

Distance and Movement Time Effects on the Timing of Agonist and Antagonist Muscles: A Test of the Impulse-Timing Theory

Stephen A. Wallace

Department of Physical Education
Institute of Cognitive Science
University of Colorado at Boulder

Laurel Wright

Department of Physical Education
University of California at Davis

ABSTRACT. The experiment examined the effects of movement time (MT) and distance on the timing of electromyographic (EMG) activity from an agonist and antagonist muscle during rapid, discrete elbow movements in the horizontal plane. According to impulse-timing theory (Wallace, 1981) MT, not distance moved, should have a pronounced effect on the timing of EMG activity (duration of initial agonist and antagonist burst and time to onset of initial antagonist burst). The levels of MT were 100 and 160 msec and the levels of distance were 27° and 45° of elbow flexion. In general support of impulse-timing theory, the results of the three EMG timing measures showed that MT had a more pronounced effect on these measures than distance. In addition, the timing of EMG activity in relation to total MT remained fairly consistent across the four MT-distance conditions.

SURROUNDING JOINTS in the human, there exist sets of muscles whose contractions must be coordinated to produce efficient movement. Agonist muscles initiate a given limb movement while antagonist muscles help brake the movement (Basmajian, 1962). Through the use of electromyographic (EMG) recordings, many investigators have sought to understand the principles by which agonist and antagonist muscular activity is regulated during the production of movement (e.g., Angel, 1974, 1975a, 1975b, 1977; Barnett & Harding, 1955; Basmajian & Latif, 1957; Bouisset, Lestienne, & Maton, 1977; Garland, Angel, & Moore, 1972; Hallett, Shahani, & Young, 1975; Lestienne, 1979; Wadman, Denier van der Gon, Genze, & Mol, 1979; Wadman, Denier van der Gon, & Derkson, 1980).

Reprint requests should be addressed to Stephen A. Wallace, Institute of Cognitive Science, Department of Physical Education, Box 354, University of Colorado, Boulder, Colorado 80309.

One general characteristic of agonist and antagonist muscles is that they seem to be reciprocally activated during the production of a rapid limb movement, a discovery made many years ago by Wachholder (Wachholder, 1928; Wachholder & Altenburger, 1926). An initial accelerative burst in the agonist muscle is followed in many instances by a silent period in this muscle during which the antagonist muscle is activated to brake the movement. Often, an additional burst is seen in the agonist muscle following the antagonist burst—however, the function of this burst is less understood (Garland & Angel, 1971). This "three burst" or "tri-phasic" activity in the agonist and antagonist muscles has been observed several times (e.g., Angel, 1974; Lestienne, 1979; Wadman et al., 1979, 1980).

One controversy existing today is how the magnitude and timing of the agonist and antagonist muscle bursts are regulated depending on the physical characteristics of the movement. Magnitude refers to the amplitude of the EMG signal which is believed to reflect changes in the number and firing rates of the motor units in the monitored muscle (Basmajian, 1962). Timing is meant here to refer to the "on-off" characteristics of the individual bursts in the agonist and antagonist muscle. Various researchers have proposed that to increase, say, the velocity of a movement requires an increase in the magnitude of agonist and antagonist muscle bursts (to increase accelerative and decelerative forces, respectively) without changes in the basic timing characteristics of these muscles (Freund & Budingen, 1978; Hallett, 1979). Others have proposed that the timing characteristics of the agonist and antagonist muscles are directly proportional to the distance of the movement (Wadman et al., 1979, 1980). Barnett and Harding (1955) concluded from their work that the onset of the initial antagonist burst is triggered at a fixed joint angle regardless of the movement speed. Thus, while a considerable amount of EMG studies have been conducted on agonist-antagonist muscle control, there does not seem to be a consensus on how these muscles are regulated in movements of different times and distances.

