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Rapid goal-directed elbow flexion movements: limitations of the speed control system due to neural constraints*

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Summary. In rapid goal-directed elbow flexion movements the influence of both movement amplitude and inertial load on the three-burst pattern and the consequences on movement time were studied. Subjects performed visually guided, self-paced movements as rapidly and as accurately as possible. An increase of both the movement amplitude and the inertial load were found to be interacting factors for the modulation of the three-burst-pattern and movement time. The first biceps burst progressively increased in duration and amplitude for larger movements, resulting in prolonged movement times. Surplus inertial loads further prolonged the agonist burst for large, but not for small movement amplitudes. The activity of the antagonist burst, in contrast, was largest in small movements and successively decreased at increasing movement amplitudes. Its duration, however, remained fairly constant. As was similarly observed for the agonist burst, surplus inertial loads lead to a prolongation of antagonist burst duration and an increase of the activity integral for large, but not for small movement amplitudes. It is suggested that in elbow flexion movements the programming of fastest goal-directed movements must take into account neural constraints and biomechanical characteristics of the agonist muscle and the antagonist muscle. Due to neural constraints of the biceps muscle, in contrast to finger movements, the concept of movement time invariance does not hold for elbow movements. Furthermore, neural constraints of the antagonist muscle lead to a limited force production of the agonist muscle at small movement amplitudes in order to avoid an overload of the braking process. The complexity of the relationship between neural and mechanical factors indicate that the size and timing of the three-burst-pattern has to be subtly adjusted to the precise nature of the task and its biomechanical characteristics.

Key words: Three-burst pattern – Rapid goal-directed movements – Speed control – Braking process

Introduction

The electromyographic analysis of rapid (ballistic) goal-directed movements in man disclosed a distinctive triphasic activity pattern in a wide variety of antagonistic muscle pairs (Wacholder 1923; Hufschmidt 1952; Hopf et al. 1973; Angel 1974; Hallett and Marsden 1973; Hallett et al. 1975; Brown and Cooke 1981; Day and Marsden 1982; Marsden et al. 1983). Hallett et al. (1975) postulated that the timing of this pattern was constant and that movements of different amplitudes were achieved by varying only the size of the bursts. This concept was consistent with the finding that rapid movements of different amplitudes were performed with the same movement time (Freund and Büdingen 1978).

However, now a large number of studies is available, which convincingly demonstrate that the triphasic EMG pattern is modifiable by unpredictable changes of peripheral inputs prior to and during the movements (Marsden et al. 1976; Adam et al. 1976; Hallett and Marsden 1979; Cooke 1980; Mortimer et al. 1981; Day and Marsden 1982; Day et al. 1983). Furthermore, in contrast to the study of Hallett (1975), which focused on relatively small amplitude movements, it was recently demonstrated (Brown and Cooke 1984; Berardelli et al. 1984) that the timing of the three-burst-pattern indeed could be modified by the movement amplitude alone. When large movements were made agonist burst duration

