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RESEARCH ARTICLE

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Common principles underlying the control of rapid, single degree-of-freedom movements at different joints

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Abstract Studies of rapid, single degree-of-freedom movements have shown different changes in electromyographic patterns for movement tasks that appear very similar (e.g., movements over different ranges of distance). However, it is not clear whether these differences are a result of joint-specific control schemes or whether they are instead due to the limited range of task parameters studied relative to the mechanical constraints of each joint (e.g., short compared with long movements relative to the range of motion of a particular joint). In this study, we measured and compared the kinematic trajectories and electromyograms recorded during various movement tasks at the wrist, elbow, and ankle. Subjects performed movements over a wide range of distances "as fast as possible," "at a comfortable speed," and against two inertial loads (at the elbow only), and they performed movements over a fixed distance at three different speeds at the wrist and ankle. For fast movements we show that, in spite of some joint-specific differences, the basic pattern of electromyographic (EMG) modulation is similar at all three joints; for example, the agonist EMG burst transitions from a fixed duration to an increasing duration with increasing movement distance at all three joints. Moreover, the distance at which this transition occurs in one joint relative to the distance at which this transition occurs in the other two joints is consistent across subjects. The transition occurs at the shortest distance at the ankle and the longest distance at the wrist. In general we suggest that the data are consistent with a single set of control rules applied at all three joints, with the biomechanical constraints at each joint accounting for the differences in the EMG and kinematic patterns observed across joints.

Key words Electromyogram · Movement · Elbow · Wrist · Ankle · Human

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Introduction

Rapid, single degree-of-freedom movements have been the subject of much research since the seminal work by Wachholder and Altenburger (1926). The muscle activity associated with these movements is commonly characterized by alternating bursts of agonist and antagonist electromyographic (EMG) activity. Various control schemes have been suggested that are defined by the modulation of the height of the agonist EMG burst, the duration of the agonist EMG burst, and/or the timing of the agonist and antagonist EMG patterns during rapid movements. However, it is not clear to what extent each scheme is limited to: (1) a specific range of the task variable investigated (e.g., "short" compared with "long" movements), (2) a specific joint (e.g., proximal compared with distal, upper limb compared with lower limb), or (3) a specific type of movement task (e.g., control of distance, load, speed, or accuracy).

Some researchers have found that the duration of the agonist burst remains constant with changes in distance (thumb, Hallett and Marsden 1979; wrist, Hoffman and Strick 1990; elbow, Brown and Cooke 1981). In contrast,

others have found that the duration of the agonist burst increases with movement distance (wrist: Berardelli et al. 1984; elbow: Brown and Cooke 1984; Gottlieb et al. 1989a). Berardelli et al. (1984) had subjects perform short and long movements at the wrist and elbow. These authors showed that the type of agonist burst modulation was contingent on the range of movement distance examined. That is, shorter movements at both the wrist and elbow were associated with modulation of agonist burst height with a fixed duration, and longer movements were associated with modulation of the duration of the agonist burst. Therefore, the differences in the agonist control patterns (e.g., height modulation at the wrist compared with duration modulation at the elbow) appear to be a result of the range of movements tested rather than different control rules.

EMG patterns associated with a specific joint may also reflect the biomechanical constraints of that joint and its muscles rather than a fundamentally distinct method of modulating EMG patterns for a particular task. As discussed by Hoffman and Strick (1993), data from movements at proximal joints, such as the elbow, appear to show control schemes that differ from those at distal joints, such as the wrist and thumb. Hoffman and Strick (1993) found that, when they added inertial and elastic loads at the wrist, subjects generated wrist movements in which the modulation of the agonist burst was similar to agonist burst modulation during elbow movements (Berardelli et al. 1984) and saccadic eye movements (Robinson 1970), respectively. From these and other results, Hoffman and Strick concluded that control rules are not joint-specific. Instead, the specific EMG patterns observed for variations of a particular task parameter may be determined by the physiological and biomechanical characteristics of each joint (cf. Cheron and Godaux 1986; Godaux 1989).

Another approach to understanding control patterns of single-joint movements is to consider the constraints imposed on the system by different movement tasks. Gottlieb, Corcos, and Agarwal have drawn a distinction between movement tasks in which movement speed is explicitly controlled ("speed-sensitive") and tasks in which some other parameter such as distance is explicitly controlled ("speed-insensitive"; Corcos et al. 1989; Gottlieb et al. 1989a, b). At the joint and movement distances originally studied (elbow, more than 18°), the task constraints and resulting control rules appeared to have a unique relationship. Speed-sensitive movements were characterized by modulation of the initial slope and a constant duration of the agonist EMG burst, whereas speed-insensitive movements were characterized by a constant initial slope and modulation of the duration of the agonist EMG burst. However, the unique relationship between the EMG parameter that is modulated and the type of task was not observed for shorter movements (Gottlieb et al. 1996a). For the elbow, the two control strategies have been combined into a single set of nonlinear control rules derived from the various task constraints (Gottlieb 1993; Gottlieb et al. 1995). Although the task-specific control strategies have not been systematically studied within this framework at many other joints, the existing data show many similarities in the control patterns. For example, Hoffman and Strick (1993, their Fig. 13) have shown that, when speed is explicitly controlled at the wrist joint, the initial slope of the agonist EMG burst is modulated. If this observation is supported with additional experimentation, it further strengthens the argument that single degree-of-freedom movements at all joints are controlled by common sets of control rules.

The goal of the present experiments was to analyze changes in agonist and antagonist EMG patterns for movements under a range of constraints at three joints (wrist, elbow, and ankle). To achieve this goal, we (1) extended the study of Berardelli et al. (1984) by analyzing the agonist and antagonist EMG patterns during movements made as fast as possible over a wide range of distances at the wrist, elbow, and ankle; (2) expanded the work of Hoffman and Strick (1993) by reducing the inertial load at the elbow to shift the biomechanical constraints toward the conditions commonly encountered at the wrist; and (3) augmented the studies of Gottlieb and colleagues (Corcos et al. 1989; Gottlieb et al. 1989a, 1990) by examining task-dependent control patterns at three joints. Our results provide further support for the idea that task-specific force requirements explain the similarities and differences in observed EMG patterns during rapid movements at the wrist, elbow, and ankle (Cheron and Godaux 1986; Godaux 1989; Gottlieb et al. 1989a; Hoffman and Strick 1989, 1993).

Materials and methods

Subjects

Experiments were performed on nine neurologically healthy individuals between the ages of 20 and 41 (two women and seven men). Informed consent was obtained from all subjects according to Medical Center-approved protocols.

Experimental setup

The subject viewed a computer monitor that displayed a cursor positioned along the horizontal axis by joint angle. A small, stationary marker corresponded to the initial position. A broad band, 3° in width, was located at the desired angular distance.

Elbow joint

The subject was seated with the right arm abducted 90°. The forearm was strapped to a rigid, lightweight manipulandum that could freely rotate only in the horizontal plane. The axis of rotation was aligned with the elbow. Full extension was defined as 90°; elbow flexions were in the negative direction. The initial position was 35°. Joint angle was measured by a capacitative transducer mounted on a shaft at the axis of rotation. Joint angle was digitally differentiated to generate joint velocity. Joint acceleration was measured by a piezoresistive accelerometer mounted 47.6 cm from the center of rotation. Joint torque was measured by a strain-gauge torque transducer mounted on a shaft at the axis of rotation. Surface EMGs were recorded from the biceps, brachioradialis, and triceps (lateral and long

heads). The signals were amplified (gain 1600) and band-pass filtered (60–300 Hz). All signals were digitized at 1000/s with 12-bit resolution. The EMG data in the figures are plotted in arbitrary units equal to the voltage output of the amplifiers.

For most experiments, movements were performed against a light mainipulandum whose moment of inertia with the motor was 0.14 Nms^2/rad. For one experiment, we compared elbow flexions against two different inertial loads. The heavy inertial load was generated by one of two methods: (1) a heavy manipulandum, whose moment of inertia with the motor was 0.45 Nms^2/rad, was used, or (2) the light manipulandum was used with a servo-controlled torque motor that further increased the effective inertial load by 0.23 Nms^2/rad for an effective moment of inertia of 0.37 Nms^2/rad. The light inertial load was generated using the light manipulandum with the servo-controlled torque motor to further reduce the effective inertial load by 0.057 Nms^2/rad, resulting in an effective moment of inertia of 0.083 Nms^2/rad.

Wrist joint

The subject was seated with the right arm abducted 30°. The elbow was flexed such that the forearm formed at 90° angle with the upper arm. The forearm rested on a static platform, and the wrist rotated about the vertical axis. The hand was placed in a splint-like device attached to a rigid, lightweight manipulandum (moment of inertia of 0.0087 Nms^2/rad) that could rotate only in the horizontal plane. The wrist was aligned with the axis of rotation of the manipulandum. The hand aligned with the forearm was defined as 0°; wrist flexions were in the negative direction. The initial position was 0°. Surface EMGs were recorded from the flexor carpi radialis (FCR), extensor carpi radialis brevis (ECR), and extensor carpi ulnaris (ECU). Transducers similar to those described for the elbow were used to measure angle, acceleration, and torque.

Ankle joint

The subject was seated such that the thigh was parallel to the floor and the knee was flexed 90°. Zero degrees was defined as the position in which the foot was parallel to the floor and formed a 90° angle with the leg. The initial position was −15°, which was defined as 15° of ankle extension (in the plantar direction). A fixed initial position was used rather than the position of the foot when the subject relaxed, because we wanted to keep the effects of gravity comparable amongst subjects. The foot was strapped into a rigid, lightweight manipulandum that could only rotate in the sagittal plane. Transducers similar to those described above were used to measure angle and torque. Because gravity contributes a changing signal to an accelerometer rotating in the vertical plane, we chose to differentiate the angle signal twice to determine acceleration. Surface EMGs were recorded from tibialis anterior (TA), soleus, and gastrocnemius (lateral and medial heads).

