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## Interaction of discrete and rhythmic movements over a wide range of periods

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**Abstract** This study investigates a complex task in which rhythmic and discrete components have to be combined in single-joint elbow rotations. While previous studies of similar tasks already reported that the initiation of the discrete movement is constrained to a particular phase window of the ongoing rhythmic movement, interpretations have remained contradictory due to differences in paradigms, oscillation frequencies, and data analysis techniques. The present study aims to clarify these findings and further elucidate the bidirectional nature of the interaction between discrete and rhythmic components. Participants performed single-degree-of-freedom elbow oscillatory movements at five prescribed periods (400, 500, 600, 800, 1000 ms). They rapidly switched the midpoint of oscillation to a second target after an auditory signal that occurred at a random phase of the oscillation, without stopping the oscillation. Results confirmed that the phase of the discrete movement initiation is highly constrained with respect to the oscillation period. Further, the duration, peak velocity, and the overshoot of the discrete movement varied systematically with the period of the rhythmic movement. Effects of the discrete-onto-rhythmic component were seen in a phase resetting of the oscillation and a systematic acceleration after the discrete movement, which also varied as a function of the oscillation period. These results are interpreted in terms of an inhibitory bidirectional coupling between discrete and rhythmic movement. The interaction between discrete and rhythmic movement elements is discussed in comparison to sequential and gating processes suggested previously.

**Keywords** Rhythmic movement · Discrete movement · Tremor · Mutual inhibition · Coupling

### Introduction

Continuous rhythmic actions, such as walking or running, and discrete actions, such as pointing or reaching to a target, are commonly recognized as two important classes of behavior in the literature. Studies on the control and coordination of movements have frequently investigated either one or the other class of movement and proposed insights into the control of either rhythmic or discrete movements. Rarely has the combination of the two been the focus of interest. Yet, many everyday tasks are complex in that they involve a combination of both rhythmic and target-oriented actions. We walk while we reach out our hand to greet somebody, and we place our footstep over an obstacle while we continue our cyclic stepping actions. In piano playing, we rhythmically move our fingers to strike the keys, while we translate our hands across the keyboard to reach the desired keys. This co-occurrence of rhythmic and discrete elements also exists at another spatio-temporal level of behavior: Oscillations are continuously present in the form of tremor, both in clinical as well as in healthy populations, and any targeted action such as the initiation of a pointing action has to occur against the backdrop of this rhythmic activity. The question that arises is how are these two types of movements combined?

The interaction between discrete and rhythmic actions has in fact been primarily addressed in the research on tremor. The first studies that focused on this interaction looked at voluntary movements performed against the background of tremor. The converging result was that the initiation of the voluntary movement appeared to be confined to a certain phase window of the tremor oscillation. Travis (1929) reported that upward finger movements seemed to be initiated during the upward phase of the tremor movement cycle, whereas downward movements occurred in the downward phase of tremor cycle (8–12 Hz). Similarly, Hallett et al. (1977) observed a variable time delay in Parkinson patients when they initiated a voluntary elbow rotation, suggesting that this delay came from waiting for a preferred phase of the

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tremor cycle. Goodman and Kelso (1983) studied a rapid finger extension task in healthy subjects with physiological tremor (8–12 Hz). They reported that the initiation of the discrete movement occurred most frequently at the peak of angular velocity in the tremor oscillation, equivalent to the moment of maximum momentum in the direction of the discrete movement. This particular moment represents the most advantageous time to initiate the voluntary movement as momentum can be exploited. From a more general point of view, the authors suggested that the perceptual-motor system is sensitive to its own dynamics and capitalizes on this opportune moment to initiate a voluntary discrete action.

This interpretation was not shared by Wierzbicka et al. (1993) or Elble et al. (1994) who reported opposite results. Wierzbicka et al. (1993) examined Parkinson patients (2–5 Hz) in an isometric finger abduction task, and Elble et al. (1994) studied patients with essential tremor (4–8 Hz) in a wrist flexion task. Both studies found that the initiation of the discrete action occurred predominantly during the phase where the tremor movement was in the direction opposite to the discrete action. In other words, they found that the tremor impeded, rather than assisted the discrete action.

As Elble et al. (1994) conjectured, these conflicting results may be the consequence of the difference in the types and especially frequencies of tremor, i.e., Parkinson tremor versus physiological or essential tremor. Further, the tasks involved movements that were either ballistic or isometric. Also important is that different data analysis methods were used to identify the initiation of the discrete action and relate it to the oscillatory component. Goodman and Kelso (1983) determined the onset of the discrete action and the tremor rhythm on the basis of kinematic signals, whereas Wierzbicka et al. (1993) conducted their analyses on the isometric force signal and the EMG signal. Elble et al. (1994) determined the onset time from the kinematics and the tremor rhythm from the electromyographic (EMG) bursts. Taken together, these numerous differences make it difficult to compare results and resolve the conflicting conclusions. This is especially relevant since, as Elble et al. (1994) emphasized, the phase relationship between EMG activity and kinematics may be different depending on the tremor frequency.

However, amidst these differences, the results that were obtained from EMG signals were less contradictory across the various studies. The EMG-based results seemed to agree that the EMG burst responsible for the discrete action tended to occur at, or slightly before the burst associated with the rhythmic component. This result is indicative that a common principle for these constraints could be the reciprocal inhibition between antagonistic units: while the extensor is active for the rhythmic contraction, the discrete flexion is delayed until the flexor is activated as part of the rhythmic movement.

Importantly, this result was also maintained when discrete movements were performed against the background of voluntary oscillatory movements, as examined

by Adamovich et al. (1994) and Sternad et al. (2000). These two studies designed a movement task that involved the simultaneous activation of voluntary discrete and rhythmic actions in a single-joint movement at frequencies considerably slower than tremor (2–5 Hz) and performed at larger amplitudes. Such constraints between discrete and rhythmic action components were interpreted and modeled by Sternad and colleagues in terms of an inhibitory coupling between the rhythmic and the discrete units. Inhibitory coupling means that, for instance, when the extensor muscle is active, the flexor is inhibited and cannot be activated for the discrete flexion movement. The beginning of the discrete movement is suppressed until the flexor is activated as part of the rhythmic movement. Adamovich and colleagues proposed a sequential activation of the two components. These interpretations contrasted with Goodman and Kelso's account that the motor system explicitly took advantage of the dynamics of the system.

The present study addresses the nature of the observed interactions between discrete and rhythmic movements by examining a single-joint task that combines voluntary discrete and rhythmic elements performed over a wide range of frequencies. By this manipulation we aim to reveal to what extent the differences in results are due to different oscillation frequencies. This is motivated by observations reporting that the relative timing between EMG activity and kinematics in rhythmic movements are frequency-dependent (Wachholder and Altenburger 1927; Sternad 2000). By analyzing the initiation and interaction of rhythmic and discrete movements, we aim to also clarify the conflicting results and interpretations due to different data analysis techniques. More specifically, if the constraint on the discrete movement's initiation is caused by an inhibitory coupling between the two control signals, the phase of the discrete movement's onset, determined from the EMG signal, should be less variable or invariant across different oscillation frequencies. If mechanical efficiency is the primary source of constraint on the discrete initiation, then the phase of the discrete movement's onset determined on the basis of the kinematic signal should be invariant (Objective 1).