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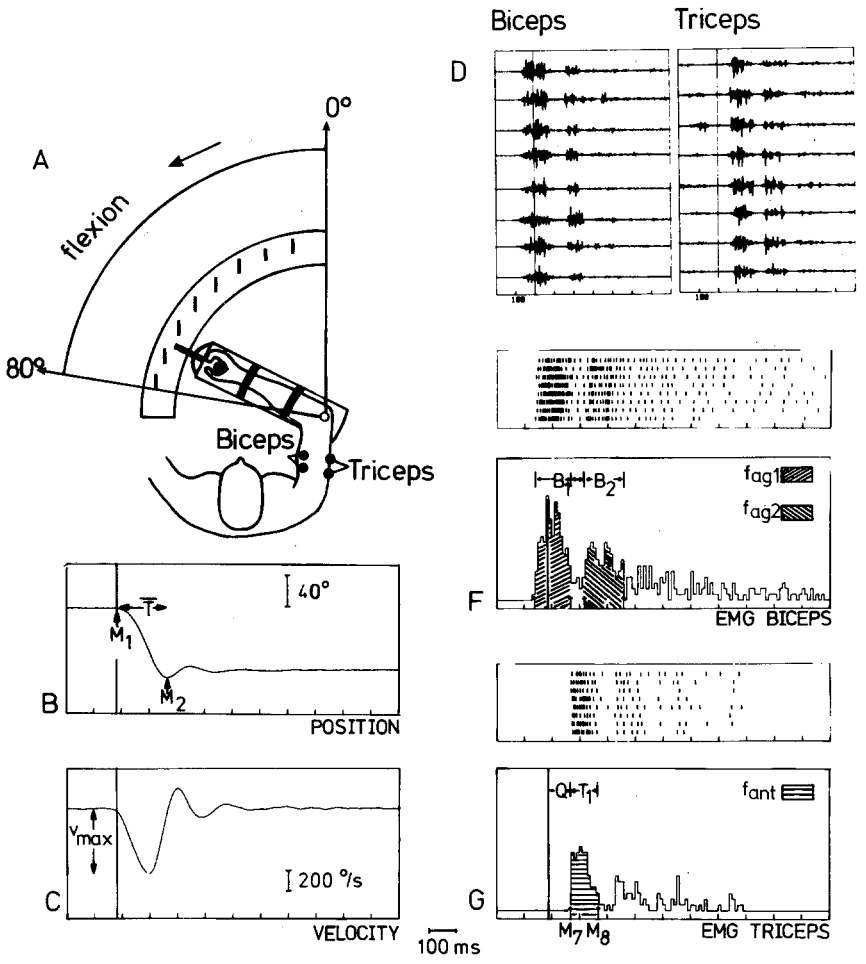

Fig. 1A–G. Schematic illustration of the experimental arrangement **A** and computer-analysis of the movement trajectories and the three-burst pattern. Representative data from an 80°-movement. The position **B**, the angular velocity **C**, the histogram of the biceps-EMG **F** and of the triceps-EMG **G** are averages from 8 consecutive movements. From the position signal **B** the movement time (T) was calculated after definition of the onset (M_1) – also indicated by the vertical lines – and the end (M_2) of the first rapid phase of the movement. From the velocity curve **C**, v_{max} was derived. **D and E** show original registrations from the biceps **D** and the triceps **E** muscle (time scale 100 ms). From the histogram in **F**, onset and end of the two biceps bursts were defined and the integrals (fag 1 and fag 2) calculated over this duration. The same procedure was performed in **G** for the triceps muscle. Besides the duration of the triceps burst (T_1) and the corresponding integral (f_{ant}), the latency from movement onset (M_1) to onset of triceps burst (Q) was measured. The raster plots at the top of the histograms – obtained after amplitude-frequency-conversion of the rectified EMGs – show the activity in individual sweeps. Time calibration applies to **B, C, F,** and **G**. Individual traces were aligned for averaging to a position trigger signal elicited at 2°

increased. The size and the timing of the burst appear to be subtly adjusted to the precise nature of the task by a highly versatile programme (Brown and Cooke 1981; Marsden et al. 1983; Meinck et al. 1984).

If the timing of the EMG bursts varies for different movement conditions then movement time may be expected to vary also. The new findings, therefore, question the long held general concept of movement time invariance. It was the aim of the present study, therefore, to look at the consequences of changes in the timing of the three-burst-pattern on movement time. The data show that application of inertial loads and variations of movement amplitudes lead to pronounced modifications of both the amplitude and the mutual timing of the three-burst-pattern together with changes in movement time. Neural constraints apparently play an important role for the performance of fastest goal-directed movements.

Methods

Experiments were performed on 16 right-handed normal volunteers between 20 and 45 years of age. The movements studied were flexions of the right forearm. Subjects were seated comfortably and grasped a vertical rod, which was attached to a manipulandum pivoted above the elbow. The upper arms were supported horizontally at the level of the shoulder. The forearms were semiprone. Angular position was measured with a linear potentiometer connected to the pivot point by means of a V-belt. The angular velocity was obtained by electronic differentiation (time constant 1 ms). Subjects were instructed to flex their elbows as rapidly and as accurately as possible to visible target zones.