Tasks and experiments

For each task, the onset of a tone signaled the beginning of the trial and the offset of the tone signaled the end of the trial (2 s). The following tasks were the basis of the experiments described subsequently:

1. *Task A.* Seven distances "as fast as possible." Subjects were asked to make 2, 5, 8, 11, 22, 33, and 45° flexion (elbow and wrist) or dorsiflexion (ankle) movements as fast as possible to the target; the subject was further instructed to avoid adjusting the final position. The subject practiced each distance several times until they were comfortable with the movement. Blocks of 10–15 trials were recorded for each distance.
2. *Task B.* Seven distances "at a comfortable speed." The task was the same as in task A, except the instruction was to move at a comfortable speed.
3. *Task C.* Seven distances as fast as possible against two different inertial loads. The task was the same as in task A, except movements were only performed at the elbow and were performed against a heavy and a light inertial load.
4. *Task D.* Three speeds. The subject was asked to perform 33° movements, flexion at the wrist or dorsiflexion at the ankle, with three different peak velocities. After each trial the subject was told the actual peak velocity and was instructed to move faster, slower, or at the same speed for the next trial. Thirty-five trials were collected at each speed. All trials outside ±25°/s of the target peak velocity were rejected prior to analysis.

The following task combinations were performed in different experiments:

1. *Experiment 1.* Task A was performed at the wrist, elbow, and ankle in five subjects. The order of the presentation of distances was varied across subjects but was preserved for each joint of a particular subject. This experiment compared the control of distance at all three joints.
2. *Experiment 2.* Task C was performed in five subjects. This experiment tested whether reducing the inertial load at the elbow would shift the kinematic and myoelectric patterns toward those more commonly seen at the wrist.
3. *Experiment 3.* Task D was performed at the wrist (with target peak velocities of 400, 350, and 300°/s) and at the ankle (with target peak velocities of 325, 275, and 225°/s) in four subjects. Task D was not performed at the elbow because data have been presented in a very similar format in previous publications (Corcos et al. 1989; Gottlieb et al. 1989b, 1992). This experiment compared the control of speed at the different joints.
4. *Experiment 4.* Tasks A and B were performed at the elbow in three subjects and at the wrist in four subjects. The order of the presentation of distances was varied across subjects but was preserved for each task performed by a particular subject. This experiment tested the hypothesis that the transition to width modulation of the agonist burst with increasing distance is not due to saturation.

Finally, we estimated the torque necessary to hold the forearm, hand, and foot in the final target positions for each of the seven distances performed during the experiments at the elbow, wrist, and ankle, respectively. We measured the moment arm and the force that was necessary to maintain the joint in the desired position. Force was recorded from a spring scale that was held perpendicular to the manipulandum; the scale was not sensitive enough to detect small forces less than 0.5 N. No measurable torque was needed to hold the forearm or hand in the final positions reached in our experiments. In contrast, at the ankle, although no torque was measured for the shortest displacements, beyond that there was a monotonic increase in the torque with amplitude.

Data analysis

The digitized EMG signals were full-wave rectified. The rectified EMG was filtered with a 25-ms moving average window for plotting EMG time series (Figs. 1, 4–6, 8) and filtered with a 4-ms moving average window for calculating EMG parameters. The baseline EMG was subtracted prior to parameterizing the data. The following parameters were calculated:

1. V_{max}, peak velocity.
2. Distance, actual movement distance. The distance from the initial position to the position 200 ms after the velocity had fallen below 5% of its peak value.
3. Q_{30}, the integral of the agonist EMG signal from the visually marked onset to 30 ms thereafter. This parameter is used to characterize the initial slope of the agonist EMG burst.
4. Q_{ag}, the integral of the agonist EMG from the market onset to the time of peak velocity. This parameter is used to characterize the area of the first agonist EMG burst.
5. Q_{ant}, the integral of the antagonist EMG from the market onset of the agonist to the end of the movement (the distance at which veloc-

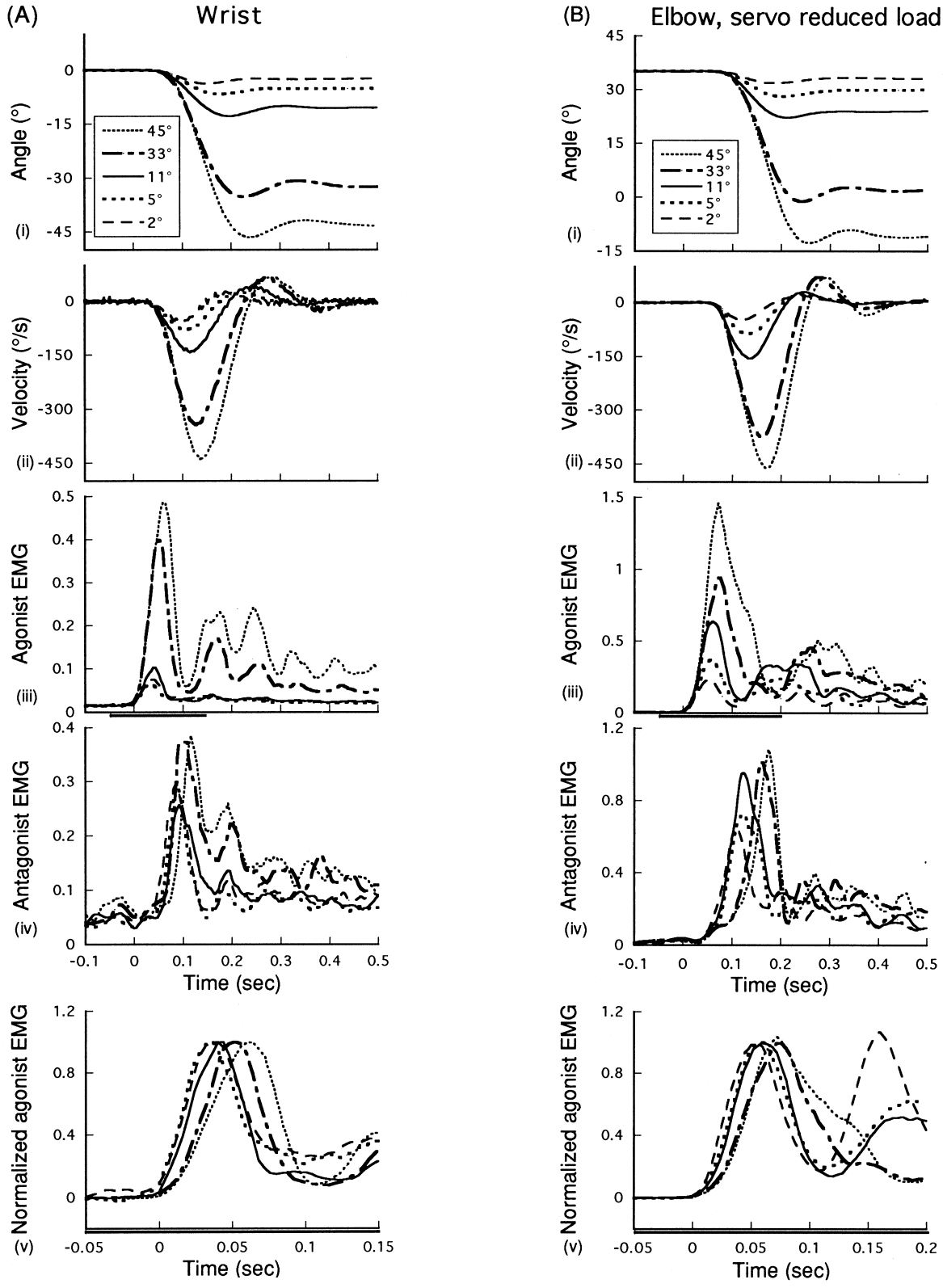

Fig. 1A–D Wrist, elbow, and ankle flexion movements from 2–45°. Averaged angle, (*i*), velocity, (*ii*), agonist EMG, (*iii*), antagonist EMG, (*iv*), and agonist EMG normalized by the peak of the first burst time series, (*v*), are shown for 2° (*large-dashed line*), 5° (*small-dashed line*), 11° (*solid line*), 33° (*dash-dots line*), and 45° (*dotted line*) flexion movements made "as fast as possible to the traget." The data are shown for the wrist (**A**), lightly loaded elbow (**B**), heavily loaded elbow (**C**), and ankle (**D**) (subject 1). Only a subset of the seven distances tested are shown for clarity. The EMGs plotted are from flexor carpi radialis and extensor carpi radialis brevis at the wrist; biceps, and triceps at the elbow; and tibialis anterior and gastrocnemius at the ankle. The normalized agonist EMG is shown on an expanded time scale; the *bold lines under the graphs* of the agonist time series represent the same period of time. Note also that the normalized EMG in **D** is not shown for the 45° movement, because no clear burst was identifiable from which to normalize

