Relation Between EMG Activation Patterns and Kinematic Properties of Aimed Arm Movements

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ABSTRACT. Aimed flexion movements of the arm of different amplitude and duration were studied. Velocity and acceleration traces of movements with equal duration but different amplitude were equal, apart from a scaling factor (ratio between movement amplitudes). After appropriate scaling, EMG activity of the first agonist burst for these movements superimposed. This was not true for EMG activity in the antagonist muscle.

For movements with equal amplitude, but different duration, the time to peak acceleration was constant for all MT's. Except for this fact, traces of acceleration, velocity, and agonist activity following the time of peak acceleration were about equal after appropriate scaling in time and amplitude. The integral of EMG activity in the first agonist burst increased linearly with peak velocity. For the antagonist burst, the integrated EMG activity increased more than proportionally.

During movements made as fast as possible, subjects used a different strategy by varying the duration of the accelerating phase for movements of different amplitude. Movement amplitude was achieved by adjusting the duration of the agonist burst and the onset time for the antagonist muscle. Amplitude of the antagonist burst was constant within a narrow range for movements of different amplitude. These results did not change when the inertial mass was doubled by loading the arm with an additional mass.

RECENTLY, SEVERAL PAPERS have appeared that describe invariant properties of aiming movements (e.g., Soechting & Lacquaniti, 1981; Ruitenbeek, 1984). These studies have shown that the velocity trace is similar for movements with different amplitude and duration. More specifically, it has been shown that all velocity traces have a similar shape after scaling in amplitude (for movements with equal duration but different amplitude) or scaling in amplitude and time (for movements with equal amplitude but different duration). These observations were obtained for movements that were not fast (duration longer than 0.25 s).

In many other papers, EMG activity was studied during aiming movements. These studies show that different movement amplitudes were affected by concomitant changes in the magnitudes of both agonist bursts (Brown & Cooke, 1981; Berardelli, Rothwell, Day, Kachi, & Marsden, 1984). Moreover, the duration of the first agonist burst increases with
the duration of the movement (Lestienne, 1979; Brown & Cooke, 1984). Since muscle contractile properties are nonlinear, this information on EMG activity is very valuable for the understanding of the motor programming of aiming movements.

In most studies (e.g., Lestienne, 1979; Brown & Cooke, 1981; Soechting & Lacquaniti, 1981), EMG activity has been recorded for a wide range of movement amplitudes and durations. However, in our studies, characteristics of EMG activity (such as amplitude and duration of agonist and/or antagonist bursts) have been related to peak velocity, amplitude, and duration of movements. By doing so, it is implicitly assumed that the trajectory and the velocity of movements differ only by scaling factors in time and amplitude. Otherwise, movement parameters such as peak velocity, movement time (MT), movement amplitude, etc. are not simply related to movement amplitude and duration. Although this invariance has been shown for slow aiming movements (Soechting & Lacquaniti, 1981; Ruitenbeek, 1984), as well as for more complex movements (Giencross, 1973; Viviani & Terzuolo, 1982; Shapiro, Zernicke, Gregor, & Diestel, 1981), it has not been shown for fast movements. Since muscle nonlinear properties become more prominent at high shortening or lengthening velocities, the invariance of fast movements and/or the invariance of the EMG activity in agonist and antagonist muscles (i.e., the motor program) cannot be concluded from previous observations.

The aim of this paper is twofold. First, we investigate whether the traces of the trajectory, velocity, and acceleration of fast movements of different amplitude and duration are identical (excluding the scaling factor in time/amplitude related to amplitude and duration of the movement). Secondly, the quantitative relation between the movement characteristics and EMG activity is investigated. In order to investigate the role of nonlinear muscle properties on the relation between EMG and movement kinematics, EMG activity is scaled in the same way as the acceleration traces. This is an important issue, since it is still an open question whether the motor system controls the trajectory of the movement or whether the motor system uses a generalized motor program and just scales the EMG activity patterns for agonist and antagonist muscles. In the latter case, the movement trajectory may not be invariant due to the muscle nonlinearities.

In order to investigate these issues, arm movements in flexion direction were studied. Flexion movements were studied, since it has been shown that the relative work of the main agonist muscles during flexion movements remains the same over a large range of velocities and over a large range of inertial loads at the wrist (Bouissett, Lestienne, & Maton, 1976). Subjects were tested in a condition in which they had to make movements of variable duration and amplitude. In another condition, subjects had to make movements over a variable distance as fast as possible. The experiments were repeated with an additional weight at the wrist. By adding a weight, accelerating forces for movements with the same distance and MT have to be larger. Therefore, it may be expected that effects
of the nonlinear muscle properties will be more evident for the loaded movements than for the unloaded movements.

Methods

Subjects

Five, right-handed subjects (3 male and 2 female, 24–31 years of age) participated in this study. Each subject was tested in all experimental conditions. One subject was tested twice: once with surface electrodes and once with intramuscular electrodes. All subjects were healthy and had no known history of neurological disorder.