The second interest in exploring a wide range of oscillatory frequencies is directed at further unraveling the nature of the interaction between discrete and rhythmic movements. The results on the phase-locking of the discrete action clearly suggest that the oscillatory movements have an inhibiting effect on the discrete movement. However, is the discrete action itself also affected by the ongoing rhythmic movements? In Adamovich et al. (1994) no effect of the rhythmic signal onto the discrete movement was identified. Further, the authors showed that once the discrete movement was started, the rhythmic movement was delayed, suggesting that the control signals were issued in sequence. Similar results of Sternad et al. (2000) were interpreted with a slightly different emphasis: the two control signals were proposed as being generated in parallel but an inhibitory mutual coupling between them led to the overt sequenc-

ing. While the discrete movement was in process, it temporarily inhibited the rhythmic movement, and the rhythmic phase was reset once the discrete movement was completed. These observations indicate a bidirectional interaction between discrete and rhythmic movements: not only does the rhythmic movement influence the discrete movement (e.g., discrete initiation), but the discrete movement also influences the rhythmic movement (e.g., phase resetting). By exploring a single-joint discrete movement performed against the background of many different rhythmic movements, i.e., over a wide range of periods, the second goal of this study is to further elaborate on the possible bidirectional interaction between discrete and rhythmic movements (Objective 2).

## Methods

### Participants

Five graduate students (four male, one female) from The Pennsylvania State University volunteered to be participants in this study. Their ages ranged from 25 to 39 years. All reported themselves right-hand dominant. None had any history of serious injury to their right arms. Prior to data collection, the participants were informed about the experimental procedure and signed the consent form in compliance with the University's Regulatory Committee.

### Experimental apparatus and data collection

The participant was seated in front of a table with his/her right forearm placed on a foam-padded horizontal metal support fixed to an axle. The height of the chair was adjusted so that the upper arm was horizontal and at the same height as the forearm. The center of rotation of the elbow joint was aligned with the axle of the apparatus. As the chest rested against the table, shoulder movements were minimized and elbow flexion and extension occurred in the horizontal plane. The participants grasped a vertical wooden handle fixed to the end of the arm support with their right hand. To ensure a fixed forearm position a Velcro band strapped the forearm moderately tightly to the arm support. Two targets were created by fixing thin 15-cm-long wooden dowels to a vertical cardboard surface. This target surface was curved and placed such that the distance between the curved path of the tips of the fingers and the target surface was 10 cm. Two dowels placed approximately 10 cm apart marked one oscillatory target. The first target (TG1) was positioned at an elbow flexion of 140°, where full extension was defined to be 180°. The minimum and maximum amplitude of the oscillation was indicated by the two dowels, which were approximately 20° apart (140±10°). The second target (TG2) was placed at 100±10° oscillation amplitude. A computer-generated metronome signal prescribed the oscillation periods for an initial pacing interval of 5 s (beep duration 50 ms, beep frequency 2500 Hz). After the initial pacing interval, a single trigger tone (200 ms, 4000 Hz) served as the stimulus to signal the onset of the discrete movement.

Joint angle position data were collected by an optical encoder (U.S. Digital H3-2048, Vancouver, Wash., USA) attached to the axle of the apparatus. The rotational resolution of the optical encoder was 0.044°. Sampling frequency was 200 Hz. EMG data was collected from one elbow flexor, the long head of the biceps brachii (BB), and one elbow extensor, the lateral head of the triceps (TL). The analog data were high-pass filtered with a cutoff at 10 Hz and were amplified with a gain of 5 K. The sampling frequency for the EMG was 400 Hz. The digitized signals were further low-pass filtered. (The relatively low sampling frequency for the EMG data was sufficient because the amplitude and frequency content of the

signal was not in focus. Further, there were no frequencies higher than 200 Hz in the signal so that aliasing was not a problem.) In addition to the EMG and kinematic signals, the auditory signal was recorded to provide information about the temporal onset of the stimuli. The collection of all signals was controlled by LabView Software (National Instruments, Austin, Tex., USA) on a Macintosh Computer (PowerCenter Pro 210, Power Computing).

### Experimental conditions

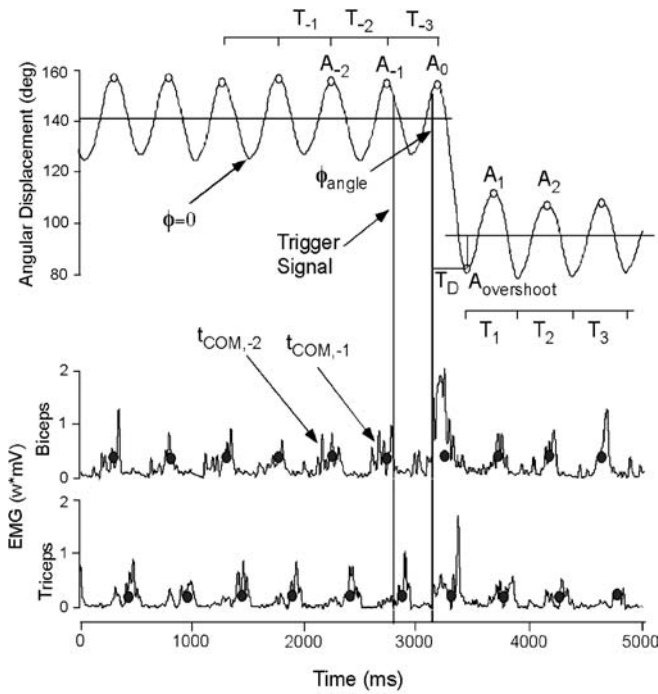
Before the beginning of each trial the participant extended his/her elbow to a mechanical stop that was located at full extension (180°). He/she was then instructed to begin oscillating around TG1 in synchrony with the auditory metronome, performing one full cycle per beep. The participant was instructed to coordinate maximum flexion excursions with the metronome beeps. After 5 s, the periodic metronome signal ceased but the participant was instructed to continue oscillating at the same frequency until he/she heard the imperative stimulus. This stimulus tone occurred after a random delay between 2 and 5 s after cessation of the initial pacing interval. The participant was instructed that upon hearing this tone he/she was to "... shift [his/her] forearm to TG2 as fast as possible, without stopping the ongoing rhythmic movements." This instruction emphasized that both the reaction to the signal and the speed of shift should be as fast as possible. The instruction was designed to force the participants to combine both the rhythmic and the discrete actions. The participant continued to oscillate around TG2 until another tone signaled the end of the trial. The participants were instructed to keep the amplitude of the oscillations at both targets bounded by the two wooden dowels, but accuracy of amplitude was not essential. No further instructions or feedback were given. Each trial lasted 15 s.

Five experimental conditions with five different oscillatory periods were presented. For the condition P1000 the prescribed period was 1000 ms, for P800, P600, P500, and P400, the oscillation periods had corresponding durations in milliseconds. One additional period condition P200 was presented but was eliminated from further analyses due to analysis problems (see below). Ten trials of each condition were performed by each participant. For each trial, the temporal onset of the imperative signal was issued at a random phase, and this random phase was drawn from a uniform distribution across oscillatory phases in the trials. The periods were presented in a fully randomized order.<sup>1</sup> The target oscillation amplitudes around each target remained the same for all five conditions. To limit the total number of trials and to prevent fatigue in the participants, only discrete flexion movements were performed. A previous experiment had not shown significant differences between discrete flexions and extensions in the same parameters measured in this experiment (Sternad et al. 2000). The duration of the entire experimental session was approximately 30 min.

### Data reduction and analysis

The joint angle data did not require filtering because the optical encoder signals were extremely smooth. The digitized EMG signals were rectified and filtered using a sixth-order zero-lag low-pass

<sup>1</sup> We constrained the target amplitude of the oscillation to one constant amplitude, even though it is generally observed that humans choose smaller amplitudes for slower periods and vice versa. The reason for a fixed amplitude was to ensure that different participants performed the oscillations with a comparable amplitude. However, the instruction to the subject did not emphasize accuracy for the amplitude and allowed for larger or smaller amplitudes if more convenient. To prescribe an oscillation amplitude that varies with the pacing frequency would have introduced more differences in the task conditions and would have been confusing to the subjects. The task itself is already a dual task and quite demanding on the concentration of the subjects.