The visual cues consisted of metal pins, 6 cm in length and 1 cm in diameter. The pins were vertically attached at angular distances of 10° on a plastic arc. Another pin (2 mm in diameter) was attached horizontally to the top of the manipulandum in order to exactly define the angular positions. When the target was accurately reached the two pins were 1 cm apart. All flexion movements were self-paced, performed at amplitudes of 20, 40, 60 and 80° (Fig. 1). During the movements the subjects had to continously look at the target pins. The inertial load of the moveable system was increased by the addition of weights (500, 1000, 1500 g) at a distance of 30 cm from the rotation axis, resulting in total inertial loads between approximately 0.16 kgm^2 and 0.41 kgm^2. Variations of movement amplitudes and inertial loads led to 12 various sets of trials. Each set consisted of 20 consecutive flexion movements, only the last 8 were recorded. Surface electromyograms (EMGs) were recorded from the biceps and triceps muscles. The individual EMGs were full-wave rectified, digitized and averaged together with angular position, velocity and acceleration, using a PDP-11 computer with a sampling rate of 500 Hz. The EMG averages were transformed to

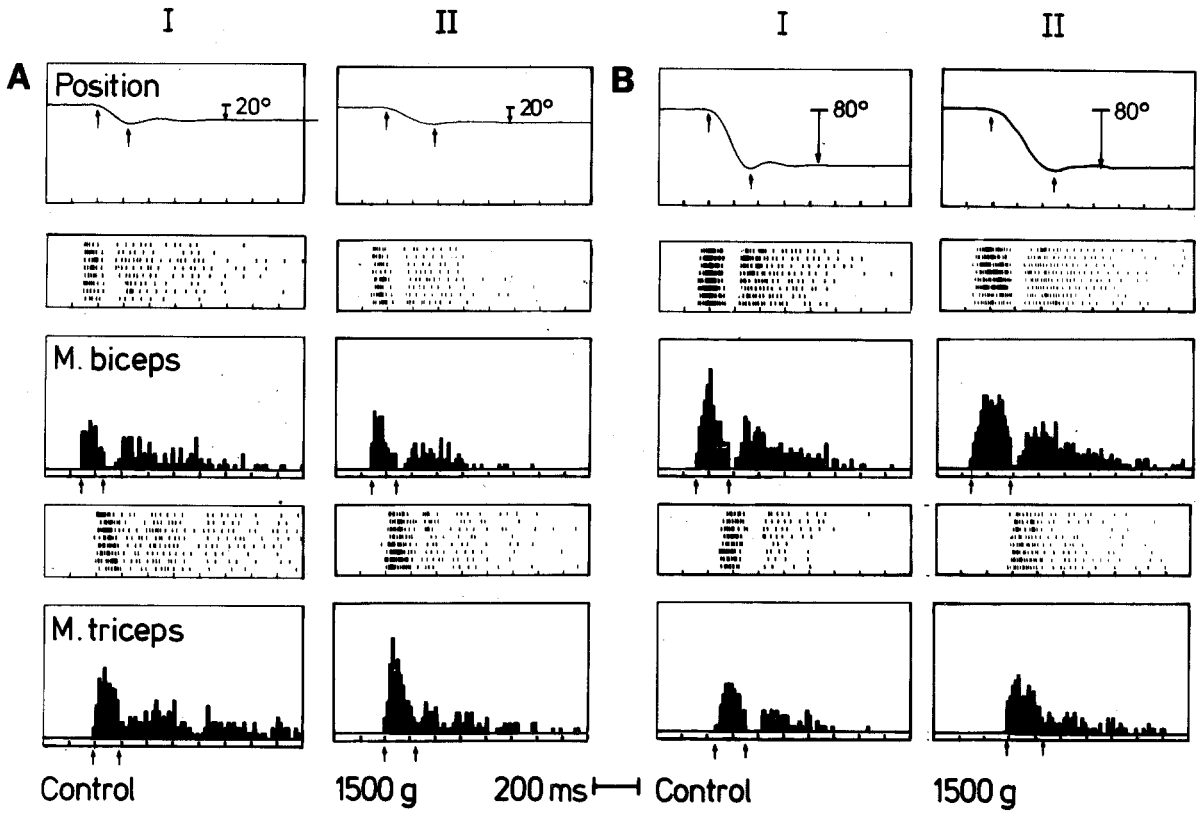

Fig. 2A and B. Effects of movement amplitude and inertial load on the three-burst pattern. **A** shows the position signals, raster plots, and the average-histograms of the biceps and triceps muscle in a control situation (I) and after application of an inertial load (II). **B** shows an 80°-movement in a control situation (I) and after application of an inertial load (II). The arrows in the various registrations indicate onset and end of the flexion movement and of the EMG-bursts, respectively. Time calibration at the bottom is valid for all registrations