Apparatus

Arm movements were recorded with a commercially available "Selspot" system that consists of a dual-axis lateral photodetector, in front of which a lens (Canon 50mm, f:0.95) is placed. With this system the position of infrared Light Emitting Diodes (LED's) is detected.

The subject sat at a table with 5 LED's equidistantly (7.5 cm) spaced on a straight line. A LED was attached to a pointer held by the subject. The weight of this pointer with the attached LED was less than 50 gms. Movements were made in a horizontal plane above the table. Position data (x-y coordinates, accuracy 0.5 mm) were sampled at 320 Hz and fed into a HP1000 computer for further analysis.

Movements were made with the right hand, always aiming at the most leftward LED. Differences in movement amplitude were obtained by variations in beginning position. The subject was seated such that the elbow-joint angle in the beginning position of the 15 cm movement was about 90°. This was done in order to eliminate, as much as possible, variations in the mechanical advantage due to the different beginning position of the movements of different amplitude. By this choice, the variations in mechanical advantage of muscles is less than about 4%. Since the mean contribution to the aiming movements was given by flexion of the arm, EMG signals were recorded from m. biceps (caput longum), m. brachioradialis, m. brachialis, and m. triceps (caput laterale and caput longum). EMG of m. brachialis was recorded at the medial side of the arm, just above the elbow joint.

EMG activity was measured with silver/silver-chloride surface electrodes. Signals were rectified and slightly smoothed (time constant 10 ms). All EMG-signals were sampled at 320 Hz and further analyzed on a HP1000 computer.

For one subject, intramuscular EMG signals were also recorded, with thin (diameter 25 μm) nylon coated karma wires (California Fine Wire Comp.). The nylon isolation was removed over about 5 mm. Two wires were inserted in both the agonist and antagonist muscle. EMG signals were first band-pass filtered (3-dB cut-off frequencies at 16 Hz and 32 kHz), rectified and filtered again (time constant 10 ms). Subsequently, the signals were sampled at a rate of 320 Hz and stored for further
analysis. No difference was observed between data obtained with surface or intramuscular electrodes, in agreement with the results of Maton, Bouisset and Metral (1969). Therefore, no distinction is made in the results between the two types of EMG data.

Procedure

Subjects had to make flexion movements with their right arm at amplitudes of 7.5, 15, 22.5, and 30 cm and with different movement times. In order to eliminate any effect of gravity, the wrist of the subject was hanging in a 3 m long rope that was attached at the ceiling of the room. In this way, the only forces exerted by the subject were in flexion/extension direction, and no weight-dependent downward forces were involved. The instruction to the subject was to vary the velocity of the movements such that movement times (MT) were about equally distributed in the range between 100 and 250 ms. Movement time (MT) was defined as the interval between the time when hand velocity exceeded a level of 20 cm/s and the time when velocity crossed the zero level.

Before each experimental session, subjects made about 50 practice trials. During these practice trials, the experimenter observed velocity profiles on an oscilloscope and informed the subject about MT of the movement. No feedback about MT was given during the experimental session. For each distance, about 200 movements were measured. In an off-line analysis, responses with equal MT's within 5% were selected for each distance. Selections were made of responses with mean MT's of 100, 125, 150, 175, 200, and 250 ms. Each cluster of selected responses contained 10 to 15 responses, which were synchronized at the time when velocity exceeded the level of 20 cm/s and that were subsequently averaged.

In another set of trials, subjects were instructed to move as fast as possible to the target position. No special requirements were made on accuracy. For each distance, 20 movements had to be made. Responses were averaged after synchronization at the time when velocity exceeded the level of 20 cm/s.

Both types of experiments were repeated with an additional mass at the wrist of 0.9 kg. (at a distance of about 25 cm from the elbow joint). Because the wrist was hanging in a rope, this additional mass increased the inertia but did not generate any gravitational forces. The subjects were allowed to practice until they felt comfortable in making the movements with the added load.

Before each trial the subjects received a warning sound. One and a half seconds later the target LED went on, indicating the start of data acquisition. At the warning signal, the subject had to lift the pointer, holding it above the start position. When the target LED went on the subject had to move to the target LED without touching the table. Only when the hand had come to a complete stand-still was the subject allowed to rest on the table. Any contact with the table during the movement was excluded, to ensure braking of the movement by the antagonist muscle and the visco-elastic properties of the arm only.
Data analysis

In order to investigate to what extent the movement trajectory and velocity and acceleration traces were invariant, apart from a scaling in amplitude and time, velocity and acceleration traces were reduced to a standard shape. The reasoning was that if muscle force increases, it leads to an increase of acceleration of the arm. This will lead to a proportional increase in movement amplitude. The mathematical relationship between position, velocity, and acceleration and their scaling factors for scaling in time and/or amplitude is explained in the Appendix.