**Fig. 1** A segment of a representative time series of joint angle data and EMG data from an elbow oscillatory movement trial with the adopted notation (only 5 s are shown as this is the window of interest). The first vertical line indicates the onset of the auditory stimulus signaling to the participant to shift the center of oscillation from target 1 to target 2. The second vertical line indicates the onset of the discrete movement as calculated from supra-threshold activation of biceps brachii. The short vertical lines in the EMG trace demarcate the segmentation of the EMG signal into cycle windows for the calculation of centers of mass (COMs), determined by the flexion minima in the joint angle trace ( $T$  cycle periods,  $A$  cycle amplitudes). Other dependent measures displayed are as defined in the Methods section

Butterworth filter with a cutoff frequency of 35 Hz. For the following analyses only the data of the biceps brachii was used. First, the timing of rhythmic burst activity during the rhythmic intervals was calculated. To this end, the EMG signals were divided into windows delineated by the joint angle excursions. The windows were defined at the times of the minima of the elbow trajectory such that each window contained the EMG burst of the rhythmic activity (see an exemplary trial in Fig. 1). Within each window, the time of the maximum activity of each burst was determined. This time was defined by the center of mass (COM) of the EMG signal within each window,  $t_{COM}$ . To obtain  $t_{COM}$ , the area under the rectified EMG curve was calculated using the trapezoid method of numerical integration, where  $t_{COM}$  was at the half-area point of the EMG signal within each window.

### Kinematic parameters

For the intervals both preceding and succeeding the discrete movement the cycle periods ( $T$ ) and amplitudes ( $A$ ) of the joint angular displacement were calculated. The cycles were indexed by negative integers,  $i = -1, -2, \dots$  before, and positive integers  $i = 1, 2, \dots$  after the discrete movement. To obtain average estimates for each trial three cycles preceding the discrete movement ( $T_{-3}-T_{-1}$ ) and three cycles following the discrete movement ( $T_3-T_6$ ) were averaged to obtain  $T_{pre}$  and  $T_{post}$  respectively. Similarly,  $T_{EMG}$  was calculated as the average time between successive  $t_{COM}$  of four EMG bursts in BB preceding and succeeding the discrete transition.

For the calculation of further dependent measures only  $T_{EMG}$  for the pre-discrete interval was used. The cycle amplitudes of the rhythmic intervals  $A_i$  were calculated as the half-amplitudes from the center of oscillation. The centers of oscillation before and after the discrete movement were computed as the average angular position of the elbow before and after, respectively. Subsequently,  $A_{pre}$  and  $A_{post}$  were calculated by averaging over three cycle amplitudes before  $A_{-1}$ , and three cycles after  $A_3$  to obtain amplitude estimates for pre- and post-discrete intervals.

### Amplitude overshoot

The angular excursion of the discrete movement,  $A_{overshoot}$ , was defined as the difference between the amplitude at the first inflection terminating the discrete movement, and the average center of oscillation of the post-discrete interval. Positive values correspond to an overshoot, negative values to an undershoot.

### Relative phase between kinematics and EMG

The phase of the EMG activity relative to the kinematics of the oscillation,  $\psi_{EMG-K}$ , was determined. The time differences between each  $t_{COM}$  and the following kinematic peak  $t_{-i}$  were calculated for five cycles prior to the discrete transition and converted into relative phase according to the equation:

$$\psi_{EMG-K} = \frac{2\pi}{5} \sum_{i=1}^5 \left( \frac{t_{COM,-i} - t_{-i}}{T_{pre}} \right)$$

### Onset of discrete movement

The temporal onset of the discrete movement,  $t_{onset}$ , was determined as the time when the EMG activity exceeded a predetermined threshold. This threshold was defined as the average peak activity value of the five bursts prior to the discrete movements plus one standard deviation. For condition P200, this algorithm selected an appropriate onset in only 30% of the total number of 60 trials across participants. As this dependent measure was central for many analyses, condition P200 had to be discarded from further analysis. Out of the remaining 300 trials, there were a total of 29 in which the EMG signal of BB did not show peak activity preceding the discrete movement that satisfied the above criteria. In these cases,  $t_{onset}$  was determined by inspection: the EMG burst associated with the discrete shift was identified and the initial rise in signal amplitude of this burst was designated  $t_{onset}$ . To verify the reliability of this manual method, two people independently performed the procedure and compared their results. The differences in the manually determined  $t_{onset}$  between the two people were of the order of 10 ms. In only 5 of 29 trials were these differences greater than 15 ms. These five trials were thus discarded from further analysis. As an additional evaluation, the manual procedure was performed on 20 random non-problematic trials. The differences between those manually determined values for  $t_{onset}$  and algorithmically calculated values were also on the order of 10 ms.

### Onset phase of discrete movement

The phase of  $t_{onset}$  relative to the oscillation was determined in two ways: (1) on the basis of the joint angle data  $\phi_{angle}$ , and (2) on the basis of the EMG data,  $\phi_{EMG}$ . To obtain  $\phi_{angle}$ , the difference between  $t_{onset}$  and the time of the valley,  $t_{valley}$ , immediately prior to the transition was calculated and converted to a phase by dividing by  $T_{pre}$ . Hence,  $\phi = 0$  rad for onsets at the valleys (see Fig. 1):

$$\phi_{angle} = \left( \frac{t_{onset} - t_{valley}}{T_{pre}} \right)$$

To obtain  $\phi_{EMG}$ , an analogous calculation was performed. In this case,  $\phi_{EMG}$  was calculated as the difference between  $t_{onset}$  and the  $t_{COM}$  immediately preceding the onset and divided by  $T_{EMG}$ .

Additionally, the phase difference between the EMG-based onset  $\phi_{EMG}$  and kinematic-based onset  $\phi_{angle}$  was calculated and defined as  $\psi_{onset}$  according to the equation:

$$\psi_{onset} = \phi_{EMG} - \phi_{angle}$$

#### Duration of the discrete movement

To quantify the duration of the discrete movement, the time measure  $T_D$  was defined between the time of the onset of the discrete movement,  $t_{onset}$  and  $A_{overshoot}$ . In addition, peak velocity of the discrete movement was determined as the maximum of the velocity signal after the time of initiation of the discrete movement.

#### Phase shift

The phase shift  $\Delta\phi$  induced by the discrete movement was calculated as follows:

$$\Delta\phi = 2\pi \left( \frac{t_1 - t_{-1} + T_{pre}}{T_{pre}} \right) \text{ mod } 2\pi$$

where  $t_{-1}$  refers to the peak in the kinematic signal immediately prior to the discrete transition, and  $t_1$  to the peak immediately after the transition. By adding one cycle period to the time difference, the oscillation was extrapolated forward in time as if unperturbed. Note that a negative value for  $\Delta\phi$  indicated that  $t_1$  occurred *earlier* than predicted by the unperturbed oscillation extrapolation, while a positive value indicated that  $t_1$  occurred *later* than predicted.

#### Analysis of variance

The data from each trial were submitted to either a 5 (period)  $\times$  2 (pre/post)  $\times$  5 (participant), mixed-design ANOVA, or a 5 (period)  $\times$  5 (participant) mixed-design ANOVA as necessary. Pre/post and period condition were within-trial factors while participant was a between-trial factor. The probability of type I error was selected as  $\alpha=0.05$ .

## Results

Figure 1 displays a section of a representative time series of joint angle data and EMG data of the BB and TL

together with the adopted notation for the kinematic variables.