In a recent critical review of the literature, Wallace (1981) developed an impulse-timing theory which attempts to predict the magnitude and timing of agonist-antagonist muscle activity based on knowledge about the physical parameters of the movement, such as movement time and distance. The impulse-timing theory states that both the magnitude and timing of agonist and antagonist muscular activity are regulated during the production of rapid, discrete movements. Based on kinematic work by Schmidt, Zelaznik, Hawkins, Frank, and Quinn (1979), Wallace argued that the total movement time determines how the accelerative and decelerative forces are distributed during a movement. More specifically, the duration of the initial accelerative force and the onset of initial decelerative force should be proportional to the total movement time. Wallace hypothesized that since the agonist and antagonist muscles contribute in a major way to the accelerative and decelerative forces, respectively, the timing of their activity should also be a function

of the movement time and distance. The magnitude of limb movement decelerative forces is determined by the underlying muscle activity. The magnitude of the initial agonist burst and the duration of the silent period remain constant because the cessation of the initial decelerative force is required in all hypothetical situations. A re-examination of the data for these postures (Wadman et al., 1978; Hallett, 1979) is limited, however, to time and distance.

Thus, the present experiment was designed to determine the impulse-timing characteristics of the agonist (biceps) and antagonist (triceps) muscles during the production of rapid elbow movements. Unfortunately, our experimental design did not allow for a direct measurement of the timing of the initial decelerative force.

Subjects were recruited from the University of Illinois at Urbana-Champaign and were paid for their participation.

Apparatus. The experimental apparatus consisted of a motor-driven apparatus which rotated the subject's forearm through a range of motion of 90° and 9 cm. The subject's forearm was supported by a rubber band which was attached to a support structure. The subject's forearm was positioned in a horizontal position and the motor was mounted on a vertical support structure. The motor was used to rotate the forearm.

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of the movement time. Furthermore, Wallace argued that the distance of limb movement has no effect on the timing of accelerative and decelerative forces and thus should not influence the timing of the underlying muscular activity. Finally, the ratio of the duration of the initial agonist burst to the total movement time and the ratio of the onset and duration of the antagonist burst to the total movement time should remain constant over virtually all movement conditions. This is so because the cessation of the initial accelerative force and onset of the initial decelerative force occurs halfway through the total movement time in all hypothetical cases examined by Schmidt et al. (1979). In an examination of the literature, Wallace (1981) found supporting evidence for these postulates (Barnett & Harding, 1955; Freund & Budingen, 1978; Hallett & Khoshbin, 1980; Lestienne, 1979). This evidence was limited, however, because in these studies the variables of movement time and distance were not carefully controlled.

Thus, the present study was designed to manipulate the variables of movement time and distance to test directly some of the postulates of the impulse-timing theory developed by Wallace (1981). In the present experiment, we monitored and recorded the timing behavior of an agonist (biceps) and an antagonist (triceps) muscle during the production of rapid elbow flexion movements in the horizontal plane. Unfortunately, our experimental set-up did not permit us to analyze EMG information regarding the magnitude of the underlying muscular activity. However, we felt the timing information would provide sufficient data to allow for a test of the impulse-timing theory.

Method

Subjects. Ten male right-hand volunteers (23–33 years of age) from the University of California at Davis participated in this study. They were not paid for their services.

Apparatus. The movement apparatus used in the experiment was bolted into the wall of an experimental testing room. The lever of the apparatus consisted of a sturdy aluminum arm which supported the subject's forearm and could freely pivot around a 2.54 cm steel rod that rotated in a ball-bearing mount. A plexiglass handle, 3 cm in diameter and 9 cm long, was attached to the end of the lever via a large bolt which could be adjusted within a slot at the end of the lever. Thus, with the subject's elbow at the pivot point of the lever, the handle could be adjusted to match the length of the subject's forearm. A cupped foam rubber pad was taped to the lever at the pivot providing comfortable support for the subject's elbow and helping to stabilize the forearm during movement. Also, the entire apparatus arm could be adjusted up or down to accommodate the height of different subjects. To record the position of the lever to within $.5^\circ$, an angular potentiometer was mounted at the bottom of the vertical steel rod. An accelerometer was mounted at the distal end of the lever. The first change in acceleration was used to measure precisely the beginning of the subject's move-