histograms and raster plots (bin width 10 ms) in order to demonstrate the regularity of innervation patterns in a compact fashion. Times and burst durations were obtained by visual inspections of the records. By means of a cursor programme the movement time (T), the maximal velocity (Vmax), the durations of the two agonist bursts (B_1, B_2) and the antagonist burst (T_1) and the latency from movement onset to the onset of the antagonist burst (Q) were calculated. Furthermore, the EMG integrals of the agonist bursts and the antagonist burst were measured in arbitrary units (Fig. 1).

Results

A typical example of the modulations of the three-burst pattern with increasing inertial loads and movement amplitudes is shown in Fig. 2. The movements were performed with an initial EMG burst separated by a period of relative silence from a second burst. The antagonist burst starts during the declining part of the first agonist burst (AI, II; BI in Fig. 2), and ceases during the rising part of the second agonist burst. The onset of the first agonist burst occurred approximately 50–70 ms prior to movement onset. As can be seen in this figure, the second agonist burst can hardly be separated from a tonic after-activity occurring in both muscles. Therefore a systematic quantitative analysis of this burst appeared to be of low value.

When first the effects of movement amplitude on the three-burst pattern are considered, the following observations can be made (Fig. 2, A,I versus B,I): Both the amplitude and the duration of the first agonist burst increase; the amplitude of the antagonist burst, however, decreases at the larger movement amplitude, its duration remains fairly constant; although the velocity of the movement clearly increases with amplitude, the movement time at an amplitude of 80° is longer than at 20°. The onset of the antagonist burst in a movement of 80° is delayed as compared to a 20°-movement (see also Fig. 5, lower part).

Figure 2 further demonstrates the effects on the three-burst-pattern induced by an increase of the moment of inertia by means of adding weights (A,I versus A,II; B,I versus B,II). At the 20°-movement hardly any change of the EMG pattern can be observed, although the movement time clearly increased. In contrast, at the 80°-movement the