Periods  $T_i$  and amplitudes  $A_i$  of the oscillatory interval before ( $i<0$ ) and after ( $i>0$ ) the discrete transition were calculated for each trial and average  $A$  and  $T$  pre and post the discrete movement were calculated for each period condition. An overview of average pre- and post-transition periods and amplitudes of the five participants for the five period conditions can be obtained in Table 1. Each value presented is the average over all ten trials performed at the respective period condition. Per instruction, the periods were close to the prescribed period. There was a tendency to accelerate the oscillation after the discrete movement, an effect further analyzed below. Most participants performed the oscillation with amplitudes that were close to the prescribed amplitude of  $20^\circ$ . However, a trend for an increase in amplitude for longer periods can be observed. This is in line with the general observation that humans oscillate with a smaller amplitude at shorter periods and vice versa. Only participant 5 performed the oscillations with noticeably smaller amplitudes although he maintained the appropriate period.

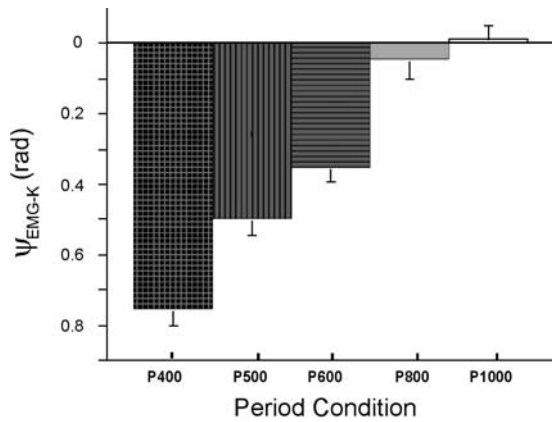
#### Relationship between the EMG and kinematic data during steady state oscillation

First the timing relationship between the EMG and kinematic signals was evaluated for the different period conditions. Relative phase  $\psi_{EMG-K}$  was determined as the difference between the centers of the EMG bursts and kinematic excursion maxima. A negative value for  $\psi_{EMG-K}$  corresponded to a phase lead of the EMG burst. Figure 2 reveals that  $\psi_{EMG-K}$  varied systematically with period condition: the EMG bursts showed a phase lead with respect to the kinematic maxima that decreased as the periods became longer. This effect of period condition on  $\psi_{EMG-K}$  was confirmed with a highly significant main effect in the  $5 \times 5$  ANOVA ( $F_{4,16}=20.48$ ,  $P<0.001$ ). Note, though, that the magnitude of the difference in the relative phase between the faster (400 ms) and the slower (1000 ms) periods was about 0.8 rad (approximately

**Table 1** Pre- and post-transition period and amplitude. The data displayed are means for each participant over the ten trials for the five period conditions, P400–P1000 (400–1000 ms). *Pre* refers to

the interval before the discrete movement, *Post* refers to the interval after the discrete movement

Participant	Pre/post	Period ( $T$ , ms)					Amplitude ( $A$ , deg)				
		P400	P500	P600	P800	P1000	P400	P500	P600	P800	P1000
1	Pre	395	488	582	763	964	23.4	24.7	23.9	26.7	26.6
	Post	384	467	557	721	834	22.0	21.8	22.3	24.7	23.8
2	Pre	394	483	582	793	994	21.1	22.1	23.2	24.5	24.6
	Post	389	475	558	742	915	21.1	19.9	21.2	21.4	22.3
3	Pre	384	497	570	768	1005	14.4	17.7	20.8	23.1	23.1
	Post	370	460	501	626	908	15.7	18.1	20.0	19.9	20.8
4	Pre	362	451	530	743	931	15.7	17.0	18.0	20.5	21.3
	Post	356	428	503	673	817	15.4	16.9	18.0	21.1	21.4
5	Pre	370	479	563	759	966	13.3	13.8	13.4	13.0	12.2
	Post	370	488	548	703	829	13.7	13.0	12.6	11.4	10.5



**Fig. 2** Relative phase between the EMG and kinematic oscillations ( $\psi_{EMG-K}$ ) during the pre-transition portion of the trials. The means and standard errors are displayed. The positive  $\psi_{EMG-K}$  indicates that the EMG burst occurred prior to the respective kinematic peak. The small phase lag of the EMG oscillation in the highest period condition (*P1000*, 1000 ms) was not significantly different from zero

one-eighth of a cycle). The slowest period condition produced a slightly positive mean  $\psi_{EMG-K}$  that did not differ from zero.

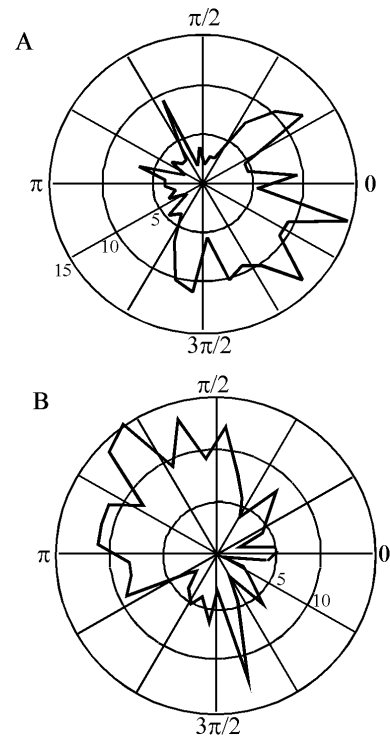
Characteristics of the discrete movement as a function of the rhythmic movement

#### *Onset of the discrete movement*

The central indicator for determining whether the ongoing oscillation had an effect on the discrete movement was the onset of the discrete movement. If only the phase of the trigger signal and an invariant reaction time determined the initiation time of the discrete movement, then the discrete movement should be observed uniformly at all phases of the ongoing oscillation. Note that the stimulus distribution was uniform across the oscillatory phases. The phase of the discrete movement's onset was determined in two ways: on the basis of the periods of the EMG signal of the biceps  $\phi_{EMG}$ , and on the basis of the periods of the joint angular kinematics  $\phi_{angle}$ . Note that our analyses of the time of initiation concentrated on the flexor only. Analyses of the triceps showed that there was no abnormal activity seen in the triceps activity before the moment of increased biceps activity.<sup>2</sup>

Our first objective was to test whether  $\phi_{EMG}$  or  $\phi_{angle}$  was invariant across periods. If the interaction between rhythmic and discrete signals takes place at the EMG

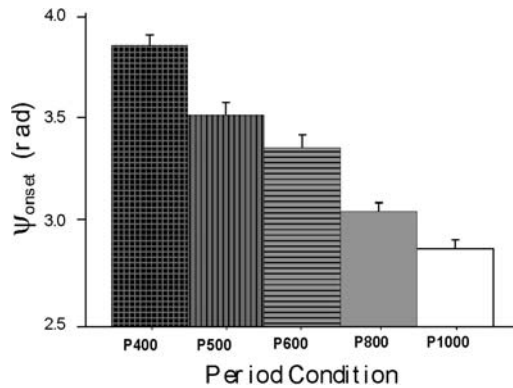
<sup>2</sup> To verify this, the integral of the burst activity of the triceps in the segment that preceded the onset was calculated. The average area of the bursts was calculated for four triceps bursts that preceded this "last" triceps burst. A comparison (*t*-test) between the average burst integral and the last burst integral did not detect any differences. This showed that there was no attenuation of the triceps activity prior to the time when we calculated the onset based on the biceps signal.