ment. Two sets of Hewlett-Packard bi-polar surface electrodes (Model #14000-13) monitored EMG activity of the agonist (biceps) and antagonist (triceps) muscles. Inter-electrode distance was approximately 2 cm and the electrodes were placed overlying the bellies of the biceps and triceps muscles. Position, acceleration, and EMG activity were simultaneously recorded on a four-channel Hewlett-Packard 740A strip chart recorder. Paper speed was fixed at 100 msec so that timing information could be determined within 5 msec.

To provide immediate knowledge of results (KR) to the subject regarding distance moved and movement time, an oscilloscope and millisecond timer were used, respectively. The oscilloscope, connected to the potentiometer, displayed the position of the subject's forearm as a white horizontal line. As the subject's forearm moved in the direction of flexion, this horizontal line moved upwards accordingly. A stationary horizontal line depicting the target was located at the top portion of the oscilloscope screen. This target line corresponded to 85° of elbow flexion (0° was full extension of the elbow) and remained constant across all distance and movement time conditions. The oscilloscope was placed on a table directly in the front of the subject's view at a distance of roughly 45 cm. The millisecond timer displayed the movement time for each movement made by the subject. Two microswitches were used to record movement time for immediate KR. The first microswitch was secured, by means of a small C-clamp, to the starting position which in this experiment was either 40° or 58° of elbow flexion. The second microswitch, also adjustable, was secured near the 85° target position. As the subject moved the lever arm away from the starting position the first microswitch was tripped which closed the circuit and started the millisecond timer. When the lever arm contacted the second microswitch close to the target the timer was stopped and displayed the resulting movement time. The millisecond timer was located above the four-channel recorder which was placed on a table to the right of the subject and out of his view as he watched the oscilloscope.

A third adjustable microswitch was positioned on the other side of the 85° target. If the lever arm contacted this microswitch a small red light would light up on a small control panel in front of the experimenter, indicating that the subject had exceeded the target tolerance for that condition (i.e., the movement was too long). The exact placements of these three microswitches depended on the experimental condition and are described in the Procedures section.

Procedures. With the subject comfortably seated and his forearm supported by the lever arm of the apparatus, the handle at the end of the lever arm was adjusted to match the length of his forearm and the height of the lever arm was adjusted to minimize the contribution of the horizontal shoulder flexor muscles. To achieve this, the subject's right shoulder was slightly elevated compared to his left shoulder. The surface electrodes were then placed on the bellies of the biceps and triceps and the two ground electrodes were placed on non-muscular and stable portions of the subject's right shoulder.

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Prior to the experimental trials, the subject was allowed several prac-
tice trials, making rapid elbow flexion movements in a ballistic fashion
such that a minimal amount of oscillation would be produced around
the movement endpoint. Since this type of oscillation is quite normal for
rapid, ballistic movements (Partridge, 1961), the subject was only told to
keep the oscillations to a minimum. It was suggested to the subject that
he concentrate on the horizontal line near the top of the oscilloscope
screen depicting the 85° target position in planning his ballistic
movements. The subject was told that his MT could be observed on the
millisecond timer after each trial.

Following the initial familiarization period during which the subject
became accustomed to moving the lever arm with some control, further
practice trials were given at a specific MT-distance condition. For each
MT-distance condition, the subject was told that his task was twofold:
To produce rapid elbow flexion movements which stayed within both
MT and distance tolerances (described below). Prior to experimental
trials in each condition, the subject was given several practice trials to
learn the correct movement. The experimental trials began when both
the experimenter and the subject were confident that the correct move-
ment had indeed been learned. Often, up to 30 practice trials were
needed for this purpose.