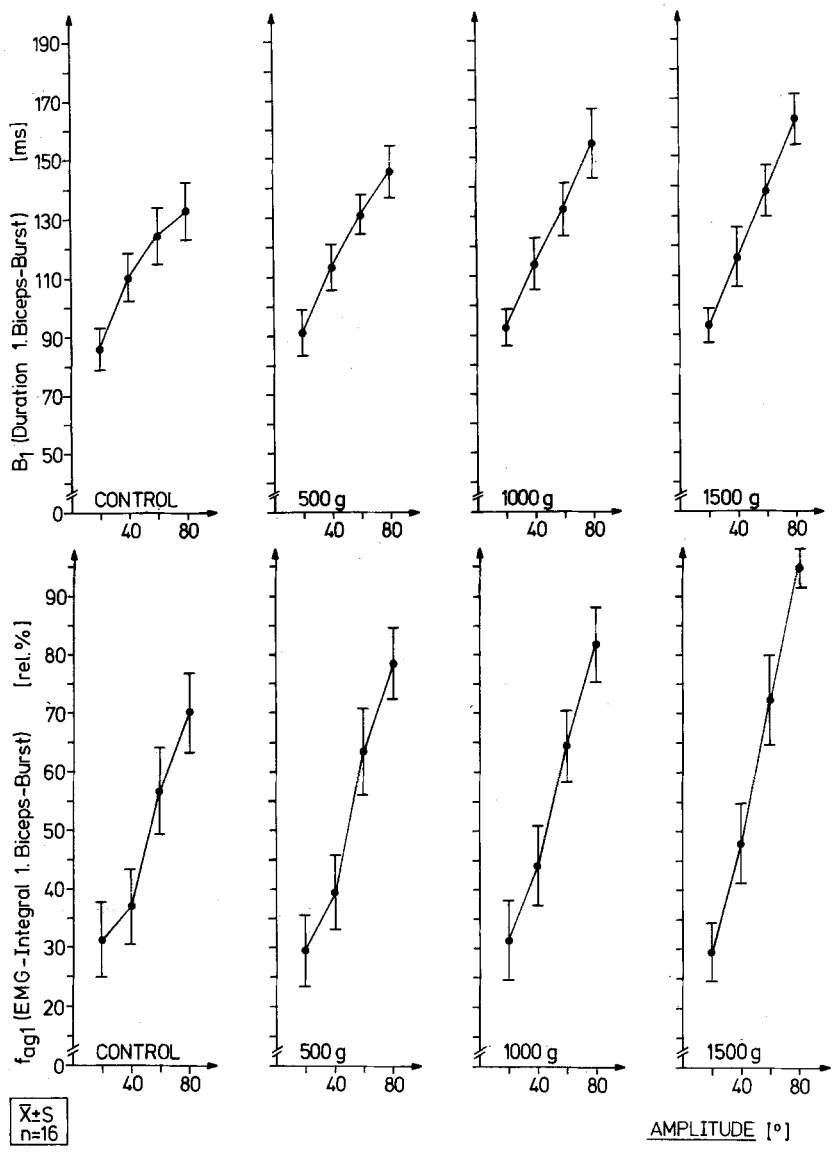

Fig. 3. Effects of movement amplitude and inertial load on the duration (top) and the integral (bottom) of the first agonist burst. Mean values and standard deviations of 16 subjects. The EMG-integrals are expressed as percentage values, 100% being defined as the maximal average EMG-integral obtained in the experiment. This usually occured at an 80°-movement against an inertial load of 1500 g

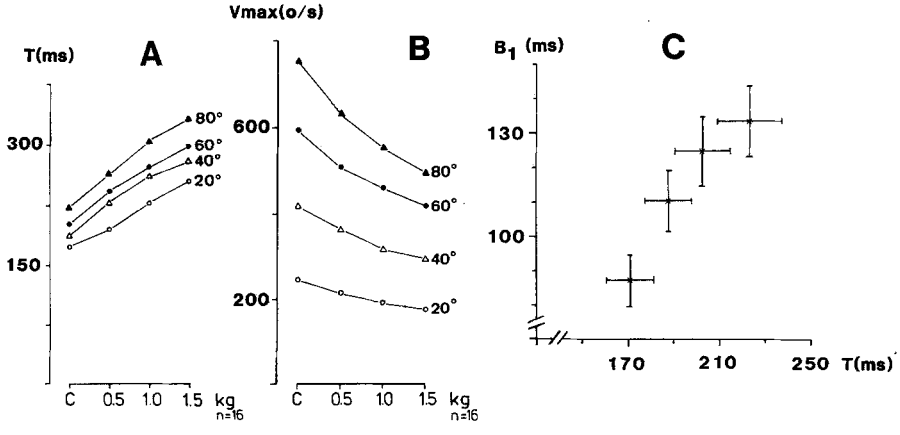

Fig. 4A–C. Interactions between agonist burst duration and movement parameters. **A and B** Effects of movement amplitude and inertial load on movement time (**A**) and maximal angular velocity (**B**). **C** Proportional relationship between agonist burst duration and movement time. Each point is the mean (±SD) agonist burst duration plotted against the mean (±SD) movement time obtained from 16 subjects at increasing (20°–80°) movement amplitudes