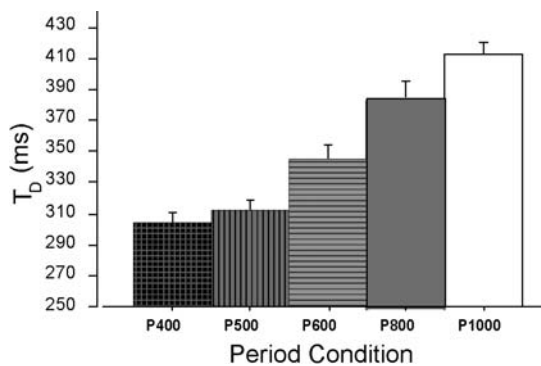
**Fig. 3A–B** Polar histograms of the phase of the discrete onset. **A** Based on EMG data ( $\phi_{EMG}$ ). **B** Based on kinematic data ( $\phi_{angle}$ ). The histograms pool data of all period conditions and participants. Note that  $\phi_{EMG}$  is generally confined to a window between  $3\pi/2$  and  $2\pi$  rad, and  $\phi_{angle}$  is generally confined to a phase window between  $\pi/2$  and  $3\pi/2$  rad

level, then  $\phi_{EMG}$  should be invariant and  $\phi_{angle}$  should vary with period, given the previous results on  $\psi_{EMG-K}$ . This expectation was not supported. Calculating the distributions of  $\phi_{EMG}$  and  $\phi_{angle}$  for each period condition and comparing the means, modes, standard deviations, and ranges across each phase measure there was no significant difference between the period conditions. Hence, Fig. 3A, B displays polar histograms of  $\phi_{EMG}$  and  $\phi_{angle}$ , respectively, in which the data of all period conditions were pooled. Both parts of the figure clearly identify a unimodal distribution ranging between  $3\pi/2$  and  $2\pi$  rad for  $\phi_{EMG}$ , and between  $\pi/2$  and  $\pi$  rad for  $\phi_{angle}$ , respectively. Both distributions have a similar range. The distribution of  $\phi_{EMG}$  indicated that the discrete burst occurred at a phase just prior to the phase at which the EMG burst would have appeared, had the oscillation continued without perturbation. The  $\phi_{angle}$  distribution revealed that the discrete onset lay in the first half of an unperturbed oscillatory cycle, near full extension. The value  $\phi_{angle}$  is defined as zero at maximum flexion (see Fig. 1). Thus, despite the random phase of onset of the stimulus, the discrete movement onset was confined to a phase window ranging across approximately  $\pi/2$  rad.

The absence of differences across period conditions could be due to the fact that variability across trials within a period condition masked systematic differences that may have existed within a trial. Hence, to further identify



**Fig. 4** Phase difference between the EMG-based phase of the discrete onset and the kinematic-based phase of discrete onset ( $\psi_{onset}$ ). The means and standard errors are displayed for the period conditions  $P400$ – $P1000$  (400–1000 ms)

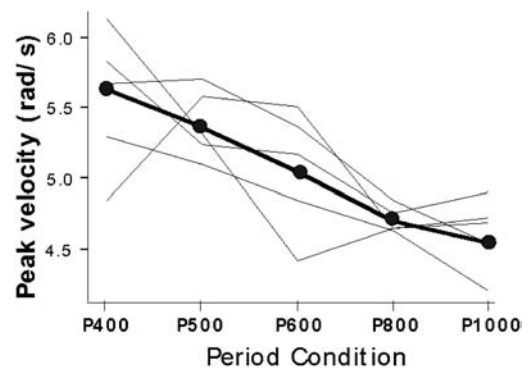


**Fig. 5** Duration of the discrete shift ( $T_D$ , means and standard errors) versus period of the oscillatory movement ( $P400$ – $P1000$ ; 400–1000 ms). The vertical axis begins at 250 ms to highlight the relationship

whether systematic differences between EMG- and kinematic-based initiation phase existed, the difference between  $\phi_{EMG}$  and  $\phi_{angle}$  was calculated for each trial. A two-way ANOVA showed that  $\psi_{onset}$  varied systematically across period conditions ( $F_{4,16}=82.66$ ,  $P<0.001$ ). Figure 4 shows that the largest  $\psi_{onset}$  occurred for the shortest period condition. Note that the  $\psi_{onset}$  for the fastest (400 ms) differed from the slowest (1000 ms) period condition by about 1 rad, which is close to the range of variation observed for  $\psi_{EMG-K}$ . As seen in Fig. 2,  $\psi_{EMG-K}$  for P400 was approximately 0.75 rad larger than for the P1000 condition.

#### Duration and peak velocity of the transition

The next set of analyses focused on the influence of the ongoing oscillation on the characteristics of the discrete movement, specifically on duration and peak velocity. Figure 5 reveals a highly significant relation between  $T_D$  and the period condition ( $P<0.001$ ). Figure 5 further shows that  $T_D$  was noticeably shorter than the corre-

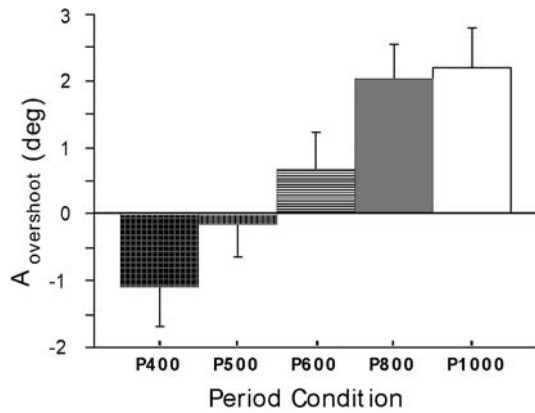


**Fig. 6** Peak velocity of the discrete shift versus period of the oscillatory movement ( $P400$ – $P1000$ ; 400–1000 ms). Mean peak velocities of individual participants are shown with thin lines and the overall mean is shown with a single bold line. The vertical axis begins at 4.5 rad/s to highlight the relationship

sponding oscillation period. To further support these findings, the peak velocities of the transition for all participants are presented across period conditions in Fig. 6. The trend of increased peak velocity for shorter oscillation periods was found in every participant, as shown by the thin lines in Fig. 6. The points connected by the thick line represent the means of all participants. This relation was of particular interest as participants were instructed to move at maximum velocity in all period conditions. Linear regression analyses on peak velocity and  $T_D$  against period revealed that four of the five participants had estimated slopes with 95% confidence intervals that did not contain zero ( $P<0.0001$ ). Two of the ten regressions had confidence intervals that did contain zero; both belonged to participant 4.

#### Overshoot

The focus is on the angular excursion of the discrete movement with respect to different periods of the rhythmic movement. Was there an overshoot or an undershoot in  $A_I$ ? Figure 7 reveals a systematic change in  $A_{overshoot}$  with the prescribed period. The positive values for  $A_{overshoot}$  speak to an overshoot, negative values to an undershoot. Even though the differences in  $A_{overshoot}$  were relatively small, ranging between approximately  $+2^\circ$  and  $-1^\circ$  of angular excursion, the effect of period condition on  $A_{overshoot}$  was still highly significant ( $P=0.002$ ). Note, though, the calculation of  $A_{overshoot}$  was based on the half-amplitude of the post-discrete oscillation ( $10^\circ$ ). Hence,  $+1^\circ$  corresponds to an overshoot of 10%.



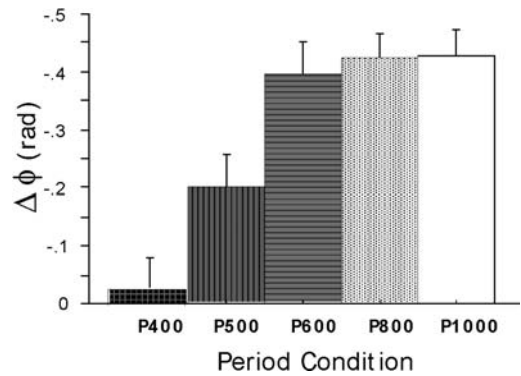
**Fig. 7** Over- and under-shoot ( $A_{overshoot}$ ) of the discrete movement (mean and standard error) at the post-transition amplitude ( $A_f$ ) for the period conditions  $P400$ – $P1000$  (400–1000 ms). A negative value for  $A_{overshoot}$  indicates that the participant undershot the target amplitude

### Characteristics of the rhythmic movement as a function of the discrete movement

#### *Period and amplitude of the rhythmic movement before and after the transition*

Was the oscillation maintained or changed in its kinematic characteristics by the discrete movement? While the discrete movement was an explicit component in the movement sequence, it can equally be interpreted as a perturbation of the ongoing oscillation. To address this question, the average periods and amplitudes preceding and succeeding the transition were compared. As Table 1 highlights, in all but two of the 25 cases, the mean oscillation period decreased following the discrete transition. The  $2 \times 5 \times 5$  ANOVA yielded both the main effect of the pre/post comparison and the interaction effect between period condition and pre/post significant ( $F_{1, 45}=35.00, P<0.001$ , and  $F_{4, 45}=7.03, P<0.001$ , respectively). The interaction signaled that the decrease in period was more pronounced for longer period conditions. However, when normalizing the performed period by the prescribed period, this interaction disappeared ( $P=0.208$ ). Note also that while the pre/post effect was significant, the magnitudes of the period drifts following the discrete transition were small. The overall average was less than 30 ms.