By definition, velocity traces of movements with equal MT's are all zero at time MT after movement onset. Since all movements have an overshoot (see Results), movement amplitude \( d \) at time MT is always larger than the final movement amplitude \( D \). According to equation A3 all velocity and acceleration traces have to coincide with the traces for the movement with amplitude \( d_m \) after scaling with \( d/d_m \), if muscle nonlinearities are absent. Therefore, amplitude \( d \) at time MT rather than final movement amplitude \( D \) was used to calculate the required scaling factor.

In order to investigate the invariance of acceleration, velocity, and EMG traces for movements with equal amplitude, but different duration, these traces had to be scaled both in amplitude and in time. The time scaling was performed according to equations A4 and A5 in the Appendix, with time zero as the time of onset of the movements (the time of synchronization).

If viscoelastic properties of the forearm during these fast movements are neglected with respect to the inertial forces, force is proportional to acceleration. Since EMG activity is related to muscle force in isometric conditions, EMG traces were scaled with the same factors as acceleration traces. Any differences between traces of EMG and acceleration after scaling reflect the influence of nonlinearities in the contractile mechanism.

Results

Movements with Constant MT and Variable Amplitude

Flexion movements with different amplitude (7.5, 15, 22.5, 30 cm) and different duration (100, 125, 150, 175, 200, 250 ms) were studied. In order to investigate the invariance of the shape of velocity and acceleration traces, the traces were scaled in time and/or amplitude. The correct scaling factors, which are different if velocity and acceleration traces are scaled in time, have been derived and explained in the Appendix.

Figure 1 shows typical average traces for acceleration, velocity, and EMG activity for agonist and antagonist muscles for movements with MT 125 ms. Figure 1 suggests that velocity traces all have a similar shape independent of movement amplitude. The same applies to the acceleration traces. In order to investigate this similarity in more detail, accel-
Fig. 1—Average traces for acceleration, velocity and EMG of agonist (biceps long head) and antagonist (triceps lateral head) muscle for movements with MT-125 ms and with amplitudes of 75 (+), 15 (∆), 22.5 (○), and 30 (∇) cm. The vertical bars in velocity and acceleration traces indicate the standard deviation at several points in time. EMG is in arbitrary units. Note the different zero position on the time axis for EMG traces.

eration and velocity traces for each movement amplitude were multiplied by the quotient between amplitude of that movement and amplitude of the 22.5 cm movement. Figure 2 shows that, after multiplication acceleration and velocity traces follow a common trajectory during the larger part of the movement. This was a consistent finding for all MT's and all subjects. Very small systematic differences between the traces were observed at the end of the movement where the movement with the smallest amplitude tended to have the largest positive velocity during the phase of overshoot (i.e., at times beyond MT). This positive phase in the velocity traces, which is related to an overshoot in the position traces, is observed in all movements for all subjects.

The SD in the acceleration trace usually increases with the value of acceleration and is maximal at peak acceleration. In general, the ratio between variability in acceleration and acceleration was about constant (see Figure 1 & 4). The same was true for velocity. The variability in
Characteristics of Aimed Arm Movements

Fig. 2—Average traces of acceleration, velocity and EMG responses for different movement amplitudes (7.5 (+), 15 (△), 22.5 (○), and 30 (□) cm) and same MT (125 ms) after multiplication by a factor d/dv. d₀ is movement amplitude of the 22.5 cm movement at the time when velocity is zero (see Methods). Multiplication factors were 2.45, 1.43, 1.0, and 0.71 for movements of 7.5, 15, 22.5, and 30 cm.

velocity varied proportionally with velocity. This result implies that variability in force (which is related to acceleration) is not simply an addition of the variability in the acceleration trace. It indicates that variability in different phases of the acceleration traces compensate, such that variability near the end of the velocity trace is still relatively small. This was found for all subjects.

Amplitude of EMG activity of the first agonist burst increases with movement amplitude. If EMG traces are multiplied with the same constant as used for the acceleration traces, their amplitudes appear to be about the same. However, the variability of EMG activity did not allow us to make very precise statements as could be done for acceleration and velocity. In general, duration of the first burst of EMG activity in the agonist muscle is about the same within the variability due to the stochastic character of EMG activity. This is in agreement with results of Lestienne (1979) and Brown and Cooke (1981).
As shown in Figure 1, onset of EMG activity in the antagonist muscle (triceps) starts at about the same time for all movement amplitudes. Its amplitude and duration increase with movement amplitude.

The results for the EMG traces were very similar for data obtained with surface electrodes and with intramuscular electrodes, which is in agreement with earlier observations (Bigland & Lippold, 1954; Maton, Bouisset & Metral, 1969).