The sequence of events for each experimental trial was as follows. The
experimenter said "Ready-Set," followed by a constant 1 sec
foreperiod. At the end of the foreperiod, the experimenter initiated a
switch on the control panel which sounded a tone and at the same time
started the strip chart recorder. The tone and the recorder stayed on for
800 msec on every trial. When the subject heard the tone he was told to
initiate his movement fairly quickly after hearing the tone. It was em-
phasized, however, that his reaction time to the tone was not impor-
tant—the tone only served as a stimulus to move. After the movement,
the subject was told whether his movement had stayed within the
distance tolerance. Also, after each trial, the experimenter told the sub-
ject his MT (the subject could observe his MT on the timer if he wished).
If the subject was within the MT tolerance he was told that his move-
ment was "Good." The subject was told that his movement was "Too
Slow" if his MT was greater than designated MT tolerance and "Too
Fast" if his MT was less than designated MT tolerance. The subject
returned the lever arm to the starting position and awaited the next trial.
The inter-trial interval was 20 sec.

The subject performed in four MT-distance conditions: 100
msec-27°, 100 msec-45°, 160 msec-27°, and 160 msec-45°. These
four conditions were presented in blocks, and the presentation of these
blocks was randomized for each subject. In the 100 msec conditions,
for example, the subject was told to produce movements such that the
time from movement initiation to tripping the second microswitch (stop-
ping the millisecond timer) was 100 msec. The MT tolerance chosen as a
result of pilot work was $\pm 10\%$ of the MT. Thus, in the 100 msec condi-
tion, a MT from 90-110 msec was acceptable whereas in the 160 msec

conditions, a MT of 144–176 msec was acceptable. To define the 27° and 45° movements, the starting positions were 58° and 40°, respectively. The first microswitch could be positioned at either of these two positions. The two remaining adjustable microswitches were placed around the 85° target position and defined the allowed tolerance depending on the distance moved. Also through pilot work, it was decided that $\pm 15\%$ of the distance moved would be the target tolerance. Therefore, in the 27° conditions, the two microswitches were placed at the 82° and 88° positions. In the 45° conditions, the microswitches were placed at the 80.5° and 89.5° positions. Thus, an attempt was made, unlike most EMG studies, to manipulate more clearly the variables of distance and MT. As a result of pilot testing it became clear that this attempt would not be easy. Subjects in the pilot work found it difficult to produce movements within stricter tolerances than $\pm 10\%$ of the controlled MT and $\pm 15\%$ of the distance moved. We would only like to emphasize that our results should be interpreted within the confines of our experimental methodology. An alternative method, similar to Lestienne's (1979), would allow subjects to produce movements over a range of movement times and distances and then categorize them into the desired tolerances. We also emphasize here that our methodology included no systematic manipulation of starting position, and thus we cannot rule out any initial or final arm position bias in our data. We have investigated this issue in another experiment, however (Wallace, Frankeny, & Larish, 1982).

On any experimental trial, if the subject's movement time exceeded the MT or the distance tolerance for a given MT-distance condition, the trial was excluded. The subject continued until 10 successful movements were completed within each MT-distance condition. All four conditions were completed within one 1½ hour session for each subject.

Design and Analyses. A $2 \times 2 \times 10$ (MT \times Distance \times Subjects) completely within-subject design was used and separate analyses of variance were performed on the following dependent measures: The duration of the initial agonist burst (Ag), the duration of the initial antagonist burst (Ant), the time from the beginning of Ag to the beginning of Ant (Ag→Ant), the *controlled* MT which was the time from the beginning of the movement to the tripping of the first microswitch defining the target tolerance, and the *total* MT which was the time from the beginning of the movement to the stabilization of the displacement record of the potentiometer. Onset and offset of EMG activity was defined as the first observable difference from baseline activity. In some cases, this was difficult to determine and such trials would be excluded from the analysis. The terminology and EMG data analysis was similar to that used by Hallett et al. (1975). Examples of the EMG and displacement records are shown in Figure 1.