The amplitudes also revealed a drift toward smaller values following the discrete transition. The amplitudes also changed systematically with the prescribed period, but again by only the relatively small average amount of  $1.22^\circ$  across all participants. None of these trends were significant.



**Fig. 8** Shift in the kinematic phase ( $\Delta\phi$ ) of the joint oscillation following the discrete transition for the period conditions  $P400$ – $P1000$  (400–1000 ms). A zero phase shift would indicate that the post-transition oscillation continues as if unperturbed. Negative phase shift indicates that the oscillation was advanced by the discrete transition

### *Phase resetting*

The preceding analyses of the kinematic parameters in the pre- and post-discrete intervals established that the oscillation was marginally but systematically affected. Another typical parameter to evaluate the nature of the effect of perturbation on an ongoing oscillation is the oscillation's shift in phase. While the signature of a forced or paced oscillator is that the oscillation returns to its original phase relationship with the forcing oscillator, an autonomous oscillator is typically shifted to a new phase relationship by the perturbation by some amount (Winfree 1990). For a quantitative evaluation of this phase resetting,  $\Delta\phi$  was calculated for each trial. Figure 8 shows the overall mean  $\Delta\phi$  for each period condition, pooled across participants. The negative values implied that the oscillation were shifted prior to the point in time where the original oscillation would have begun a new cycle if no perturbation had occurred. In less than 10%, a phase delay (a positive value) was observed. These delays occurred predominantly in the fastest period condition. Results of an ANOVA yielded the main effect of period significant ( $F_{4,16}=6.45, P=0.003$ ). Tukey's pairwise comparisons revealed that this discrepancy between period conditions occurred only between the two shortest period conditions and each of the three longer period conditions.

## Discussion

Can we initiate a voluntary action at any time or are there limitations, especially when the limb is in motion? At a fast scale of analysis, limbs are always in motion due to physiological tremor. If for instance we initiate a reach, is there a particular phase in the tremor oscillation that facilitates or impedes the new discrete action? Several previous studies investigated the initiation of a voluntary discrete movement when performed against the background of ongoing movement tremor, e.g., Parkinson's

tremor, examining whether tremor oscillations impede or delay the initiation of a new movement. Similarly often, a new limb movement is initiated when the limb is already involved in other slower rhythmic motions such as in walking. How is a new movement compromised with an ongoing movement? In this paper we specifically address how a discrete action interacts with an ongoing voluntary rhythmic action.

A pervasive finding of a number of studies on this topic was that the initiation seemed to occur preferentially at a particular phase window of the voluntary or tremor rhythm (e.g., Adamovich et al. 1994; Elble et al. 1994; Goodman and Kelso 1983; Staude et al. 1995; Staude and Wolf 1997; Sternad et al. 2000; Wierzbicka et al. 1993). However, the results were not in agreement about which phase window was preferred. For instance, Goodman and Kelso (1983) argued that the onset of the discrete movement tended to occur at the mechanically most efficient moment of the tremor, whereas Wierzbicka et al. (1993) and Elble et al. (1994) found that tremor oscillations impeded rather than assisted the new discrete movement. This contradiction between findings was probably caused by the fact that different studies used different populations with different tremor frequencies, different movement paradigms, and different data analysis techniques, and reliance on EMG, kinematic, or kinetic signals. Hence, the first objective of this study is to bring clarity into these results and interpretations.

The present experiment examined a task in which a discrete movement was performed against the background of a voluntary rhythmic movement. As voluntary movements were used, the oscillation could be controlled to cover a range of different frequencies ranging between periods from 400 to 1000 ms (1–2.5 Hz). We conjectured that due to the different oscillatory periods, different relationships between EMG and kinematic signals arose that lead to the contradictory results.

A second objective was to document the relationship between the discrete and rhythmic movement components in a more detailed fashion. In two of the previous studies, a similar experimental paradigm was investigated whereby voluntary rhythmic movements were merged with a discrete movement (Adamovich et al. 1994; Sternad et al. 2000). Several interaction effects were reported between the two task components that gave rise to different interpretations about the underlying mechanism. By a comprehensive analysis of a range of movement periods, we aimed to further clarify which of three different mechanisms that were proposed in the literature are likely explanations for the observed effects.

#### Relationship between EMG and kinematic signals across different oscillatory periods

Wachholder and Altenburger (1927) were the first to observe different phase relations between EMG and kinematic signals of rhythmic movements that were performed at different periods (see also Sternad 2000).

This early result was replicated in the present study showing that maxima in EMG bursts preceded the respective maxima in the kinematic signals. Importantly, the phase delay between the two maxima varied with movement period such that for very slow periods the flexor burst and the corresponding inflection point were virtually coincident. For faster periods, the EMG burst occurred increasingly earlier. Wachholder and Altenburger (1927) interpreted these findings on the basis of interacting active and passive forces: while the EMG signal reflects the ‘active’ force exerted on the limb by the muscular contraction, there are also ‘passive’ forces like inertia, elasticity, and friction. As for relatively fast movements the inertial force plays an important role, the contraction of the flexor has to occur early to overcome the inertial force from the previous extension. The flexor burst also ceases early as inertia passively carries the limb further until at the end of the excursion. Elastic forces aid in the reversal of the movement. For relatively slow rhythmic movements, inertia plays a subordinate role and activity of the flexor muscle coincides with the beginning or transition from extension to flexion. Wachholder’s observations were clearly supported and quantified in terms of relative phase between the two rhythmic signals. Note, though, that the relative phases between EMG and kinematic landmarks for the slowest (1000 ms) and fastest (400 ms) movements were maximally about 0.80 rad corresponding to only one-eighth of a cycle.

#### Mechanical efficiency or neural inhibition as constraint for the discrete initiation?

Given these period-dependent differences in the phasing of kinematic and EMG activity due to different passive and active forces, the question arises what effect this has on the initiation of a new discrete movement. Is the initiation of the discrete movement sensitive to the dynamics of the rhythmic movement such that it exploits mechanical advantages such as momentum? If so, the discrete initiation should be close to maximum velocity and should be less variable in the kinematic signal compared to the EMG activity over the different period conditions. Conversely, if the interaction is due to interactions at the neural level that constrain the onset of the discrete initiation burst, the phase of the onset should be less variable with respect to the EMG activity over the different period conditions. To resolve this question, the moment of initiation of the discrete movement was calculated for both the EMG and kinematic signals of the rhythmic movement. Both phases of initiation showed a clear unimodal distribution, confirming that the discrete movement onset was indeed confined to a phase window of the rhythmic movement in accord with previous results.

However, counter to the contrasting hypotheses, no differences in phase onsets could be identified across periods, neither for the onset defined by the EMG activity, nor by the kinematic signal. Similarly, the ranges

and standard deviations of onset phases for each period condition did not statistically differ from each other. This lack of differences can be explained by the finding that the differences in phase relation between EMG and kinematics in the tested period conditions proved to be too small and the variability too high to produce visible shifts in the modal distributions. The range of the unimodal distribution (within and across period conditions) was approximately one-fourth of a cycle, while the range of phase differences between EMG and kinematic signals between the slow and fast condition was only one-eighth of a cycle. In sum, our results do not permit us to identify the primary level of expression of the interaction between the discrete and the rhythmic movement.