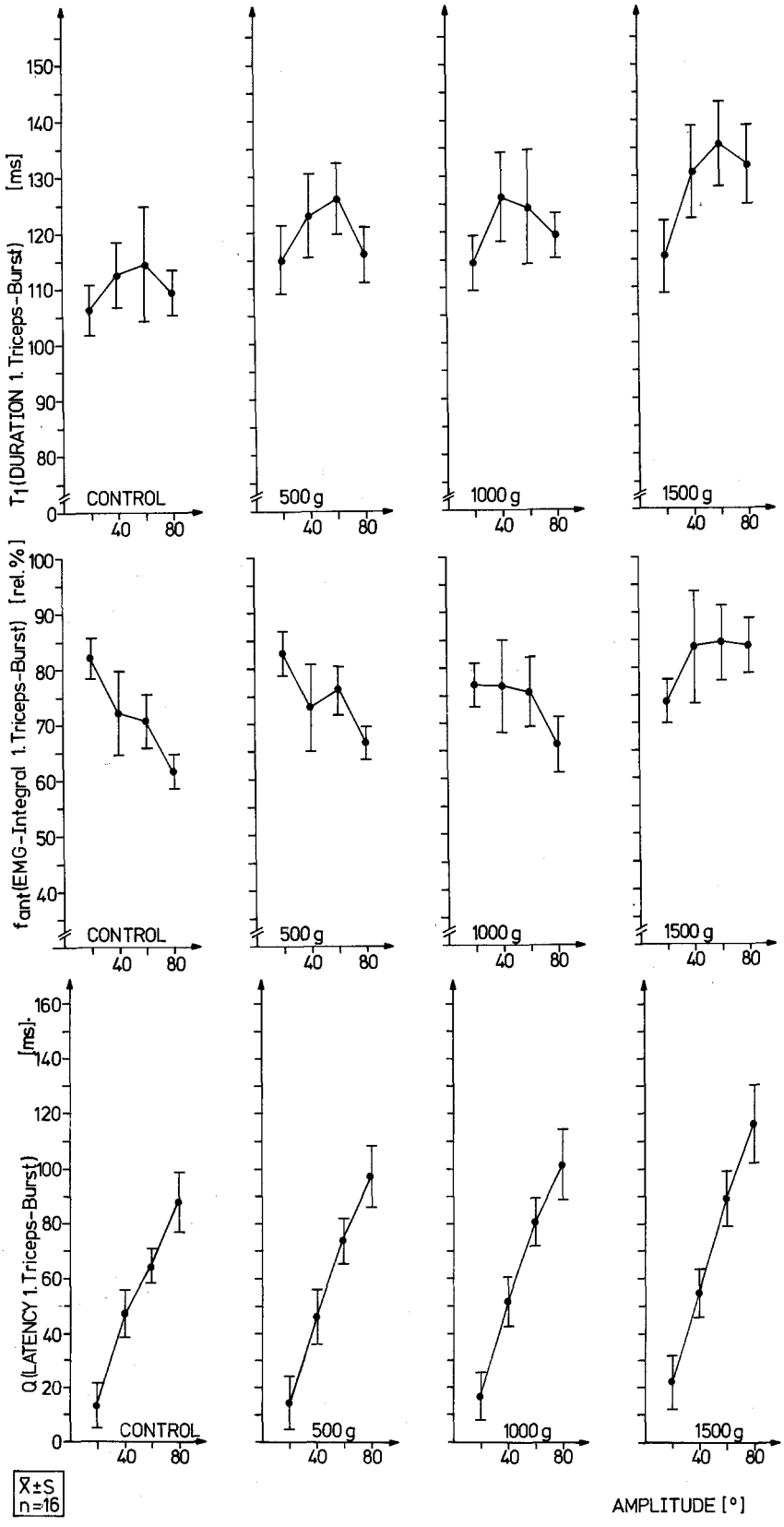

Fig. 5. Effects of movement amplitude and inertial load on the duration (top), the EMG-integral (middle), and latency (bottom) of the antagonist burst. Standardization of EMG-integral as in Fig. 3