Results

Controlled MT

It was necessary to compare the actual controlled MT's in the various



Fig. 1—Examples of four MT-distance conditions. Ag→Ant, the time from the beginning of the initial agonist burst to the beginning of the initial antagonist burst. movement time

conditions were 27° and 45° movements. The main effect of distance was significant, whereas the main effect of movement time was not significant.

Total MT

The subject's movement time to the stabilization of the displacement record

acceptable. To define the 27° conditions were 58° and 40°, respectively positioned at either of these two microswitches were placed defined the allowed tolerance so through pilot work, it was moved would be the target positions, the two microswitches ns. In the 45° conditions, the and 89.5° positions. Thus, an dies, to manipulate more clearly. result of pilot testing it became easy. Subjects in the pilot work within stricter tolerances than % of the distance moved. We r results should be interpreted al methodology. An alternative would allow subjects to produce t times and distances and then nces. We also emphasize here ematic manipulation of starting any initial or final arm position his issue in another experiment, 1982).

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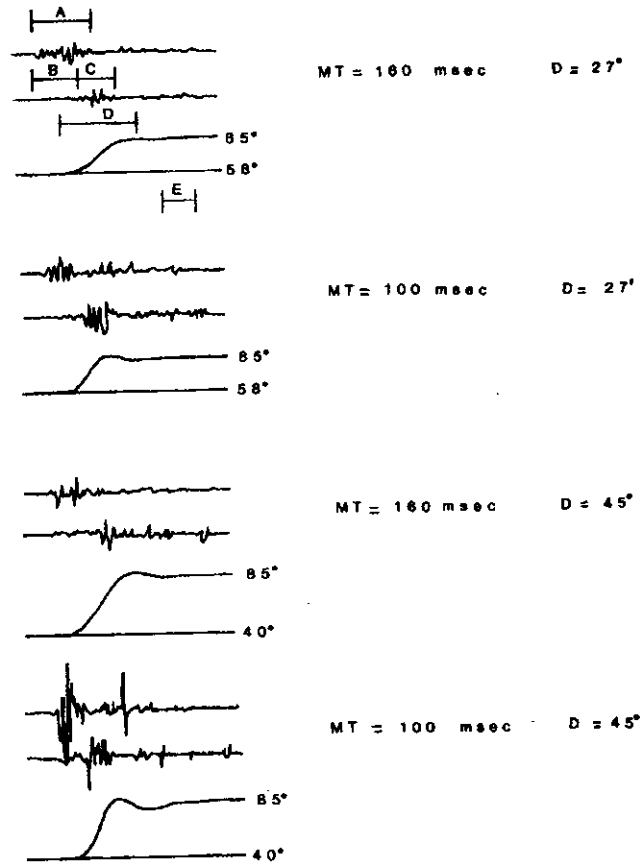


Fig. 1—Examples of representative EMG and displacement records of one subject in the four MT-distance conditions. A is Ag, the duration of the initial agonist burst. B is Ag—Ant, the time from the beginning of the agonist burst to the beginning of the antagonist burst. C is Ant, the duration of the antagonist burst. D represents the total movement time. E represents 100 msec.

conditions as a manipulation check for this variable. The means in 100 msec=27°, 100 msec=45°, 160 msec=27°, and 160 msec=45° conditions were 100.56, 102.83, 157.82, and 158.41 msec, respectively. The main effect of MT was highly significant, $F(1,378) = 7216.75$, $p < .01$, whereas the main effect of Distance and the MT × Distance interaction failed significance.