However, when the differences between the two phase onset measures were computed within each trial, thereby avoiding across-trial variability, systematic changes were seen across the different period conditions. While on average the difference was approximately half a cycle, as seen in the two overall distributions, smaller differences were observed for the longer periods and larger differences for the shorter periods. These differences correspond to the phase difference between EMG and kinematic signals during steady state oscillations. Hence, this shows that within each trial the difference in phase is maintained at the moment of initiation. In other words, the EMG-kinematic relation that is specific for each period condition due to the dynamics of the rhythmic movement remains reliable at the onset of the discrete movement. In principle, a better way to address our question would have been to determine the onset of the discrete movement separately in the EMG and the kinematic signal. This, in fact, was done by Wierzbicka et al. (1993) who developed an algorithm to detect deviations in the force signal from the extrapolated fitted rhythmic trajectories. The problem, however, is that this procedure is very unreliable due to noise in the signal.

All studies in the literature that examined EMG activity for the initiation of the discrete movement focused their analyses on the agonist. While this is a reasonable choice for defining a threshold for the onset, it could also be possible that the discrete signal attenuates the antagonist, in the present study the triceps, prior to the activation of the flexor. This hypothesis was tested, but no indication for it was found. However, it was also evident that the triceps showed a different activity pattern during the discrete flexion. During the high activity of the biceps that produces the flexion movement, there is also considerable activity of the triceps, giving evidence to some co-contraction. A spike in the triceps follows immediately after the large biceps burst that evidently leads to the return extension of the movement. This characteristic behavior occurs simultaneously with the discrete flexion and does not precede the determined moment of initiation of the discrete movement. Further exploration of the triceps behavior is interesting but is outside the purview of the present study.

Looking at the obtained modal distributions of the onset phases, the results showed that the EMG burst of the

discrete movement occurred between  $3\pi/2$  and  $2\pi$  rad of the ongoing rhythmic EMG activity. This means that the discrete initiation is constrained to occur at, or slightly advanced to, the burst of the ongoing rhythmic activity, in accord with some previous studies (Wierzbicka et al. 1993; Staude et al. 1995; Adamovich et al. 1994; Sternad et al. 2000). As such, this result appears to be conform with the assumption of a simple inhibitory mechanism that suppresses the muscular activity associated with the discrete movement at phases other than the one when the rhythmic burst is active anyway. When determining the corresponding onset phase in the kinematic signal, this phase was between  $\pi/2$  and  $\pi$  rad, which corresponds to the second half of the extension segment of the elbow movement (upward direction in Fig. 1). As such, the increased EMG activity happened half a cycle before the time of maximum momentum. At first sight, this result appears incompatible with an initiation of the discrete movement at the mechanically most efficient time (Goodman and Kelso 1983). However, this calculation did not take into account the delay between EMG activity and the overt realization of the discrete movement. As this time delay changes across periods, this contradiction cannot be resolved easily. It remains to say that a mechanism that exploits mechanical advantage needs to solve the inverse dynamics problem, while the same functional behavior can be accounted for by a simple inhibitory mechanism. Other results are needed to resolve this opposition of accounts.

Trying to address the same problem, Elble et al. (1994) calculated the onset of the discrete movement on the basis of the kinematic signal and related this onset time to the ongoing EMG bursts of the tremor, that is they performed the onset analysis in reverse fashion to our analysis. They concluded that tremor impeded the discrete movement. We reasoned that this result probably only reflects the time delay between EMG activity and the overt kinematics, and the authors did not provide sufficient information to further evaluate this interpretation. Earlier results by Wierzbicka et al. (1993) similarly stated that the tremor impeded rather than assisted the discrete isometric action. In contrast to Elble and colleagues' analyses and our data analyses, this study determined the discrete onset separately for the force and the EMG signal. The initiation of an increased isometric force occurred at a time where the EMG impeded this force increment. Similar to our study, the EMG bursts of the discrete and rhythmic movement were close to coincident. While there seems to be a convergence in this result among several experiments, this conclusion needs to be taken with a grain of salt: while the *initiation* of the discrete movement may not be coincident with maximum momentum, the unfolding of the discrete movement itself may still utilize this momentum.

Bidirectional interaction of discrete and rhythmic movement components: characteristics of the discrete movement as a function of the rhythmic movement

The second main interest of exploring a discrete movement performed against the background of rhythmic movement over a wide range of frequency was related to the bidirectional nature of the interaction between discrete and rhythmic movement. Effects of the rhythmic onto the discrete component were already evidenced in the initiation constraint discussed above. But there are two other results that strongly speak to this directional effect. Firstly, the duration of the discrete movement and the peak velocity measured at the transition were systematically influenced by the period of the oscillatory movement. This is noteworthy because in all trials participants were instructed to react as fast as possible and to produce shifts with maximum velocity. Peak velocities were between 4.5 and 6.0 rad/s or, on average, 290 degrees/s over an amplitude of 40°. This is remarkably slow compared to rapid elbow movements, as reported for instance by Gottlieb and colleagues (Gottlieb 1998; Gottlieb et al. 1989). Single elbow rotations of the same amplitude were performed with peak velocities between 500 and 600 degrees/s. This effect is probably also due to the fact that the oscillations were constrained to an approximately constant target amplitude. Although subjects tended to slightly increase their amplitudes for longer periods, the target constraint implied that the peak velocities of the oscillatory movements increased for shorter periods. Hence, the velocities of the oscillations had a marked effect on the following discrete movement. It appears that the ongoing oscillation determines the level of drive and co-contraction of the flexors and extensors which, in turn, limits the peak velocity of the discrete movement (Feldman 1980; Latash 1992).

Secondly, the amplitude overshoots observed at the first inflection terminating the discrete shift also showed a systematic dependence on the oscillatory period. For slower periods an overshoot was observed, for faster periods an undershoot was predominant. If these over- and undershoots were caused by passive inertial forces, then the overshoot should be smallest for slow periods and highest for fast periods. The opposite pattern was observed. Hence, this effect may be attributed to elastic forces that are more prominent in fast oscillations and produce high restoring torques that lead to an earlier reversal of the movement. Alternatively, the undershoot in fast oscillations may be due to an increased level of co-contraction that is also associated with faster oscillatory movements. Although it cannot be decided at this point whether it is the neural or mechanical factor that is prevalent, it remains to hold the view that background oscillations produce different neural and mechanical effects that generally slow down and shape the kinematic features of the discrete movement. Note that these results are not in agreement with Adamovich et al. (1994) who reported no influence from the rhythmic onto the discrete

movement after the initiation. The authors developed an averaging technique in which a set of position trajectories was carefully selected such that the distribution of discrete onsets was uniform. Averaging this subset of trajectories cancelled the rhythmic part of the trajectory and extracted only its discrete part. When comparing this discrete portion with an isolated discrete movement no differences were found. This finding was interpreted that the discrete movement itself was unaffected in the combined task. Given that this averaging technique used a non-representative subset of trials and also did not control the oscillation periods, it is hard to reconcile these results with ours. It should also be emphasized that, strictly speaking, the identified interaction effects cannot speak to a causal relationship between the two processes but only indicate a correlation. However, logically speaking, such correlations between the discrete and the rhythmic units can only arise from two sources: there is a coupling between the two processes, or, it can be due to a third process that has an influence on both. However, the present data cannot decide between these options.