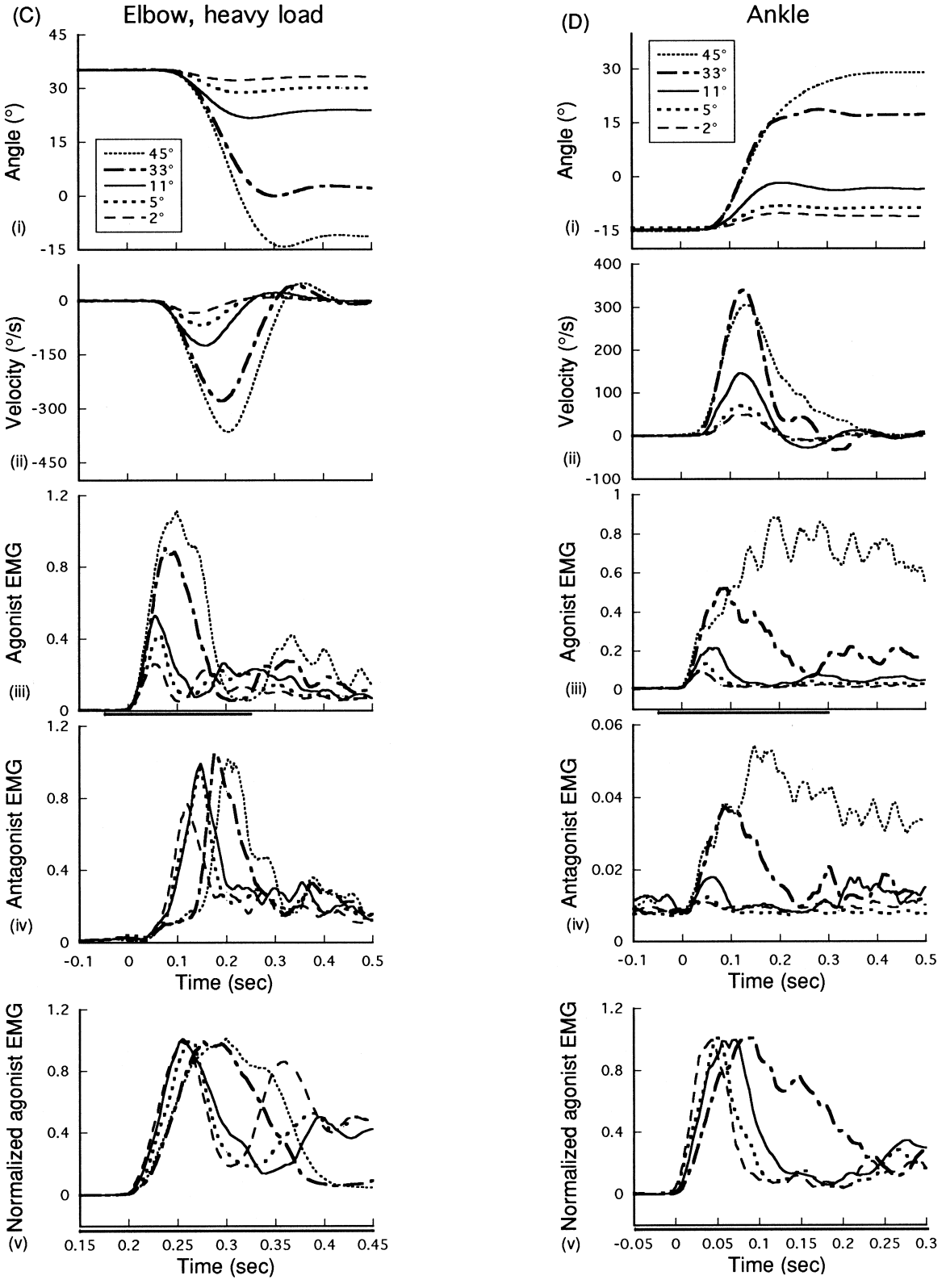
(C)
Elbow, heavy load
(D)
Ankle
Angle (°)
Velocity (°/s)
Agonist EMG
Antagonist EMG
Normalized agonist EMG
45°
33°
11°
5°
2°
(i)
(ii)
(iii)
(iv)
(v)
Time (sec)

Fig. 1C, D

ity drops below 5% of V_{max}). This parameter is used to characterize the area of the antagonist burst.

In addition, net muscle torque at the elbow was calculated by multiplying acceleration by the effective moment of inertia.

Finally, each averaged agonist EMG time series was normalized by its peak value so that the burst durations can be more readily compared (e.g., Figs. 1, 4–6, 8). Note that this process changes the slope of the lines, resulting in a greater decrease in the slope of EMG traces with large peak values as compared to those with smaller peak values. Agonist burst duration was calculated by marking the visually identified beginning and end of the burst on the averaged agonist EMG traces. Table 1 was generated by plotting duration against amplitude and identifying the points that clearly deviated from a near-zero slope line (a zero slope line represents constant burst duration). The first point at which this deviation was observed was identified as the first distance tested at which duration modulation was observed.

Figures 1–8 are taken predominantly from one representative subject to emphasize that the similarities and differences at the three joints are not subject-dependent. One of the nine subjects did not conform to the patterns described, although the subject was consistent with some reports in the literature (see footnote to Table 1).

Results

Effects of movement distance on wrist movements performed "as fast as possible to the target"

Figure 1A shows averaged data from five of the seven distances over which subject 1 performed wrist flexion movements as fast as possible. All movements showed a slight overshoot (Fig. 1A, i). For shorter movements (2°–11°), no changes in the duration of the agonist EMG burst (as shown by the normalized EMG; Fig. 1A, v) or the time to peak of the antagonist burst (Fig. 1A, iv) were observable. For longer movements (33°–45°), the duration of the agonist burst and the time to the peak of the antagonist burst increased.

Effects of movement distance on elbow movements performed as fast as possible to the target

Figure 1B, C shows averaged data from five of the seven distances over which subject 1 performed elbow flexion movements as fast as possible against a light inertial load (Fig. 1B) and against a heavy inertial load (Fig. 1C). The lightly loaded movements (Fig. 1B, i) showed more overshoot than did the heavily loaded movements (Fig. 1C, i). For both sets of elbow flexions, during shorter movements (2°–11°), no changes in the duration of the agonist EMG burst (as shown by the normalized EMG; Fig. 1B, C, v) or time to peak antagonist burst (Fig. 1B, C, iv) were observable. During longer movements (33°–45°), the duration of the agonist burst and the time to antagonist peak increased. The differences between movements made against the two inertial loads will be described in the section entitled Effect of inertial load on the control of distance at the elbow.

Effects of movement distance on ankle movements performed "as fast as possible to the target"

Figure 1D shows averaged data from five of the seven distances over which subject 1 performed ankle dorsiflexion movements as fast as possible. The movements (Fig. 1D, i) were less oscillatory than the wrist or "light elbow" movements. For longer movements, the velocity profile (Fig. 1D, ii) deviated from the bell-shaped curve reported for step movements at most other joints; this deviation occurred when the movements were in the range in which relatively large, steady state elastic forces were readily observed (see Materials and methods). For shorter movements (2°–5°), no changes in the duration of the agonist EMG burst (Fig. 1D, v) or the time to peak of the antagonist burst (Fig. 1D, iv) were observed. For longer movements (11°–45°), the duration of the agonist burst increased and a tonic maintenance activation (Fig. 1D, iii) became prevalent; at the largest distance the burst phase could not be distinguished from the tonic maintenance activation at the end of the movement (Fig. 1D, iii).

Parameters characterizing the data for wrist, elbow, and ankle movements over seven distances

Figure 2 shows parameterized measures for wrist, elbow, and ankle movements made as fast as possible by subject 1. Peak velocity (Fig. 2A–C, i) rose monotonically with distance except for the longest ankle movement (45°), which was near the limit of the range of movement. Q_{30} (Fig. 2A–C, ii), which characterizes the initial slope of the agonist EMG, increased and then reached a plateau at longer distances for wrist and elbow movements. However, Q_{30} continued to increase even for the largest movements at the ankle. Q_{ag} (Fig. 2A–C, iii), the area of the agonist burst, rose monotonically with distance for movements at all three joints. At the wrist and elbow, Q_{ant} (Fig. 2A, B, iv), the area of the antagonist burst, initially increased rapidly with distance; at larger distances, Q_{ant} either rose more slowly, reached a plateau, or decreased (Figs. 2B, iv, 3). Although it is not clear from this data set whether or not Q_{ant} reached a plateau at the wrist, most subjects showed a plateau or slow rise in Q_{ant} for the longer distances; none showed a decrease. At the ankle, Q_{ant} (Fig. 2C, iv) was negligible at the shortest distances and only very small at the longer distances. Figure 3 shows the relationship between Q_{ant} and distance over an extended range of distances for the light and heavy elbow. It shows that the inertial load can transform the shape of curve from one in which Q_{ant} decreased with an increase in distance (at the longer distances) to one in which Q_{ant} plateaus.