Total MT

The subject's total MT, from movement initiation to movement stabilization, was much larger than the controlled MT. The displacement records indicated that typically one or more oscillations occurred

within the target tolerance area before the movement settled down (see Figure 1). The main effects for MT and Distance were significant, $F(1,378) = 115.76$ and $F(1,378) = 32.67$, p 's $< .01$, respectively. Thus, it appears that both variables of controlled MT and Distance significantly affected total MT. As controlled MT increased from 100 to 160 msec, total MT increased for both the 27° and 45° movements. Also, as distance increased from 27° to 45°, total MT increased for both 100 and 160 msec movements.

EMG analyses

Ag. The main effect of controlled MT was significant, $F(1,378) = 405.21$, $p < .01$. The MT \times Distance interaction was also significant, $F(1,378) = 19.59$, $p < .01$. Simple main effects analysis indicated that only controlled MT affected Ag. Distance had no significant affect on Ag.

Ant. The main effect of controlled MT was significant, $F(1,378) = 111.40$, $p < .01$. Also, the MT \times Distance interaction was significant, $F(1,378) = 4.15$, $p < .05$. Simple main effects analysis indicated that as controlled MT increased from 100 to 160 msec, Ant also increased for both the 27° and 45° movements. Distance had no effect on Ant.

Ag-Ant. Only the main effect of controlled MT was significant, $F(1,378) = 342.73$, $p < .01$. For both 27° and 45° movements, Ag-Ant's significantly increased as controlled MT increased from 100 to 160 msec.

Ratios. Table 1 depicts the means and ratios of the three EMG parameters, Ag, Ant, and Ag-Ant, to total MT. It can be seen that Ag remained on from 33% to 44% of the total MT across the four MT-Distance conditions. The Ant was activated for 34% to 40% of the total MT and the onset of this burst occurred 21% to 30% into the total MT. To simplify, we can summarize by saying that for all MT-Distance conditions, both Ag and Ant were activated for slightly over one third of the total MT. The onset of Ant tended to occur slightly prior to the cessation of Ag.

Table 1
Means (in msec) and ratios of EMG parameters to total movement time (MT)

Dependent Measures	MT-Distance Condition			
	100-27°	100-45°	160-27°	160-45°
Ag	98	110	149	143
Ant	103	108	133	127
Ag-Ant	63	65	99	96
MT	296	315	333	354
Ratios				
Ag/MT	.33	.34	.44	.40
Ant/MT	.35	.34	.40	.36
Ag-Ant/MT	.21	.21	.30	.27

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Discussion

The present experiment provided several lines of evidence in support of the impulse-timing theory as developed by Wallace (1981). One prediction of the theory is that MT, not distance, affects the timing of EMG behavior. In support of this prediction, the present data indicate that when controlled MT (either 100 or 160 msec) is the same, the distance traveled by the limb (either 27° or 45°) does not affect the timing of the EMG activity. As controlled MT increases from 100 to 160 msec, however, timing of EMG activity also increases in both distance conditions. Thus, it has been shown that the timing or phasing of EMG activity in agonist and antagonist muscles was significantly more dependent on movement time than distance traveled. According to impulse-timing theory, this must be the case because it is only movement time that determines how the accelerative and decelerative forces (products of agonist and antagonist muscle contractions, respectively) are distributed during a movement.