Signals of acceleration, velocity and EMG of movements with amplitudes of 15 and 30 cm with and without an additional load of 0.9 kg are shown in Figure 3. Figure 3A shows average acceleration and velocity traces. The signals obtained with and without the additional load are very similar. This becomes obvious if the standard deviation in the velocity and acceleration traces in Figure 1 is considered. The standard deviation in traces in Figure 3 is about the same as that in Figure 1.

Figure 3B shows the signals after multiplication of the traces by a factor \( \frac{d\theta}{\omega} \), which was close to 2 in this particular case. It shows that the acceleration and velocity traces nearly coincide. Any differences were small and not systematic between subjects.

EMG responses of the loaded and unloaded movements, after multiplication by the factor \( \frac{d\theta}{\omega} \), are shown in Figure 3C. It appears that the duration of the initial burst is about the same for all movements. For movements with the same amplitude and duration, EMG activity in both

Fig. 3—Average traces of acceleration, velocity, and EMG responses for normal and loaded (0.9 kg) movements with MT 150 ms and amplitudes of 30 cm (Δ and O, resp.) and 15 cm (Δ and +, resp.): (A) Traces of acceleration and velocity, (B) traces of acceleration and velocity after multiplication by \( \frac{d\theta}{\omega} \), and (C) EMG traces after multiplication by \( \frac{d\theta}{\omega} \).
agonist and antagonist muscles is always larger for the loaded movement than for the unloaded movement. The difference is considerable in the antagonist (triceps) muscle. After scaling of the EMG signals, the agonist bursts do not completely coincide, whereas the acceleration traces do. In general, if differences between EMG traces were observed after scaling, it was the EMG trace belonging to the movement with the smallest amplitude that exceeded the size of the other traces. However, comparison with Figures 2, 5, and 6 shows that usually any differences are small.

If elastic and viscous properties of the forearm during these movements are neglected with respect to inertial forces, then acceleration is proportional to muscle force. If one considers that the inertia of the forearm is about 0.07 kg·m², the added weight increases the inertia by a factor of about 2. During isometric contractions, EMG activity is linearly related to muscle force (Lippold, 1952). If a similar relation holds between EMG activity and force during these movements, then EMG activity for movements with the same load should be expected to coincide after scaling. Also, one has to expect EMG bursts in the agonist muscle to be twice as large for the loaded movements. As already mentioned, differences between EMG traces of the agonist muscle for corresponding movements after scaling are in general smaller than in Figure 3C. The fact that traces sometimes do not coincide may have three reasons. One reason is that, depending on the load, on movement amplitude, or movement duration, different synergistic muscles are activated with variable intensity. This possibility will be dealt with later. The other explanations may be that EMG activity is not proportional to muscle force because of nonlinearities in muscle mechanical properties, or that viscoelastic components may not be neglected. These issues will be dealt with in the Discussion.

In the antagonist muscle, the EMG traces of the loaded movements are much larger than the EMG patterns for the unloaded movements. Considering the twofold increase of the inertial mass by the load, the braking of the movement requires forces about twice as large. The data in Figure 3C show that the antagonist EMG activity increases disproportionately. This points to nonlinearities in the contractile mechanism of the muscle during lengthening (see Discussion).

**Movements with Variable MT and Constant Amplitude**

Figure 4 shows traces for movements with equal amplitude but different MT. Peak acceleration is reached at about the same time for all movement times. The time from movement onset to peak acceleration was in the range between 30 and 40 ms. The intersubject variability of the mean times to peak acceleration for different movement amplitudes was about 6 ms. Between subject variability of the mean times to peak acceleration was 5 ms and was the same for all movement amplitudes. Any differences in times to peak acceleration were not significant in an analysis of variance, $F(4,4) = 1.06; p > 0.05$. The time of peak deceleration is not the same for all movement times in agreement with earlier
Fig. 4.—Average traces of acceleration, velocity, and EMG activity of movements with equal amplitude (22.5 cm) but different MT: 125 (○), 150 (●), 175 (△), 200 (+), and 250 (×) ms. The vertical bars in velocity and acceleration traces indicate the standard deviation at several points in time.

observations (Zelaznik, Schmidt, Gielen, & Milich, 1985). For MT 125 ms, the acceleration trace changes sign very rapidly and reaches peak deceleration earlier for traces of movements with smaller MT. This is consistent with the large burst of activity in the antagonist (triceps) muscle, which causes a large deceleration of the movement. For longer MT’s the antagonist activity starts later in the movement and is smaller. This probably explains the longer duration of the accelerating and decelerating phase and the longer duration of the velocity traces.