Effect of inertial load on the control of distance at the elbow

Figure 4 shows averaged data for elbow movements made as fast as possible over two distances (8°, 22°) and with two inertial loads. Movements performed against the lighter load were faster and more oscillatory than those performed against the heavier load (Fig. 4, i). This demonstrates that reducing the inertial load shifted the kinematic patterns of elbow movements toward those observed during wrist movements. In addition, the duration

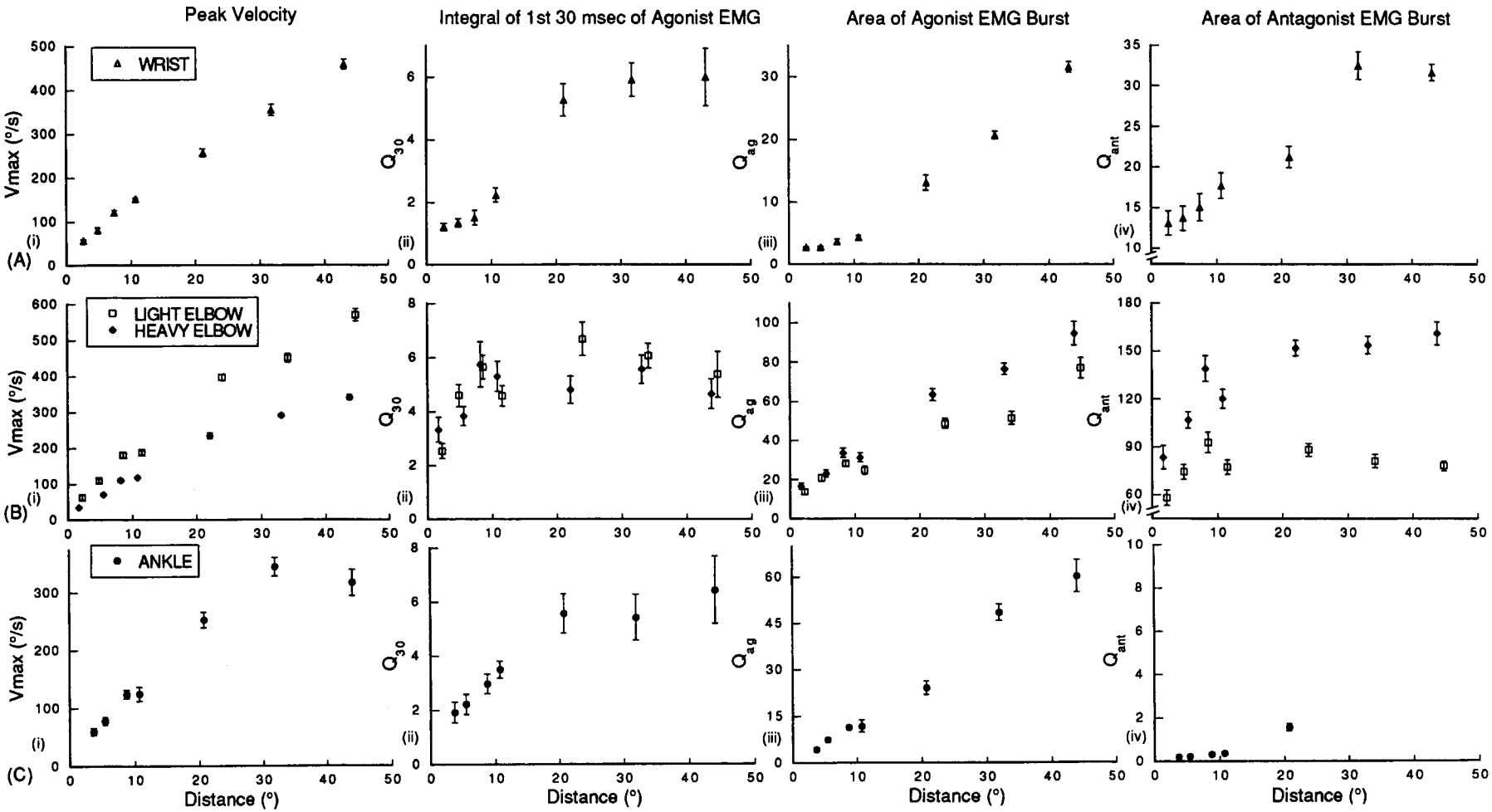

Fig. 2A–C Parameterization of movements made over seven distances at the wrist, elbow, and ankle. Parameters and standard error bars characterizing wrist flexion (**A**; *triangle*), "light" elbow flexion (**B**; *open square*), "heavy" elbow flexion (**B**; *closed diamond*), and ankle dorsiflexion (**C**; *closed circle*) movements made "as fast as possible to the target" over seven distances (2°, 5°, 8°, 11°, 22°, 33°, 45°; subject 1). The following parameters are plotted as a function of the distance of the movement: V_{max} (peak velocity), (*i*); Q_{30} (represents the initial rate of rise of the agonist EMG), (*ii*); Q_{ag} (area of agonist EMG), (*iii*); Q_{ant} (area of antagonist EMG), (*iv*). Q_{ant} is not shown for the two largest ankle movements. The algorithm that calculates Q_{ant} assumes a clear burst profile and uses an integration interval from the agonist onset to the time the velocity profile drops to 5% of V_{max}. Therefore, for the longest distances at the ankle, Q_{ant} corresponds in part to the integration of the tonic maintenance level of antagonist activity due to the deviation from the assumed EMG profile and the assumed velocity profile

of the agonist burst was the same for both movement amplitudes performed against the lighter load and for the 8° movement performed against the heavier load. However, the duration of the agonist burst was increased for the 22° movement against the heavier load (Fig. 4, iii). Therefore, the transition from constant duration to duration modulation occurred at a longer distance when movements were performed against a lighter inertial load than it did for movements against a heavier inertial load.

Figure 5 shows the normalized agonist bursts during movements of several distances with the wrist, light elbow, and heavy elbow. Burst duration was prolonged for the 22° movement only for the heavy elbow but not for the light elbow or wrist. In contrast, burst duration was prolonged for the 33° and 45° movements in all three experimental conditions. Thus, reducing the inertial load shifted the elbow EMG patterns toward those observed at the wrist. Note, however, that the smallest duration of the agonist burst was about 80 ms at the wrist, whereas it was about 100 ms at the elbow. In addition, the minimum burst duration at the elbow was the same for both inertial loads.

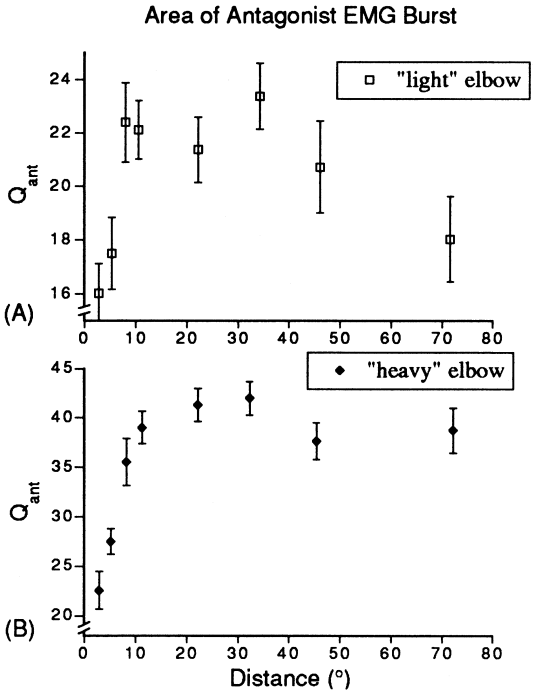

Fig. 3A, B Inertial load can transform the characteristics of the relationship between Q_{ant} and distance. Mean Q_{ant} and standard error bars as a function of distance are shown for subject 3, recorded during "light" (**A**; *open square*) and "heavy" (**B**, *closed diamond*) elbow flexion movements. The range of distances tested was increased to 72° to show a greater range of Q_{ant} behavior. In this subject, at larger distances Q_{ant} decreases with increasing distance for movements made against a light inertial load, whereas Q_{ant} plateaus with increasing distance for movements made against a heavy inertial load

Effect of joint on the transition to duration modulation for control of distance

At a specific joint, the transition to modulation of the duration of the agonist burst occurred at different distances for individual subjects. However, the relative distance at which the transition to duration modulation of the agonist burst was observed varied consistently across joints within each subject (Table 1). Movements at the elbow showed the transition at the same or shorter distances than at the wrist. In addition, if the transition appeared for elbow and wrist movements at the same distance, the observed change in EMG burst duration was greater at the elbow than at the wrist. This may indicate that the transition at the elbow may have occurred at a shorter distance in the range between the distances tested. Moreover, movements at the ankle joint consistently showed duration modulation of the agonist EMG burst at shorter distances than at the elbow. Although the order in which the transition occurred at each joint was predictable when the lightest loads were applied, the manipulation of the inertial load can modify the distance at which the transition occurs; therefore, the consistent pattern of transition at the shortest distances at the ankle, moderate distances at the elbow, and longest distances at the wrist was dependent on loading conditions.

Speed control at the wrist and ankle

Rapid movements of constant distance and external load performed at three speeds are shown in Fig. 6 at the wrist (Fig. 6A; subject 1) and ankle (Fig. 6B; subject 2). Q_{30} (characterizing the slope of the agonist EMG) decreased with decreasing speed at both the wrist and ankle. The duration of the agonist burst was constant at the wrist for the three speeds (Fig. 6A, v). In contrast, the duration of the agonist burst was not clear at the ankle for the three speeds (Fig. 6B, v). It appeared that in general at the wrist, ankle, and elbow joints, as speed decreased, the duration of the agonist EMG burst either remained constant (for the faster movements) or increased (for the slower movements). The same result was observed at the elbow and wrist when movements of the same distance were made as fast as possible and at a comfortable speed. None of the slower movements exhibited shorter burst durations than those generated during faster movements. Regardless of the duration of the agonist burst at the wrist and ankle, Q_{ag} and Q_{ant} (area of the agonist and antagonist bursts) decreased with decreasing speed (Fig. 7).