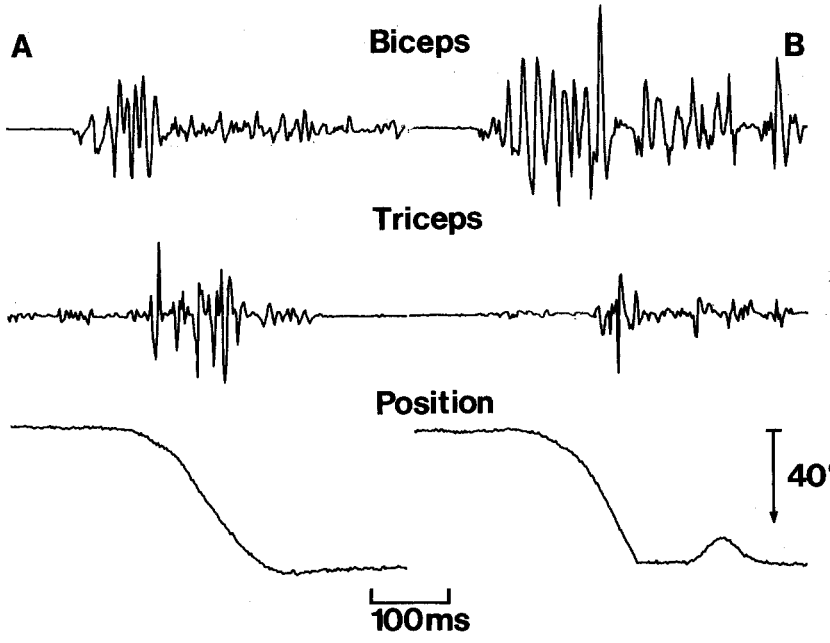

Fig. 6A and B. Limitation of agonist force production in a rapid goal-directed movement (**A**) as compared to a movement against a mechanical halt (**B**)

duration of both the first agonist and the antagonist burst increases at augmented inertial load, combined with a decrease of overlap; nevertheless again movement time is increased (Fig. 2; BII). It is also noteworthy that the shape of the first agonist burst is changed. Inspection of single sweeps showed that this change of shape is due to an intermittent segmentation of the first agonist burst combined with its prolongation. This phenomenon was also recently described by Brown and Cooke (1984).

Figure 3 demonstrates the changes of the duration and the EMG integral of the first agonist burst under all experimental conditions investigated. The duration of the first agonist burst and the movement amplitude show a clear proportional relationship. Increasing the inertial load causes a further increase in burst duration only at larger movement amplitudes. The integrals show similar changes. From a comparison of the duration and the integrals obtained in the various sets of trials, it can be derived that the augmentation of the integrals is not only induced by a prolongation of the burst but also by enhanced activity amplitude.

Figure 4 summarizes the effects on both movement time and maximal velocity in 16 subjects. Although the subjects are instructed to move as rapidly as possible the movement time is not kept constant but increases with both inertial load and amplitude (part A). Maximal velocity distinctly increases with movement amplitude but decreases with inertial load (part B). In Fig. 4 (part C) also the mean movement times and the duration of the first agonist burst are compared. It can clearly be seen that a proportional relationship between these two parameters exists. From these data it can be suggested that the duration of the first agonist burst determines movement time.

Figure 5 comprises the influence of movement amplitude and inertial load on the antagonist burst. The duration of this burst somewhat increases with inertial load, the duration versus movement amplitude-curves show a consistent non-linearity (concave downward). The EMG-integral, on the other hand, shows an inverse relationship with the movement amplitude in the control situation, which is successively lost or even reversed at increasing inertial loads (1000 g, 1500 g). The latency of the antagonist burst (Q; for definition see legend of Fig. 1) increases with movement amplitude, consequently the antagonist burst finds the triceps muscle in a more and more stretched position.