Another prediction of the impulse-timing theory is that the timing of EMG activity (Ag, Ant, and Ag-Ant) in relation to total MT should remain constant in all discrete movement conditions, because the major accelerative and decelerative forces are deemed to occur over exactly the first and second half of the total MT, respectively, regardless of distance or total MT (Schmidt et al., 1979). Several points must be made about this issue in relation to our results. First, the ratios of EMG parameters to total MT were less than the predicted 50% for each parameter (Table 1). The Ag/MT and Ant/MT were roughly 36% while Ag-Ant/MT was approximately 25%. Thus, Ag and Ant consumed approximately one third of the total MT and Ant was activated before Ag was completely finished. An explanation why the ratios are less than 50% might be that very few of the movements produced by subjects in the experiment could be halted without one or more oscillations around the endpoint, particularly in the faster movement conditions (see Figure 1). Each oscillation takes a certain amount of time resulting in a disproportionate increase in MT relative to the EMG parameter value in the numerator of the ratios. Second, the finding that the onset of Ant occurred before Ag was completed suggests, at least in the movements performed in this experiment, that this type of braking process is needed to prevent severe overshooting of the desired distance. In fast movements, if the antagonist muscle is not activated until after the agonist burst is completed the desired endpoint might be overshoot by a considerable degree. Third, there were tendencies for the ratios to be larger in both the 160 msec conditions. This was particularly true for the Ag/MT and Ag-Ant/MT ratios. We do not, at this time, have a concrete explanation for this finding. To investigate this issue further, it would be desirable to conduct an experiment whereby several levels of MT are manipulated. In this regard, Lestienne (1979) found that the duration of the initial agonist burst remained fairly constant in relation to the total MT ($25\% \pm 5\%$), across several levels of MT. The onset of the initial an-

movement settled down (see Distance were significant, p 's $< .01$, respectively. Thus, MT and Distance significantly increased from 100 to 160 msec, and 45° movements. Also, as MT increased for both 100 and

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EMG parameters
Time (MT)

Distance Condition	Condition	
	160-27°	160-45°
10	149	143
08	133	127
65	99	96
15	333	354
	.44	.40
	.40	.36
	.30	.27

tagonist burst relative to total MT, however, was shown to decrease from 50% to 10% as MT reduced. This finding, clearly different than the present study, suggests that as MT reduces, the antagonist muscle must be activated proportionately sooner in the movement. Because actual MT's were not reported by Lestienne (1979) it is difficult to compare his findings to ours.

Changes in the timing of EMG activity witnessed in this study cannot be accounted for by speed of movement, per se. According to a speed-control model proposed by Freund and Budingen (1978), speed of movement is controlled by changes in the intensity of motor unit activity as reflected by the magnitude of the EMG signal. The timing of agonist and antagonist muscular activity is proposed to remain constant across all movement conditions. Thus, according to the speed-control model, the timing of EMG activity in all four conditions of the present study should have been the same in spite of clear changes in average velocity.¹ This was not the case. Lestienne's (1979) experiment also provided data against the speed-control model since the timing of EMG activity (duration of agonist burst and onset of antagonist burst) decreased as velocity increased. Lestienne, however, concluded that his data was supportive of the speed-control model because relative timing of agonist duration (agonist duration over MT) remained constant over several velocities. But we think this interpretation is incorrect because Freund and Budingen (1978) were not referring to relative timing. As a matter of fact, both the Freund and Budingen (1978) and Lestienne (1979) contain data supportive of impulse-timing theory. First, subjects in the Freund and Budingen (1978) study produced movements in the same MT over different distances. Thus, from an impulse-timing standpoint, it would be predicted that the duration of agonist burst would be constant, which was the reported result. Second, in the Lestienne (1979) study, when subjects produced movements with different MT's over the same distance, it was shown that the timing of EMG activity decreased as MT decreased (which resulted in higher velocities) and the relative timing of the agonist burst in relation to the MT remained constant (around 25% of the MT) for all movement speeds. Both of these latter two findings are consistent with impulse-timing predictions. It appears that part of the confusion in interpretations of EMG studies is that many times the authors have not acknowledged that speed of movement depends on both the distance and the MT and that the characteristics of the EMG records are going to be strongly dependent on the type of movement that is produced in the experiment.

Finally, our results have a bearing on other theoretical issues pertaining to the control of reciprocal muscle activity. Wallace (1981) discusses

¹By dividing the total distance travelled by the total movement time, we calculate the average velocities in the 100 msec-27°, 100 msec-45°, 160 msec-27°, and 160 msec-45° conditions to be approximately 91°/sec, 142°/sec, 81°/sec, and 126°/sec, respectively.