Figure 5 shows traces for movements with variable MT and the same amplitude of 30 cm after scaling according to equation A4 in the Appendix. After scaling in time, peak acceleration comes earlier for traces belonging to the longer MT’s. This is in agreement with the data in Figure 4, since peak acceleration came at about the same time for all MT’s before scaling. As a result, the time of peak velocity shifts to smaller values for longer MT’s after scaling. Considering the SD for the time to
Fig. 5—Traces of acceleration, velocity, and EMG activity after scaling for movements with equal amplitude (30 cm) and different MT (125 (□), 150 (○), 175 (△), 200 (+), and 250 (×) ms). Units of EMG are arbitrary but the same as in Figure 6.

peak acceleration of about 6 ms, and the fact that each acceleration trace in Figure 5 is obtained by averaging 10 to 15 movements, the differences in time to peak acceleration in Figure 5 after scaling are systematic and appear to be significant in an analysis of variance, $F(4,4) = 14.2; p < 0.05$.

For movements with different MT, the amplitude of EMG activity in the first agonist burst is larger for shorter MT’s (Figure 4). After scaling, the amplitude of EMG activity in the first agonist burst is more or less the same for all MT’s. However, the time course after scaling is somewhat different. In Figure 4, all bursts of activity follow approximately a common trajectory during the rising phase. This explains why, after time scaling of EMG activity, the EMG burst for short MT’s rises less steeply than for longer MT’s.

For short MT’s (125 and 150 ms), EMG activity in the antagonist muscle starts at about the same time after scaling. For longer MT’s, onset
of EMG activity in the antagonist muscle is hard to define due to the relatively small amplitude of the antagonist burst with respect to the tonic EMG level. The duration and amplitude of the antagonist bursts after scaling is clearly different for different MT's.

In Figure 6, results are shown for movements of different MT with the added inertial load after scaling according to equation A4 in the Appendix. With regard to the traces of velocity, acceleration and EMG activity in agonist and antagonist muscles the same conclusions can be drawn as for the unloaded movements. One difference is that in general the amplitude of the agonist and antagonist bursts is larger for these loaded movements than for the unloaded movements in agreement with results of Lestenne (1979).

Considering the biphasic shape of the velocity traces in all figures, these results clearly show that all movements have an overshoot. This shows that the assumptions made by Meyer, Smith, and Wright (1982),

Fig. 6—Traces of acceleration, velocity, and EMG activity after scaling for movements with equal amplitude (30 cm) and different MT: 125 (C), 150 (O), 175 (△), 200 (+) and 250 (×) ms. Units of EMG are arbitrary but the same as in Figure 5. Inertial mass of the arm was increased by an additional load of 8.9 kg at the wrist which doubles the inertia.
that the second half of each force-time curve is an inverted mirror image of the first half and that the force at the beginning, middle and end of the temporal movement interval is zero, are not correct; at least not for these type of movements.

Relative Contribution of Synergistic Muscles

The duration of the first burst in the three agonist muscles (biceps caput longum, brachialis, brachioradialis) was the same in all the experiments. In brachialis and brachioradialis, the amplitude increased with movement amplitude and with shorter MT's just as in the biceps muscle. Since the variability of EMG did not allow a detailed comparison of EMG traces, EMG activity integrated over the duration of the burst was chosen as a measure of the contribution of a muscle to flexion or extension forces. EMG activity integrated over the duration of the first agonist burst was plotted in Figure 7 for several muscles as a function of peak velocity in the movement. Since the velocity traces for different MT's are similar, the result in Figure 7 does not change when integrated EMG is plotted as a function of 1/MT or mean velocity instead of peak velocity. The only difference exists in a constant scaling (compressing or extending) of the horizontal axis.

Figure 7A shows that over the range of peak velocities in this study, integrated EMG, in all synergistic muscles acting as agonists, increases linearly. This was found true for all subjects. Since the amplitude of EMG is no quantitative measure of muscle force, because synergistic muscles may differ in type of muscle fibres, it is not possible to determine quantitatively the contribution of different synergists. However, the fact that data points for different muscles follow a similar linear relationship indicates that the relative weight of the activation of the synergistic muscles remains constant for the movements. This is in agreement with results of Bouisset et al. (1976) who found that the relationship between the activities of the main flexor muscles remains constant whatever the velocity and inertia may be. The same result was found if an additional mass was attached at the wrist.

Since straight line fits of the data in Figure 7A do not pass through the origin, a linear relation between integrated EMG and peak velocity is not valid over the whole range up to very low velocities. Instead, for lower velocities another relationship is necessary to describe the data in agreement with data of Lestienne (1979) who found a quadratic relationship. This deviation from a linear relation for small peak velocities can be easily understood from biomechanical considerations (see Appendix).

For the antagonist muscles, integrated EMG does not follow a linear relationship. This was always very clear for all subjects for the antagonist muscles but not for agonist muscles and is probably due to nonlinear muscle mechanical properties (see Discussion). Figure 7B shows that the EMG activity in the long head and the lateral head of the antagonist muscle (triceps) varies in a very similar way. Therefore, the relative weight of the activation of the antagonistic muscles is the same for all
movements in this study. A relation similar to that in Figure 7B was found for movements with an added load at the wrist.