Transition to duration modulation of the agonist EMG burst

Figures 1 and 5 showed that, as movement distance increased, modulation of the agonist EMG changed from a variable initial EMG slope with a constant duration to a constant EMG slope and variable duration. Presumably, an increase in the initial slope of an EMG burst resulted from the recruitment of additional motor units during this

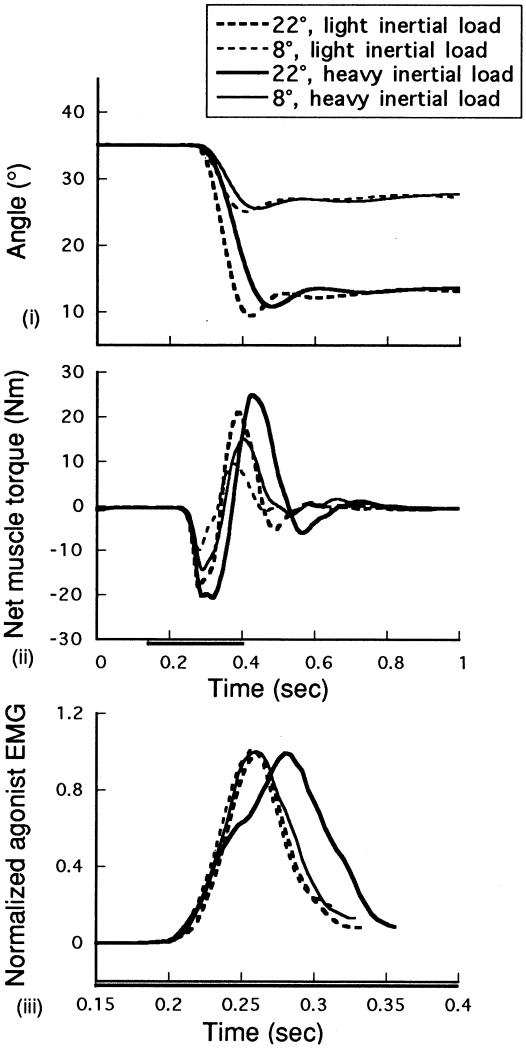

Fig. 4 Transition to duration modulation of the agonist EMG occurs at shorter distances with heavier inertial loads. Averaged angle, (*i*); net muscle torque, (*ii*); and agonist (biceps) EMG normalized by the peak of the first burst, (*iii*), are shown for movements of two distances (8°, *thin lines*; 22°, *thick lines*) with two inertial loads (light load, *dashed line*; heavy load, *solid line*) made "as fast as possible" (subject 1). The normalized agonist EMG is shown on an expanded time scale. The *bold lines underneath the net muscle torque and EMG graphs* represent the same period of time

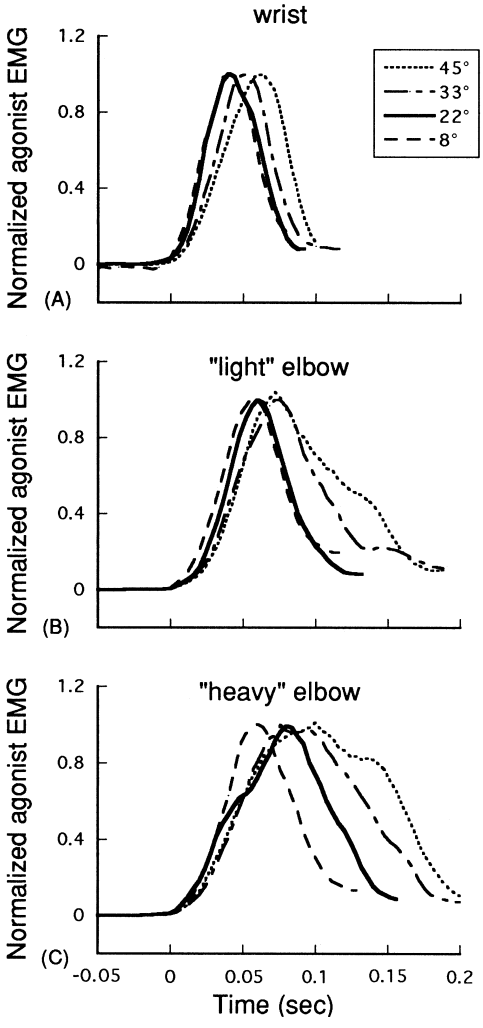

Fig. 5A–C Reduced inertial loading of elbow movements shifts the myoelectric pattern towards that observed at the wrist. The agonist EMG normalized by the peak of the first burst is shown for 8°, 22°, 33°, and 45° movements at the wrist (**A**), "light" elbow (**B**), and "heavy" elbow (**C**)

Table 1 Shortest distance tested at which duration modulation of agonist EMG is observed

	Subject 1	Subject 2	Subject 3	Subject 4	Subject 5[a]
Ankle	8°	22°	33°	11°	22°[a]
"Heavy" elbow	22°	22°	45°	22°	33°[a]
"Light" elbow	33°	33°	None up to 45°	45°	45°[a]
Wrist	33°	45°	None up to 45°	None up to 45°	None up to 45°

[a] Subject 5 did not conform to the patterns most commonly observed. Instead of exhibiting a smooth transition from the narrow burst observed during short movements to increasingly longer bursts as movement distance increases, this subject used a second burst of increasingly greater height typical of the pattern described by Brown and Cooke (1984). The distance marked for the transition is the distance at which the second burst combined with the first. The data in this table were obtained from the five subjects who took part in tasks A and C

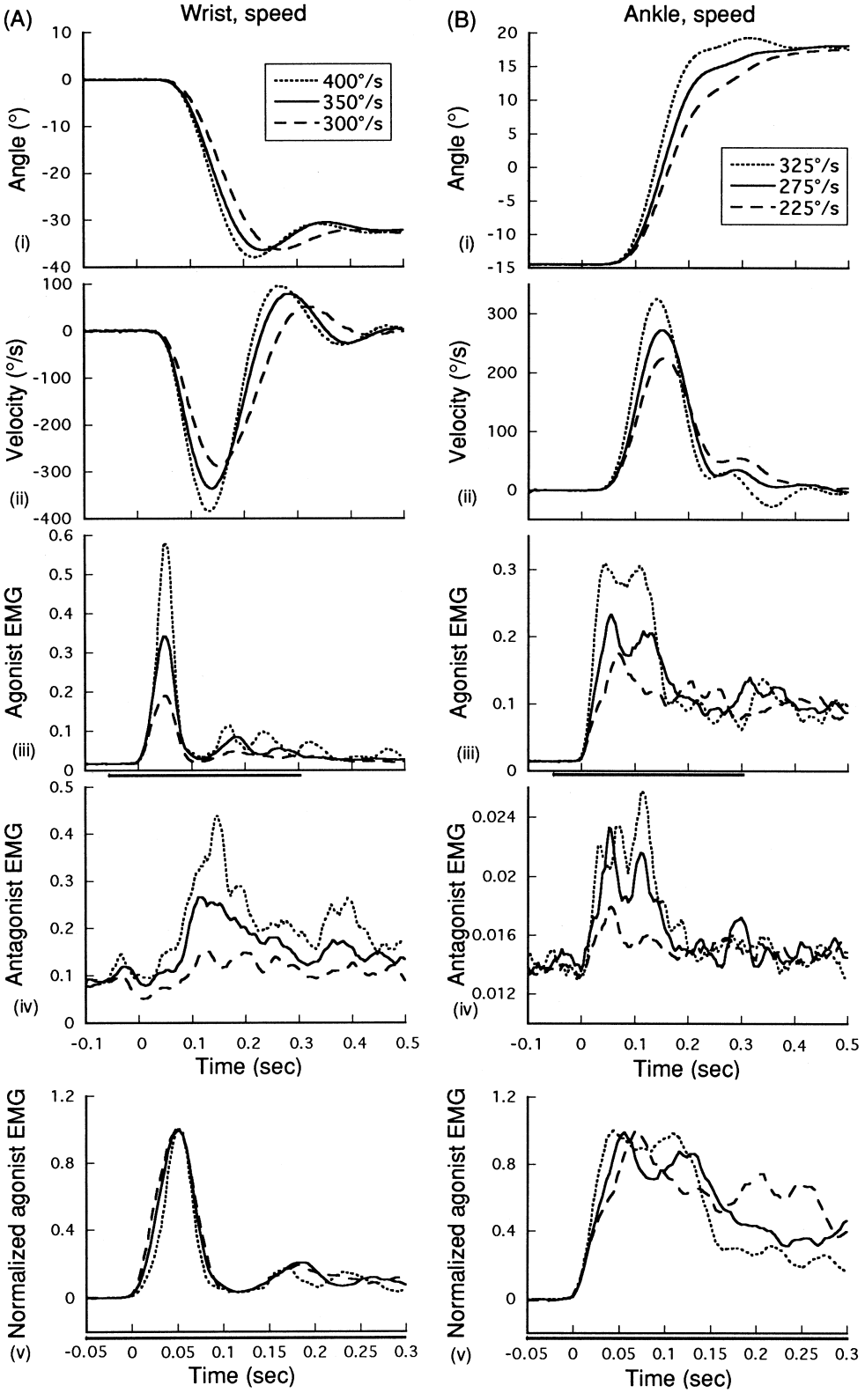

Fig. 6A, B Speed control at the wrist and ankle. **A** The data shown are from 33° wrist flexion movements performed at three different spped: V_{max}=400°/s (*dotted line*), 350°/s (*solid line*), and 300°/s (*dashed line*). Averaged angle, (*i*), velocity, (*ii*), agonist (flexor carpi radialis) EMG, (*iii*), antagonist (extensor carpi radialis) EMG, (*iv*), and agonist EMG normalized by the peak of the first burst, (*v*), time series are shown for each speed (subject 1). **B** The data shown are from 33° ankle dorsiflexion movements performed at three different speeds: V_{max}=325°/s (*dotted line*), 275°/s (*solid line*), and 225°/s (*dashed line*). Averaged angle, (*i*), velocity, (*ii*), agonist (TA) EMG, (*iii*), antagonist (gastrocnemius, lateral head), (*iv*), agonist EMG normalized by peak EMG, (*v*), time series are shown for each speed (subject 2). The *bold lines underneath the graphs* of the agonist EMG represent the same period of time

brief interval. Figure 8 shows that saturation (recruitment of all available motor units) is not necessary for duration modulation to occur. It compares wrist movements performed over two distances under the instructions to move at a comfortable speed and as fast as possible. Under either set of speed instructions, both height (as shown by the agonist EMG; solid lines compared with dotted lines, Fig. 8, iii) and duration modulation of the agonist burst were used in the control of distance (as shown by the normalized agonist EMG; solid lines compared with dotted