Figure 6 gives an example of the interaction of the first agonist burst and the braking antagonist burst. In this experiment, the elbow flexion movement was performed against a mechanical halt. In this condition, in which a braking activity by the antagonist burst is not necessary, both the amplitude and the duration of the first agonist burst were increased. This finding showed that in goal-directed movements, although to be performed as rapidly as possible, only a submaximal power of the agonist was energised, obviously because the braking capacity of the antagonist has to be taken into account.

Discussion

In this study self-paced visually guided elbow flexion movements had to be performed as rapidly and as accurately as possible. In contrast to other investigations about goal-directed movements, the targets were presented as real targets in space and not displayed as bars on an oscilloscope (Hallett et al. 1975; Hallett and Marsden 1979; Brown and Cooke 1981; Brown and Cooke 1984; Berardelli et al. 1984).

It was the aim of the present study to analyse the movement performances in rapid goal-directed movements when both the movement amplitude and the moment of inertia were changed. Special attention was paid to the modulation of the EMG-pattern and the movement times. In such movements an increase of both the movement amplitude or the moment of inertia require higher force production. This is true for the accelerating agonist activity, as well as for the decelerating antagonist activity (torque = moment of inertia × angular acceleration). As was observed in fastest voluntary goal-directed movements of the hand and forearm muscles (Freund and Büdingen 1978; Hallett and Marsden 1979), an increase of the amplitude in such movements is automatically combined with higher angular velocity and acceleration, thereby keeping the movement time constant (speed control system; Freund and Büdingen 1978). However, our investigations show that for elbow movements this speed control system is not as effective as in the distal muscles: the movement time is not kept constant at increasing movement amplitudes, although the movement velocity is enhanced. Correspondingly, the first agonist burst was progressively prolonged with increasing movement amplitude (see also Brown and Cooke 1984; Berardelli et al. 1984), suggesting that this parameter determines the movement time. Such difference between distal and proximal muscles is probably due to the fact that the biceps brachii must overcome larger inertial loads, i.e. the mass of the forearm. It can be assumed that at movements with large amplitudes and high acceleration, the recruitment of motor units in a restricted time-span is insufficient to produce the flexion force required. This limitation is compensated for by a prolonged recruitment time. Such a neural constraint appears even more distinct if the inertial load is augmented by means of adding weights (Fig. 2; B,II). Especially in this particular situation, an increase in the duration of the first agonist burst was occasionally produced by the appearance of two phasic components within the first agonist burst. A further mechanism apparently compensating for the neural constraints of the biceps at high force production is the release from overlap with the antagonist muscle.

However, modulation of the agonist burst duration was also observed at low movement amplitudes even without inertial loads (Fig. 3). In this case neural constraints of the antagonsit burst seem to play an important role in the genesis of this phenomenon. The antagonist burst surprisingly produces lower activity at larger movement amplitudes, which clearly contrasts to the agonist burst. Viscoelastic properties of the active antagonist muscle, tendons and joints which contribute to the braking process (Lestienne 1979) are not linear over the range of passive extension, but distinctly increase at extreme elbow flexion. Consequently, less antagonist EMG activity is required when the joint is moved over large amplitudes than when it is moved over small amplitudes (cf. Marsden et al. 1983). According to this mechanical influence, the overall braking capacity is more likely limited at low amplitude movements. Consequently, in order to adjust the performance of the movement to the limited capacity of the braking process, lower acceleration forces are apparently used, resulting in lower torques to be counteracted by the antagonist. This strategy can even better be demonstrated when surplus inertial loads are added. In this situation, the resulting increase of the moment of inertia is compensated for by further decrease in angular velocity (see Fig. 4; part B) in order to keep the torque counteractable. The complexity of these relations indicates that the size and timing of the EMG-pattern has to be subtly adjusted to the biomechanical aspects and neural constraints of the muscles involved. The agonist muscle in fastest goal-directed movements only exerts forces which can be braked by the antagonist muscle. Such a dependence of the agonist burst on the capacity of the braking process can clearly be demonstrated when goal-directed movements are compared with movements against a mechanical halt (see Fig. 6). In the latter condition, the agonist muscle is able to exert much higher forces, because any limitations of the braking capacity can be neglected.

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