For extension movements, where triceps acts as an agonist muscle, the integrated EMG of triceps muscle increased linearly with peak velocity, whereas that for biceps muscle did not. Therefore, the different behavior of flexor and extensor muscles in Figure 7 does not reflect differences in intrinsic muscle properties of flexor and extensor muscles but is related to whether the muscle acts as agonist or antagonist muscle.

**Aiming Movements Made as Fast as Possible**

The results for movements made as fast as possible are shown in Figure 8. Except perhaps for the 7.5 cm movement, all acceleration traces share a common trajectory during the first part of the accelerating phase. As a consequence the initial phases of all velocity traces overlap with the exception of the 7.5 cm movement. Traces of smaller movements start to deviate earlier from the common trajectory passing over into a decelerating phase. The duration of the accelerating phase increases with movement amplitude. This may be related to the longer duration of the first agonist burst. These results are in agreement with results of Wadman, Denier van der Gon, Geuze, and Mol (1979). Due to the initial common trajectory of movements, time to peak acceleration is not the same for these movements in contrast to the observations for movements with
variable duration and amplitude. The fact that the traces for the 7.5 cm movement show some differences with the other traces is probably due to an earlier observation (Denier van der Gon, personal communication) that subjects adopt a different strategy for making movements of very small amplitude as fast as possible.

There was a clear relation between onset of the antagonist burst and movement amplitude: onset of the antagonist arrived later in the movement for larger movements. The size of the antagonist burst was about the same for all movements in agreement with results of Wadman et al. (1979) and Brown and Cooke (1981). The fact that amplitude and duration of the antagonist burst remains about constant for movements of different amplitude again illustrates a clear difference with movements that are not as fast as possible. For the latter type of movements a large variation in amplitude and duration of the antagonist EMG burst was observed (see Figures 1 & 4).

For movements with the additional load at the wrist, the results are qualitatively the same. This is shown in Figure 9. However, quantitatively the movement characteristics differ. The amplitude of the acceleration

![Graphs of acceleration, velocity, and EMG activity for movements made as fast as possible. Movement amplitudes 30 (□), 22.5 (○), 15 (∆), and 7.5 (+) cm.](image-url)
Fig. 9—Traces of acceleration, velocity, and EMG activity for movements made as fast as possible with an additional mass at the wrist of 6.9 kg (l = 0.656 kg·m²). Movement amplitudes are 30 (□), 22.5 (○), 15 (∆), and 7.5 (+) cm. These data are from the same experimental session as those in Figure 8. Units for EMG are the same in this figure as in Figure 8.

Traces is smaller for the loaded movements despite the larger size of the first agonist bursts. In agreement with the smaller acceleration, the velocities reached during the loaded movements are smaller. As a consequence the duration of the loaded movements increases (see Wadman et al., 1979).

The results for EMG activity in the agonist burst are the same as in Figure 8. Amplitude of the agonist burst increases with movement amplitude and has a longer duration. For smaller movements the antagonist activity starts earlier. The size of the burst is constant within a smaller range and is the same as for the unloaded movements.

Discussion

The results of this study show that velocity and acceleration traces for aiming movements with different amplitude and constant MT have iden-
tical shape. Although the variability of EMG did not allow us to make exact statements, EMG activity in the first agonist burst appeared to scale in time and amplitude similarly to the acceleration traces. This was not the case for EMG activity in the antagonist burst.

Clear differences were observed between acceleration traces of movements with variable duration and amplitude and movements made as fast as possible. In the latter, time to peak acceleration was not constant, but all acceleration traces followed a common initial trajectory.

For movements with different MT and constant amplitude, the time to peak acceleration was the same. After the time of peak acceleration, the acceleration trace changed gradually, becoming slower for the longer MT’s. This result is not compatible with the notion that traces of velocity and acceleration for movements with different MT can be rescaled to an invariant shape. It may be a planned strategy of the motor program generator to have peak acceleration at the same point in time for all movements. However, since the peak of EMG activity in the agonist bursts shifts in time, coming later for shorter MT’s, it probably does not reflect a property of the motor program but may be a consequence of muscle nonlinearities. It may partly be explained by the force-velocity relation (Hill, 1938). It is well known (e.g., Bigland & Lippold, 1954) that at a constant activation muscle force decreases with increase of shortening velocity, up to a maximal shortening velocity, where the muscle cannot produce any force, whatever its activation. For flexion movements of the forearm, flexion force falls off to 50% of the isometric force at a hand velocity between 0.5 and 1 m/s (Asmussen, Hansen, & Lammert, 1965; Jørgensen, 1976). This nonlinearity will have a larger impact on the movement trajectories for the short MT’s that show the highest velocities. Clark and Stark (1974) found in a simulation study for saccadic eye movements that peak acceleration came at the same time for saccades of different amplitude and duration. In their study, this was caused by the time constant of muscle contraction and by the force velocity relationship, supporting our explanation implicating the role of muscle properties.