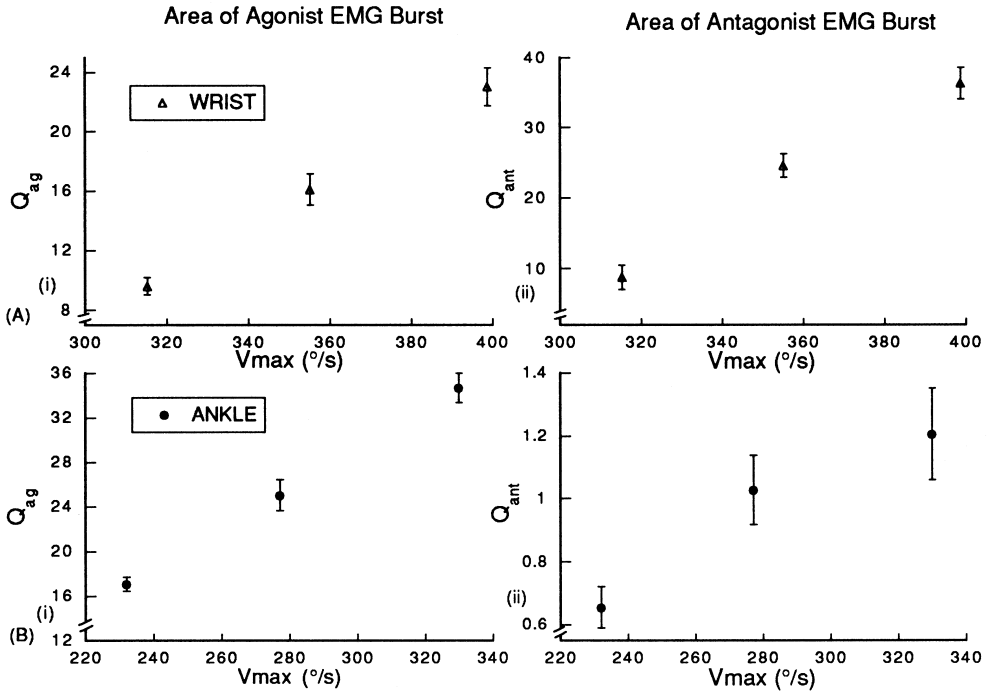

Fig. 7A, B Parameterization of speed control experiments at wrist and ankle. Q_{ag} (area of the agonist burst), (*i*), and Q_{ant} (area of the antagonist burst), (*ii*), are plotted as a function of V_{max} for 33° wrist flexion (**A**; *triangle*; subject 1) and ankle dorsiflexion (**B**; *closed circle*; subject 2)

lines, Fig. 8, v). However the agonist EMGs of movements performed at a comfortable speed both rose at a slower rate than the agonist EMGs of movements performed as fast as possible (shown by the agonist EMG; thin lines compared with thick lines, Fig. 8, iii). Since the slope of the agonist burst for the 33° movement performed at a comfortable speed was less than that for the 8° movement performed as fast as possible, saturation of the motoneuron pool did not occur under the comfortable speed condition even though duration modulation was observed. Therefore, in the comfortable speed condition, modulation of the agonist burst resulted from the selection of motoneuron pool activation patterns rather than from saturation or full recruitment.

Discussion

This study was motivated by seemingly contradictory findings between experiments on the wrist and elbow. Based predominantly on studies at the elbow, Gottlieb and colleagues (1989b) proposed that strategies are used to plan movements, and they identified a set of rules associated with each of two strategies. They directly associated the speed-insensitive and speed-sensitive strategies with pulse-duration and pulse-height modulation, respectively, of the motoneuron pool innervating the agonist muscle. They also described rules for the associated modulation of the antagonist muscle activation. However, Hoffman and Strick (1989) reported that, at the wrist, a task identified by Gottlieb and colleagues as speed-insensitive was associated with muscle activation patterns characteristic of the speed-sensitive strategy. Consequently, Hoffman and Strick suggested that EMG patterns are based on the force requirements of a task rather than on the "speed sensitivity" of a task. Further work by Gottlieb and colleagues, in which they studied elbow movements over short distances, confirmed the finding of Hoffman and Strick that the strategies cannot be directly associated with unique patterns of muscle activation (Gottlieb 1993; Gottlieb et al. 1996a). In addition, Hoffman and Strick confirmed the earlier findings of Gottlieb and colleagues that pulse-duration modulation may occur even when the force requirements of a task do not exceed those that can be generated by pulse-height modulation (Hoffman and Strick 1993). Although these results brought the authors closer together in their views, some questions remained to be clarified by this study.

In this study, we compared rapid, single degree-of-freedom movements across different joints within individual subjects. We have four important findings. First, the agonist bursts associated with rapid movements of increasing distance at the wrist, elbow, and ankle were all modulated in height (pulse-height modulation) for small movements and in duration (pulse-duration modulation) for longer movements. The relative distance at which the transition to duration modulation of the agonist burst was observed varied consistently across joints in all subjects (Fig. 1, Table 1). Second, reducing the inertial load of the arm shifted the kinematic and myoelectric patterns toward those observed at the wrist (Figs. 1, 4, 5). Third, for fast elbow flexion movements, the area of the antagonist burst increased with movement amplitude for small movements and then decreased with movement amplitude for larger movements. Changes in the inertial load transformed the relationship between area of the antagonist burst and movement distance (Fig. 3). Finally, we confirmed and more clearly demonstrated that modulation

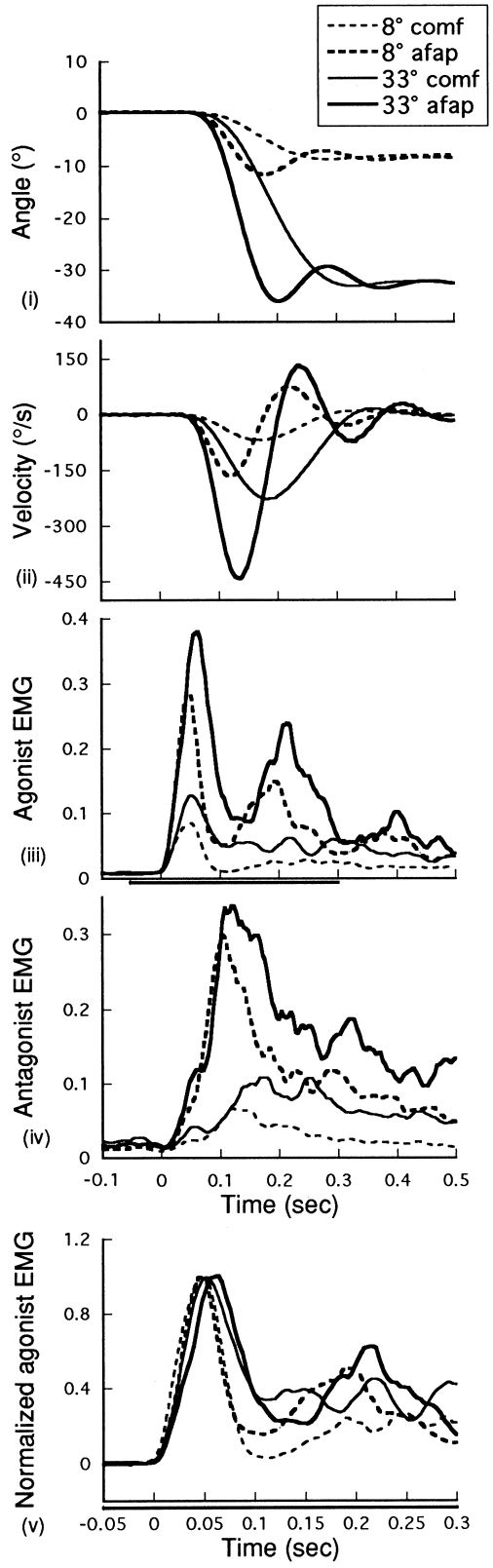

Fig. 8 Transition to duration modulation of the agonist EMG occurs at sub-maximal levels of activation. Averaged angle, (*i*), velocity, (*ii*), agonist (flexor carpi radialis) EMG, (*iii*), antagonist (extensor carpi radialis) EMG, (*iv*), and agonist EMG normalized by the peak of the first burst, (*v*), are shown for 8° (*dashed line*) and 33° (*solid line*) wrist flexion movements made "as fast as possible to the target" (*thick lines*) and at "a comfortable speed" (*thin lines*; subject 2)

of the duration of the agonist burst is not simply a saturation phenomenon (Fig. 8). These data are all consistent with the hypothesis that a single control scheme can be applied at all three joints (Cheron and Godaux 1986; Godaux 1989; Gottlieb et al. 1989a; Hoffman and Strick 1989, 1993). According to this hypothesis, the differences in kinematic and myoelectric patterns observed at the wrist, elbow, and ankle may be explained by the biomechanical constraints of each joint.