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however, was shown to decrease timing, clearly different than the other conditions, the antagonist muscle must be active before the movement. Because actual timing (Freund 1979) it is difficult to compare his

work was witnessed in this study cannot be compared, per se. According to a speed- and distance study by Budingen (1978), speed of movement and the intensity of motor unit activity were proportional to the EMG signal. The timing of agonist and antagonist muscle activity was proposed to remain constant across different conditions to the speed-control model, but the conditions of the present study showed clear changes in average timing. Freund's (1979) experiment also proposed a model since the timing of EMG onset (onset of antagonist burst) decreased as speed increased. However, he concluded that his data was not as good because relative timing of agonist and antagonist remained constant over several conditions. This conclusion is incorrect because Freund's data showed timing to be dependent on relative timing. As a matter of fact, Freund's (1978) and Lestienne (1979) contain contradictory theories. First, subjects in the Freund study showed movements in the same MT over different impulse-timing standpoints, it would be expected that the relative timing of agonist and antagonist burst would be constant, but in the Lestienne (1979) study, the relative timing of EMG activity decreased as MT increased (with different MT's over the same distance). The relative timing of EMG activity decreased as MT increased (with different velocities) and the relative timing of EMG activity remained constant (around 25% of total movement time). Both of these latter two findings are contradictory. It appears that part of the discrepancy in EMG studies is that many times the relative timing of movement depends on the speed of movement and that the characteristics of the EMG activity are dependent on the type of movement.

Other theoretical issues pertaining to muscle activity. Wallace (1981) discusses

impulse-timing theory (Wallace, 1981) whereby the timing of agonist and antagonist activity seems to be much more dependent on MT than distance travelled. The relative timing of EMG activity was fairly constant across the different MT-distance conditions although some departure from constancy was detected in the two longer MT conditions. Future work is clearly warranted which examines this relative timing issue across several MT levels. Also, it will be important to test the predictions of impulse-timing theory with regards to the regulation of the magnitude of EMG activity in agonist and antagonist muscles during the production of different movements.

whether agonist and antagonist muscle activity is controlled centrally in the brain or at, so-called, lower levels. There is convincing evidence for both views (e.g., Angel, 1977; Garland, Angel, & Moore, 1972; Hallett, Shahani, & Young, 1975; Wadman et al., 1979). Apart from Lestienne's work (1979) and the present study, other investigations have found a constancy in the timing of muscle activity relative to the step cycle in the cat (Engberg & Lundberg, 1969) and fixed ratios of EMG activity in the ankle, knee, and hip, despite wide variations in upright posture due to ankle rotation (Nashner, 1977). Other constancies have been found in kinematic parameters (see Shapiro, Zernicke, Gregor, & Diestel, 1981, for a review). Neural constraints upon coordinated muscle actions have been defined in different ways. One view, advocated by Shapiro et al. (1981) as well as Schmidt (1980), is that the fixed ratios are invariable features of some centralized motor program. That is, the relative timing of EMG activity or of the limbs is controlled centrally by the brain. A different perspective is advocated by Kugler, Kelso, and Turvey (1980) in which the fixed ratios are a result of the dynamic properties of coordinative structures—groups of muscles that are constrained to act as a functional unit. Presumably, the witnessed ratios are not due to instructions from the brain, but are emergent properties of the coordinative structure located at "lower levels" in the motor system.

In summary, our results appear to conform well to impulse-timing theory (Wallace, 1981) whereby the timing of agonist and antagonist activity seems to be much more dependent on MT than distance travelled. The relative timing of EMG activity was fairly constant across the different MT-distance conditions although some departure from constancy was detected in the two longer MT conditions. Future work is clearly warranted which examines this relative timing issue across several MT levels. Also, it will be important to test the predictions of impulse-timing theory with regards to the regulation of the magnitude of EMG activity in agonist and antagonist muscles during the production of different movements.

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