In view of the results of Asmussen et al. (1965) and Jørgensen (1976), the effect of the force velocity relation on the movement trajectories in this study is rather small. Simulation studies showed that with the parameters as found by Asmussen and Jørgensen, peak velocities of 3 m/s cannot be reached in movement times of 150 ms or shorter. This indicates that the force-velocity relation may not be valid during all dynamic conditions, at least not with the parameters as found by Asmussen et al. (1965) and Jørgensen (1976), or that it may be compensated by other nonlinearities. A possible explanation may be found in the observation of Partridge (1967) who found that muscle alone without reflex feedback can show an appreciable inertial compensation. This compensation may give rise to forces delivered by the muscle to the load, which varied by almost 10,000 times depending on the load impedance. Another explanation for the apparent absence of the effect of the force-velocity relation may come from the nonlinear summation of twitches of motor units.
Burke, Rudomin and Zajac (1970, 1976) have shown that at recruitment the first interval between action potentials of the motor unit may be very short (about 10 ms). After these two action potentials, also called a "doublet", firing rate of the motor unit drops to a range between 7 and 30 spikes/s. It has been shown (Burke et al., 1976) that these doublets may cause a more than twofold increase in twitch force that may go up to a factor of 5. This phenomenon has an effect just opposite to the force-velocity relationship and may explain why, after scaling, the shape of the traces of EMG velocity, and acceleration does not seriously depend on movement amplitude and duration.

The comparison of the acceleration and velocity traces after scaling was based on the assumption that viscoelastic components of force could be neglected with respect to inertial components. Mechanical properties can then be subdivided into 3 components: short range muscle stiffness, reflex contributions, and intrinsic mechanical properties of the muscle and the surrounding tissue. It has recently been shown that the contributions of the intrinsic mechanical properties are very small with respect to the other two contributions (Vincken, Gielen, & Denier van der Gon, 1983). Since it has been shown (e.g., Cooke, 1980) that during high velocity movements the stretch reflex is nearly absent in the stretched muscle, reflex contributions to the viscoelastic properties may be neglected during these rapid movements, except perhaps near the end of the movements. Moreover, since movements start with nearly-relaxed muscles the effect of short range stiffness is absent (Rack & Westbury, 1974). As a consequence, the viscoelastic properties during the movements are very small and may be neglected with respect to inertial forces.

The linear relationship between EMG and muscle force during stationary isometric contractions (Lippold, 1952; Bigland & Lippold, 1954) suggests that EMG is a good measure of the activation of the muscle in stationary conditions. However, several nonlinearities are involved in the production of muscle force in dynamic conditions, giving rise to a nonlinear relation between EMG and force during movements. A particular problem concerns the relationship between muscle force and lengthening velocity. This relation is important for understanding quantitatively the effect of the antagonist muscle in braking the movement. Unfortunately, no quantitative data during normal muscle activation patterns are available. Either data have been obtained during artificial muscle stimulation (Joyce, Rack, & Westbury, 1969) or, in case normal movements were concerned, only indirect information was obtained (Bigland & Lippold, 1954; Gielen & Houk, 1984). Data from Gielen and Houk (1984) in humans and data obtained from the decerebrate cat (Houk, personal communication) suggest that, during constant activation, muscle force initially increases for small stretch velocities but decreases for larger lengthening velocities. This is in agreement with the data in Figure 4, because the antagonist activity is very small for slow movements with the longer MT's but increases progressively for movements with higher velocities. A precise interpretation of the antagonist burst seems hard. First of all there is the electromechanical time-constant governing the
translation of EMG to muscle force. Due to this time constant, the antagonist muscle not only has to break the movement, thereby acting against the kinetic energy of the limb, but also has to oppose the force in the agonist muscle that remains after the first burst (Sherif, Gregor, Liu, Roy, & Hager, 1984). This may explain why the EMG burst in the antagonist muscle increases more than proportionally when larger accelerating and decelerating forces are required (Figures 1, 2, 4, 5, & 7).

APPENDIX

Scaling of velocity and acceleration traces

It is assumed that during the fast movements studied in this manuscript viscoelastic forces may be neglected compared to the inertial forces. Then muscle force is linearly related to acceleration of the arm. Moreover, it is assumed that the shape of acceleration and velocity traces is the same for movements with different amplitude and MT except for a scaling in amplitude and time.

Velocity $v(t)$ of a movement is related to acceleration $a(t)$ by equation A1:

$$v(t) = \int_0^t a(\tau) d\tau + v_0$$  \hspace{1cm} (A1)

where $v_0$ is the velocity at time $t = 0$. Position $s(t)$ during the movement is related to velocity by a similar integral equation:

$$s(t) = \int_0^t v(\tau) d\tau + s_0$$

$$= \int_0^t \int_0^\tau a(\sigma) d\sigma d\tau + v_0 t + s_0$$  \hspace{1cm} (A2)

where $s_0$ represents the beginning position at time $t = 0$. For the arm movements in this study beginning velocity is zero: $v_0 = 0$.