Characteristics of the rhythmic component as a function of the discrete movement

The most prominent effect is the shift in the phase of the rhythmic movement after the discrete movement that was reported in slightly different ways in previous studies. Sternad et al. (2000) calculated phase shifts in the same fashion as in this study and reported a phase advance for oscillation periods of 333 and 500 ms. The present study revealed these effects in a more detailed fashion showing systematic variations in the phase shift as a function of period. The oscillatory phase was more advanced for slow periods than for fast periods. The overall tendency for shortening of the cycle is straightforward, as the duration of the discrete movement is generally shorter than one oscillatory cycle. Whereas for the 400 ms period, the discrete duration is 300 ms, or 75% of the cycle period, the duration for the 1000 ms period condition is 400 ms, or 40% of the cycle. Hence, for 400 ms and for 1000 ms the advance should be 25% and 60% of a cycle. The fact that the observed phase shifts are only between  $-0.05$  and  $-0.40$  rad is due to the fact that the discrete duration was, by instruction, always as fast as possible and varied less than the controlled oscillatory period. Also, the phase shift was determined not at the cycle directly after the discrete transition but three cycles later to estimate the lasting shift in the rhythm. Interpreting the rhythmic movement as an autonomous oscillation, such phase shift was interpreted as the signature of a perturbed but continued oscillation (e.g., Winfree 1990).

The comparison between the steady state oscillations before and after the discrete movement revealed another long-term effect from the discrete movement. After the discrete transition the periods became slightly faster regardless of period condition. The effect was small but consistent and replicated previous findings from Sternad

et al. (2000). One account for this finding is that for the discrete action an increased level of contraction in the flexor or co-contraction was required that persisted in the post-transition interval. On the other hand, many other experimental paradigms on rhythmic timing have observed such accelerations during a longer trial or after a pacing metronome has been switched off (e.g., Sternad et al. 1999). Reasons for this tendency are yet unclear. Alternatively, systematic drifts have been reported toward the preferred frequency (Yu et al. 2002). The fact that in the present study larger drifts were observed for the slower periods is reminiscent of this tendency. However, this phenomenon is unlikely to be the major contributor to the present finding, as a wide range of periods was investigated and acceleration was observed even in the fastest condition.

#### Modeling the mechanisms for the observed interactions between discrete and rhythmic components

Adamovich and colleagues found similar phase shifts as reported in this paper but together with their lack of effects on the discrete movement they arrived at a different interpretation of their results. The authors concluded that the control signal for the rhythmic movements was temporarily interrupted and restarted after the termination of the discrete movement (Adamovich et al. 1994). However, this sequential account does not provide any explanation of why the onset of the discrete movement was confined to a particular phase window of the rhythmic movement. In conjunction with the arguments above, we want to argue that a sequential on-off mechanism is unlikely to account for the observed behavior and some form of inhibitory coupling is more likely.

On the basis of similar results on the constraints of the discrete initiation, Staudé et al. (1995) proposed a simple gating model. The interaction between the discrete and the rhythmic movement is assumed to occur at the level of the control signals, where the rhythmic signals suppress the discrete components by a periodic gating process. In a modified version of the model, Staudé and Wolf (1997) proposed a linear superposition of the control signals of the discrete and rhythmic components. This additive interaction is assumed to occur at some lower level, i.e., the discrete movement can only become overt when the sum of the rhythmic and discrete components is greater than a threshold. These two propositions are distinct from the sequential control suggested by Adamovich et al. (1994) in the sense that both discrete and rhythmic commands can be activated simultaneously in an unspecific way, i.e., whatever the timing relation between them, the mechanism gives rise to the specific onset effect. However, the problem is now that these propositions do not account for the previously discussed and reported phase shift. The superposition only produces a phase delay, not an advance. In addition, neither Adamovich and colleagues' rationale nor Staudé and coworkers'

model can account for an effect of the rhythmic movements on the discrete movement itself as detailed above.

Sternad et al. (2000) proposed a cross-coupling model with mutual inhibition that reproduced the distribution of phase shifts and onset phases. The rhythmic component inhibits the discrete component at certain phases, and in return, the discrete component, once it operates, inhibits the rhythmic component. The onset effect is thus explained by a process close to the gating mechanism of Staudé et al. (1995): the discrete movement occurs at a phase window where the rhythmic component does not inhibit it. The phase shift is due to the difference in the time of the discrete movement and the oscillatory period, as the rhythmic movement is suppressed and re-started with the initiation and termination of the discrete movement. But again, this effect is due to the designed cross-coupling rather than an on-off sequencing of the control signals. Both components are activated simultaneously and in an unspecific way. At its stage of development, however, the model designed by Sternad et al. (2000) does not account for the difference in the duration of the discrete movement obtained for different periods. It remains that a bidirectional coupling appears most appropriate to capture both the effect of the rhythmic movement on the discrete one and the effect of the discrete on the rhythmic one. But further modeling work is needed to fully capture the detailed interactions between the two task components.

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## References

- Adamovich SV, Levin MF, Feldman AG (1994) Merging different motor patterns: coordination between rhythmical and discrete single-joint movements. *Exp Brain Res* 99:325–337
- Elble RJ, Higgins C, Hughes L (1994) Essential tremor entrains rapid voluntary movements. *Exp Neurol* 126:138–143
- Feldman, AG (1980) Superposition of motor programs. 1. Rhythmic forearm movements in man. *Neuroscience* 5:81–90
- Goodman D, Kelso JAS (1983) Exploring the functional significance of physiological tremor: a biospectroscopic approach. *Exp Brain Res* 49:419–431
- Gottlieb GL (1998) Muscle activation patterns during two types of voluntary single-joint movements. *J Neurophysiol* 80:1860–1867
- Gottlieb GL, Corcos DM, Agarwal GC (1989) Strategies for the control of voluntary movements with one mechanical degree of freedom. *Behav Brain Sci* 12:189–250
- Hallet M, Shahani BT, Young, RR (1977) Analysis of stereotyped voluntary movements at the elbow in patients with Parkinson disease. *J Neurol Neurosurg Psychiatry* 40:1129–1135
- Latash, ML (1992) Virtual trajectories, joint stiffness, and changes in the limb natural frequency during single-joint oscillatory movements. *Neuroscience* 49:209–220
- Staudé G, Wolf W (1997) Quantitative assessment of phase entrainment between discrete and cyclic motor actions. *Biomed Technik* 42:478–481
- Staudé G, Wolf W, Ott M, Oertel WH, Dengler R (1995) Tremor as a factor in prolonged reaction times of parkinsonian patients. *Mov Disord* 10:153–162

- Sternad D (2000) Kurt Wachholder: pioneering electrophysiological studies of voluntary movements. In: Latash ML, Zatsiorsky VM (eds) *Classics in movement science*. Human Kinetics, Champaign Ill. pp 375–407
- Sternad D, Dean WJ, Newell KM (1999) Force and timing variability in rhythmic unimanual tapping. *J Mot Behav* 32:249–267
- Sternad D, Dean WJ, Schaal S (2000) Interaction of rhythmic and discrete pattern generators in single-joint movements. *Hum Mov Sci* 19:627–664
- Travis LE (1929) The relation of voluntary movement to tremors. *J Exp Psych* 12:515–524
- Wachholder K, Altenburger H (1927) Beiträge zur Physiologie der willkürlichen Bewegungen. Fortlaufende Hin- und Herbewegungen. IX. *Pflüger's Arch* 214:625–641
- Wierzbicka MM, Staude G, Wolf W, Dengler, R (1993) Relationship between tremor and the onset of rapid voluntary contraction in Parkinson's disease. *J Neurosurg Psych* 56:782–787
- Winfree AT (1990) *The geometry of biological time*. Springer-Verlag, New York Heidelberg Berlin
- Yu H, Russell DM, Sternad D (2002) Task-effector asymmetry in a rhythmic continuation paradigm. *J Exp Psychol Hum Percept Perform* (In press)