The same rules are used to control distance at the wrist and elbow

Agonist

Based on this study and numerous prior ones, it now seems clear that movements performed as fast as possible over shorter distances are associated with agonist EMG bursts of constant duration and with slopes and amplitudes that increase with movement distance. Similar movements over increasingly longer distances are associated with duration modulation of the agonist burst with constant slopes. Consequently, our results are consistent with those of numerous prior studies, many of which reported only fixed duration of the agonist burst or only duration modulation of the agonist burst (thumb: Hallett and Marsden 1979; wrist: Berardelli et al. 1986, 1984; Hoffman and Strick 1990; Mustard and Lee 1987; elbow: Benecke et al. 1985; Berardelli et al. 1984; Brown and Cooke 1984, 1981; Cheron and Godaux 1986; Corcos et al. 1988; Gottlieb et al. 1996a; Wadman et al. 1979). It is also clear that, for both short and long movements, the area of the agonist burst rises monotonically with distance (thumb: Marsden et al. 1983; wrist; Hoffman et al. 1990; Mustard and Lee 1987; elbow: Benecke et al. 1985; Gottlieb et al. 1989a; Marsden et al. 1983).

Antagonist

This study has also confirmed prior ones regarding the timing of the antagonist burst during single-joint movements made as fast as possible (Benecke et al. 1985; Cheron and Godaux 1986; Gottlieb et al. 1989a; Hoffman and Strick 1993; Marsden et al. 1983; Mustard and Lee 1987). We and others found that the timing of the antagonist burst activity is nearly constant for short movements and is increasingly delayed for longer movements. A delay in the timing of the antagonist burst is observed when the duration of the agonist burst increases (Gottlieb et al. 1992).

Although there is considerable confusion in the literature regarding the modulation of the area of the antagonist EMG burst with respect to movement distance, we believe that we can provide a single framework for understanding this relationship. Our results clearly demonstrated that, during elbow movements against a light inertial load, the area of the antagonist burst initially in-

creased and then decreased with distance (Fig. 3). We suggest that a decline in the area of the antagonist EMG occurs when viscoelastic forces and/or gravity contribute a relatively large portion of the braking force (Cheron and Godaux 1986; Gottlieb et al. 1992; Lestienne 1979). For wrist movements, the area of the antagonist burst does not decrease with distance (Fig. 2B in this paper; Hoffman and Strick 1990; Mustard and Lee 1987) presumably because the nonlinear viscoelastic properties of the lengthening antagonist muscle do not provide sufficient force to brake large wrist movements. In contrast, fast elbow flexions in the vertical plane are associated with a decrease in antagonist EMG area with distance over the whole range (Cheron and Godaux 1986). In this case, gravity aids in decelerating the movement, thereby reducing the required antagonist activation. Our results also show that the relationship between antagonist area and distance can be transformed by varying the experimental conditions (e.g., inertial load; Benecke et al. 1985; Fig. 3 in this paper). For example, the addition of an inertial load shifted a negative relationship between longer distances and antagonist EMG area into a plateau or positive relationship. Our explanation for this finding is that, since a larger inertial load requires a larger braking force, it was necessary to increase the active braking force provided by the antagonist. Thus, we suggest that the amplitude of the antagonist burst is matched to the required active braking force.

The same rules are used to control distance at the ankle as are used at the wrist and elbow

Agonist

The initial modulation of the ankle agonist (TA) with increasing distance was similar to that observed at the wrist and elbow in the horizontal and sagittal planes (Berardelli et al. 1984; Cheron and Godaux 1986; Gottlieb et al. 1996a; Hoffman and Strick 1989). However, at larger distances, the initial slope and duration of the agonist EMG both increased and a tonic maintenance EMG became apparent.

To explain these differences, we must consider the effects of passive mechanical constraints. Because the maximum torque due to gravity occurred when the ankle was at 0° and the aforementioned differences were greatest when the ankle was at 30°, gravity probably does not play a major role in either the prolonged agonist burst or the tonic maintenance agonist EMG. Rather, the need to overcome the passive mechanical constraints of the ankle joint and musculature with greater dorsiflexion is a more likely explanation for the prolonged agonist bursts and the tonic maintenance EMG seen with larger movements. We found that the torque required to hold the ankle at the final position increased with the amount of dorsiflexion (see Materials and methods). This is not surprising, because the final position for the 45° movements (30°) was near the limits of the range of motion at the ankle (Kapandji 1982; Kendall and McCreary 1983). In fact, our data are similar to those on rapid wrist movements performed against linear viscoelastic loads (Hoffman and Strick 1993).

Antagonist

For the smallest movement, the antagonist activity was extremely low. With increasing distance, the antagonist became more active, but the activity was always early and small. Presumably this occurred because gravity and the viscoelastic properties of the joint and musculature generated enough force to stop the movement (Cheron and Godaux 1986; Gottlieb et al. 1992; Lestienne 1979). Antagonist activity may correspond to the low-level, early onset activity often observed at the elbow. The role of this antagonist activity may be to counteract the centrifugal force that may tend to separate the joint surfaces (Karst and Hasan 1987). In addition, the slow contraction time of the soleus implies that the antagonist force may be slower to develop and decay, thereby obviating the need for delaying the antagonist burst.

The same rules are used to explicitly control speed at the wrist, ankle, and elbow

For rapid movements of fixed distance and external load, decreasing movement speed is associated with a reduction in slope of the agonist EMG (wrist: Hoffman and Strick 1990; Mustard and Lee 1987; our Fig. 6A; elbow: Corcos et al. 1989; Gottlieb et al. 1992 and ankle: our Fig. 6B). The duration of the agonist burst is constant with small reductions in movement speed but is increased for further reductions in movement speed. The apparent contradiction among studies that report either a constant duration or an increase in duration of the agonist burst as speed slows (e.g., Corcos et al. 1989; our Figs. 6A, 8; Mustard and Lee 1987; our Fig. 6B) may be accounted for by the range of speeds examined relative to the fastest speeds at the joint studied. In all cases, the area of both the agonist and antagonist EMG bursts declined with decreasing speed (wrist: Mustard and Lee 1987; elbow: Corcos et al. 1989; Gottlieb et al. 1992; Marsden et al. 1983; thumb: Marsden et al. 1983; ankle: our Fig. 7B). There is some discrepancy, however, with regard to the timing of the antagonist burst. Decreasing movement speed can be associated with an increasing delay of the antagonist onset (wrist: Mustard and Lee 1987; elbow: Corcos et al. 1989; Gottlieb et al. 1992; Lestienne 1979; Marsden et al. 1983; thumb: Marsden et al. 1983), or with no change in timing (wrist: Hoffman and Strick 1990; our Fig. 6). Further research is required to clarify precisely how agonist duration and latency scale with speed.

Transition to duration modulation of the agonist EMG burst

Why is there a transition from slope modulation with constant duration (pulse-height) to constant slope with duration modulation (pulse-duration) of the agonist EMG burst? We will consider four potential contributing factors: (1) contraction time, (2) saturation of agonist recruitment, (3) saturation of antagonist recrutment, and (4) systematic modification of the pattern used at maximal effort.

When studying movements made "as fast as possible," why do we not we observe an agonist EMG pattern of maximum height, with pulse duration modulation beginning at zero duration and increasing with distance? This would seem to be the appropriate input for movements "as fast as possible." However, brief movements that have an acceleration time comparable with twitch pulse time may not be effectively modulated by the duration of the EMG burst, because the duration of force cannot be made any shorter than the twitch time. For such movements, a smaller pulse height may be necessary to reduce the force generated to an appropriate level (Gottlieb 1993). For example, biceps and TA have similar contraction times (mean twitch contraction of small bundles of fibers for biceps, 52 ms; TA, 58 ms (Buchthal and Schmalbruch 1969)). Therefore, we would expect similar minimum EMG burst durations for both muscles, which is consistent with our data.

Given this minimum EMG burst duration, Hoffman and Strick proposed that "when force output cannot be augmented by further pulse-height modulation of the agonist burst, then additional force is generated by pulse duration modulation" (Hoffman and Strick 1993, p. 5213). This notion of saturation, suggested by several previous authors (Benecke et al. 1985; Berardelli et al. 1984; Cheron and Godaux 1986), is appealing because it is consistent with the idea that the control patterns are critically related to the biomechanical constraints of the joint and the load. For example, the stronger the agonist muscle in a particular subject, the more force that can be developed with maximum recruitment, so the longer the movement at which one expects to first observe pulse duration modulation. Consistent with this, Hoffman and Strick (1993) showed that a weak subject used duration modulation and a strong subject used height modulation for movements over the same range of distances.

There are, however, certain situations in which a transition based on saturation of agonist motor unit recruitment could cause problems. For example, if the antagonist muscle is weak, it may not be able to generate enough force to stop the movement at the desired target. The system could compensate for a weaker antagonist by switching to pulse duration modulation of the agonist at lower levels of activation to avoid overpowering a weak antagonist. This switch changes the timing of the force development and kinematics, which could allow the system to take advantage of muscle properties to stop the movement (Lestienne 1979; Wierzbicka and Wiegner 1992).

Even a balance between saturation of the agonist and of the antagonist cannot explain the transition from pulse height to pulse duration that occurs when movements are made at submaximal efforts (Fig. 8; Gottlieb et al. 1990; Hoffman and Strick 1993). We suggest that the transition from pulse-height to pulse-duration modulation of the agonist activation is determined by the characteristics of the muscle groups at maximal effort (e.g., strength, twitch characteristics) and that movements made at submaximal effort are performed by systematic modifications of the pattern used at maximal effort, e.g., the slopes of the agonist and antagonist EMGs are reduced to reduce speed. Such a systematic adjustment would allow the system to modify an existing control pattern to control speed rather than having to generate an entirely independent pattern.