If the acceleration $a(t)$ is multiplied by a factor $\lambda$, the new movement amplitude $s'(t) - s_0$ increases by $\lambda$:

$$s'(t) - s_0 = \lambda \int_0^t \int_0^\tau a(\sigma) d\sigma d\tau$$

$$= \lambda \int_0^t \int_0^\tau a(\tau) d\tau d\tau$$

$$= \lambda \cdot (s(t) - s_0).$$  \hspace{1cm} (A3)

If the acceleration $a(t)$ is speeded up in time ($a'(t) = a(\beta t)$), then the new movement amplitude is given by:

$$s'(t) - s_0 = \frac{1}{\beta} \int_0^\beta \int_0^\beta a(\tau) d\tau d\beta$$

$$= \frac{1}{\beta^2} [s(\beta t) - s_0].$$  \hspace{1cm} (A4)

So for a time scaling by a factor $\beta$ the acceleration will increase with the square of $\beta$ but the duration of the acceleration has to be $\beta$ times shorter.

By combination of a scaling of acceleration both in amplitude and time

$$s'(t) - s_0 = \frac{1}{\beta} \int_0^{\beta t} \int_0^{\beta \tau} a(\beta \sigma) d\sigma d\tau$$

$$= \frac{1}{\beta^2} [s(\beta t) - s_0].$$  \hspace{1cm} (A5)
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In summary, when acceleration is multiplied by a constant, movement amplitude will be amplified by the same constant. If the acceleration is scaled in time by a constant, movement amplitude will decrease with the square of this constant. Consequently, if a movement with fixed amplitude is made twice as fast, the acceleration (and, therefore, the force required) has to be four times larger. More generally, for movements with amplitudes S1 and S2, and with movement times MT1 and MT2, the acceleration signal of the second movement will coincide with that of the first movement after scaling in time by a factor of (MT2/MT1) and after multiplication by a factor (MT1/MT2)(S1/S2).

In order to make the velocity signals coincide, the velocity signal of the second movement has to be scaled in time by a factor (MT2/MT1) and has to be multiplied by the factor (MT1/MT2)(S1/S2).

Relation between EMG and movement parameters

In this approach, it is assumed that EMG is a measure of isometric force: \( \text{EMG}(t) = \alpha F(t) \). This linear relation has been found in numerous studies e.g., Lippold (1952); Bigland and Lippold (1954).

In a linear approximation, forces during normal limb movements can be divided into three components:
1. an inertial component \( F(t) = m a(t) \), necessary for acceleration and deceleration of the limb. The parameter \( m \) represents the mass.
2. a viscous component \( F(t) = B v(t) \), necessary to overcome friction. The parameter \( B \) represents the linear viscosity.
3. a stiffness component \( F(t) = k_s(t) \), necessary to compensate for spring-like properties.

1. For fast movements, inertial force components will be dominant over viscous and stiffness components. Therefore, the integral of EMG over the time \( T \) (T is duration of the first agonist burst) is given by

\[
\int_{-\Delta t}^{T} \text{EMG}(t) \, dt = \int_{0}^{T} \alpha m a(t) \, dt
\]

\( \Delta t \) is the delay between EMG and force and is about 70 ms (see Gielen, van den Heuvel & Denier van der Gon, 1984).

Equation A7 shows that for high peak velocities (short MT's) the integral of the first agonist burst should be proportional to peak velocity. If the inertial mass is increased twofold by adding a weight, the slope of the linear relationship with peak velocity increases twofold.

2. For lower velocities (longer MT's), inertial components may be neglected and viscous and stiffness components are more important. These are given by:

\[
\int_{-\Delta t}^{T} \text{EMG}(t) \, dt = \int_{0}^{T} \alpha B v(t) \, dt
\]

\( \alpha B \) \( s(T) - s(0) \) \n
and

\[
\int_{-\Delta t}^{T} \text{EMG}(t) \, dt = \int_{0}^{T} \alpha k s(t) \, dt
\]

\( \alpha k \) \( s(T) \) \( s(0) \) \n
The viscous and stiffness components do not depend on mass and, therefore, these force components should be the same for movements made with different inertial mass.
Characteristics of Aimed Arm Movements

Because of the viscous and stiffness components, integrated EMG will increase with peak velocity for low velocities but not according to a straight line. Due to the small values of viscosity and stiffness (see Discussion), the integrated EMG will rise very slowly for low peak velocities and will increase more steeply when the inertial component becomes larger than the stiffness and viscous components. Over the whole range the relation will have a shape similar to a quadratic relation, as has been found by Lestienne (1979).

REFERENCES


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