the limb in the final position (see Fig. 13.8 in Kalaska and Drew 1993; Sergio and Kalaska 1998). This prediction remains to be examined for wrist movements.

A final important question is whether M1 neurons show a form of functional specialization in which one population of neurons participates in generating the agonist burst in a muscle and another population participates in producing the antagonist burst in the same muscle. If single neurons are related to only a single burst of muscle activity, then the changes in discharge rate for these neurons would be relatively fixed in relation to movement onset as movement direction varied. On the other hand, if single neurons participate in generating both bursts of muscle activity, then the changes in discharge rate for these neurons would vary in timing with movement direction, as does the activity of single muscles. These neurons would be active before movement onset for some directions of movement and after movement onset for movements in nearly the opposite direction. These issues can be examined only in animals trained to make step-tracking movements that require an antagonist burst.

Our study has shown that several wrist muscles are coactivated in different combinations for each movement direction. Similar observations have been made for movements at other joints (e.g., Buchanan et al. 1989; Jamison and Caldwell 1993; Macpherson 1988; Schieber 1995). In addition, we have demonstrated that temporal relations between individual muscles vary with movement direction (Flanders et al. 1996). For example, the closely related synergists, ECRL and ECRB, are coactivated temporally for some directions of wrist movement but are active over separate time intervals for other directions of movement. Our lesion study indicates that the appropriate temporal coactivation of muscles requires an intact M1 because its removal was associated with incorrect sequencing of synergistic muscles (Hoffman and Strick 1995). The well-known branching of single corticospinal neurons to influence neurons in multiple motor nuclei may be a mechanism for coactivating muscles (Fetz and Cheney 1980; Kasser and Cheney 1985; Shinoda et al. 1979, 1981). However, an understanding of how synergists may be activated during distinct time intervals will require greater attention to the temporal patterns of neuron activity (see also Bennett and Lemon 1996).

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