EMG patterns and the biomechanical characteristics of different joints

In this section, we will highlight several biomechanical factors including agonist compared with antagonist muscle strength, agonist strength compared with load, and other mechanical properties of muscle that we believe underlie the differences in the EMG patterns observed at the elbow, wrist, and ankle. In addition, range of motion and muscle twitch contraction time have already been discussed as important factors. Note that although many factors influence the biomechanical characteristics of a joint, we believe that predictable changes in EMG patterns result from changes in a given factor.

For the movements tested in this study, the mean ratio of agonist (flexor or dorsiflexor) strength to antagonist (extensor or plantarflexor) strength, as determined by the torques generated in maximum voluntary isometric contractions, are about 2.0–2.1 at the elbow (Colebatch and Gandevia 1989), 1.25–1.3 at the wrist (Colebatch and Gandevia 1989), and 0.44–0.6 at the ankle (Sepic et al. 1986). If a relatively weaker antagonist necessitates an earlier transition to duration modulation, elbow flexion movements should be most affected by this factor. This hypothesis would best be tested directly by preferentially strengthening the triceps over the elbow flexors and examining whether the transition to duration modulation occurs at a longer distance. In contrast, a similar strengthening paradigm at the ankle should have no effect.

The ratio of elbow (flexed 90°) to wrist agonist strength is about 5.5 (Colebatch and Gandevia 1989) and the ratio of elbow to ankle agonist strength is about 0.9 (Colebatch and Gandevia 1989; Sepic et al. 1986). (Note that the elbow to ankle ratio was done on different subject groups, so the comprison may not be as reliable as the elbow to wrist ratio.) With all other factors the same, the transition to duration modulation at the elbow would be expected when torques exceed 5.5 times those generated during wrist movements at the point of transition to duration modulation. The torques generated during elbow flexion movements are about 10 times those generated during wrist movements of the same extent (from the ini-

tial positions tested in this study). Therefore, we would expect that the transition to duration modulation would be seen at shorter distances during elbow flexion movements than during wrist flexion movements. Although the transition to duration modulation often occurred at shorter distances at the elbow than at the wrist, the transition at the elbow occurred at somewhat larger torques than expected based on this one factor alone.

The individual muscle properties and their mechanical positions in the body must also be considered. For example, muscle fiber length affects the range over which significant forces can be applied. Both the length-tension properties of the muscles and the effective moment arms vary with position and affect the torque generated. To compare the combined effects of these static properties, we would need to measure the maximum voluntary isometric contractions over the whole range of the movements studied. Even then, a simple interpretation would probably not be possible. Another factor that is more difficult to measure is the dynamic viscoelastic properties of the muscles. Consequently, many factors combine to yield the biomechanical characteristics of a joint.

In spite of the difficulty in separately identifying the effect of each mechanical constraint, we can experimentally manipulate the loading conditions to test that the observed changes in the kinematic and myoelectric patterns are in the expected direction. As discussed by Cheron and Godaux (1986) and further discussed and shown by Hoffman and Strick (1993), changing the loading conditions should result in predictable changes. For example, since the moment of inertia at the elbow is about 15 times that at the wrist while flexor strength at the elbow is only 5.5 times that at the wrist, reducing the moment of inertia at the elbow should shift the mechanical characteristics toward those observed at the wrist. As expected, the kinematic and EMG patterns observed during elbow flexion movements with the reduced moment of inertia were shifted toward those observed in wrist movements as compared to elbow movements with the larger moment of inertia.

Utility of the terms “speed-sensitive” and “speed-insensitive”

Since modulation of the initial slope of the agonist EMG is sometimes used for the control of movement distance (i.e., at short distances) and can no longer be exclusively attributed to the control of speed, do the terms “speed-sensitive” and “speed-intensitive” provide insight or add confusion to the study of motor control (Hoffman and Strick 1993)? The answer depends on the level of analysis at which one is trying understand the control of movement. One level relates to defining the factors that differentiate movement tasks. One of the important advances in the study of motor control has been the recognition that the control of movement is exquisitely sensitive to very specific task demands and that, as a result, movements are not all controlled the same way. For example, moving a fixed distance with a load places different demands on the central nervous system from moving a fixed distance with constraints placed on movement accuracy or movement speed. At this level, the terms speed-insensitive and speed-sensitive provide insight because they reflect fundamentally different constraints on the central nervous system within which the controller must function.

Another level of analysis relates to understanding the EMG patterns that accompany movement. Originally, the terms speed-insensitive and speed-sensitive were used to distinguish how different kinematic tasks led directly to different motoneuron pool modulation schemes. However, the terms are no longer useful as descriptors of motoneuron pool modulation. The utility of the terms is only in bringing to the foreground that the way in which muscles are used is critically dependent on the task.

Single-joint movement studies in the general context of motor control

Although in this paper we only studied movements constrained to 1 degree of freedom, we believe the results yield insights into the control of movement that can generalize to other classes of movements. First, although we completely constrained the path, we did not provide support for the surrounding joints. Therefore, it was still necessary for the nervous system to appropriately activate the musculature at other joints. In addition, the modulation of the EMG patterns observed in single degree-of-freedom elbow flexion movements has been shown to generalize to multijoint pointing movements over different distances and speeds in which the elbow musculature was a primary mover (Almeida et al. 1995; Gottlieb et al. 1996b, 1996c). Moreover, for 2 degree-of-freedom (elbow and shoulder) planar limb movements over different distances, speeds or loads in the same direction, torques at the elbow and shoulder are linearly related and the EMG patterns at each joint are modulated in a similar manner as in 1 degree-of-freedom movements (Gottlieb et al. 1996c). Consequently, we view our study as a particular case of the class of constrained movements, which the nervous system deals with using the same strategies that it uses for unconstrained movements. As such, they yield insight into how a simple motor program may be used at different joints by adjusting the parameters to the force requirements of the task.

The application of a simple motor program to different joints is consistent with the known physiology and anatomy of many nervous system structures. The preponderance of topographical maps within nuclei related to motor control (e.g., motor cortex, premotor areas, ventrolateral thalamus, basal ganglia, and cerebellum) and topographically mapped projections to motor-related nuclei provide a structure in which similar neural processing can be directed toward and tuned for specific joints. Two brain structures that have been specifically associated with the generation the parameters of the triphasic burst EMG pattern discussed in this paper are the cerebellum and the basal ganglia. The cerebellum has been implicated in con-

trolling the relative timing of the bursts; disorders of the cerebellum are associated with inappropriate delays in the onsets of the antagonist burst and the second agonist burst at the elbow, wrist, and fingers (Hore et al. 1991; Wild and Corcos 1997). The basal ganglia has been implicated in the scaling of the first agonist burst; people with Parkinson's disease (a basal ganglia disorder) fail to scale height and duration appropriately for a given task (Berardelli et al. 1986; Hallett et al. 1977). However, the details of the motor circuitry involved in selecting the motor program and in defining the specific parameters for a particular task are still unknown even in the simple tasks examined in this study.

Summary of EMG patterns during rapid, single degree-of-freedom movements at different joints

Consider a hypothetical supported joint being rotated in the horizontal plane in a range in which the passive viscoelastic properties are relatively small. We suggest that the agonist EMG modulation with respect to distance can be described as follows: for movements made "as fast as possible," at first the initial slope of the agonist EMG increases while maintaining a constant burst duration. At a critical point, which we believe is related to the force twitch time, the force developed in the agonist by motor-unit summation and the ability of the antagonist to brake the movement, the agonist pattern transitions to a constant slope with increasing duration. Modifying the load affects the distance at which the critical point is reached, e.g., if the inertial load is increased then the transition will occur at a shorter distance, presumably corresponding to the development of the same levels of force during the minimal pulse duration time. Although our data of elbow flexion movements with different inertial loads is consistent with this, there was not enough resolution in the movements studied to conclusively test this. If the muscles are strengthened, then we predict that the transition would occur at a longer distance, because more force could be developed by the agonist during the minimal pulse duration time and more force could be generated by the antagonist to stop the movement. Although the muscle properties and dynamics during maximal effort may determine the critical point of transition from pulse-height to pulse-duration modulation, the transition will occur even though critical forces are not reached (i.e., it is not a force saturation phenomenon). For example, as shown in Fig. 8, the transition from pulse height to pulse duration occurs even though the forces involved are much lower during slower movements of similar distances.

Similarly, we suggest the modulation of the antagonist EMG can be described as follows: under maximal effort, the peak antagonist burst occurs at the same time for short movements and increasingly later for longer movements. The delay begins at the point at which the modulation of the agonist burst transitions to duration modulation (point of transition). For small movements, there may be no antagonist burst at all, if the passive viscoelastic forces and external forces (e.g., gravity) are large enough to brake the movement. The area of the antagonist burst with respect to distance initially rises and then may plateau or even decrease. If the force needed to brake the movement increases for a specific movement distance (e.g., by increasing inertial load), the area of the antagonist increases and the burst will occur later if the point of transition has been surpassed. For movements made with reduced effort (e.g., at a comfortable speed), the slope of the antagonist burst and the antagonist area will decrease relative to those during a similar movement made at maximal effort.

We conclude that a common set of control rules is used at different joints to generate the observed EMG patterns and that a combination of biomechanical factors including muscle properties, strength of the muscle groups, viscoelastic properties of the joint, and external loading conditions determine the control pattern used to perform a particular rapid, single-joint movement task. Moreover, changes in these biomechanical constraints affect the control patterns in predictable ways. This allows for experimental manipulations that shift EMG patterns toward those more commonly observed at other joints.

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