Effector dynamics of rhythmic wrist activity and its implications for (modeling) bimanual coordination

Arne Ridderikhoff a,*, C. (Lieke) E. Peper a, Richard G. Carson b, Peter J. Beek a

a Institute for Fundamental and Clinical Human Movement Sciences (IFKB), Faculty of Human Movement Sciences, Vrije Universiteit, Van der Boechorststraat 9, 1081 BT Amsterdam, The Netherlands
b Perception and Motor Systems Laboratory, School of Human Movement Studies, The University of Queensland, Brisbane, Australia

Abstract

To examine the role of the effector dynamics of the wrist in the production of rhythmic motor activity, we estimated the phase shifts between the EMG and the task-related output for a rhythmic isometric torque production task and an oscillatory movement, and found a substantial difference (45–52°) between the two. For both tasks, the relation between EMG and task-related output (torque or displacement) was adequately reproduced with a physiologically motivated musculoskeletal model. The model simulations demonstrated the importance of the contribution of passive structures to the overall dynamics and provided an account for the observed phase shifts in the dynamic task. Additional simulations of the musculoskeletal model with added load suggested that particular changes in the phase relation between EMG and movement may follow largely from the intrinsic muscle dynamics, rather than being the result of adaptations in the neural control of joint stiffness. The implications of these results are discussed in relation to (models of) interlimb coordination in rhythmic tasks.

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* Corresponding author. Tel.: +31 20 444 8454; fax: +31 20 444 8529.
E-mail address: a.ridderikhoff@fbw.vu.nl (A. Ridderikhoff).
1. Introduction

Rhythmic limb movements involve many different processes embodied in a variety of anatomical structures. Despite these variations in substrate, a number of pervasive dynamical properties of rhythmic movement can be identified. To some extent (Beek, Peper, & Daffertshofer, 2002), rhythmic limb movements can be modeled as autonomous limit cycle oscillators, allowing for a systematic formal analysis of their kinematics (Beek & Beek, 1988; Beek, Rikkert, & van Wieringen, 1996; Beek, Schmidt, Morris, Sim, & Turvey, 1995; Kay, Saltzman, & Kelso, 1991). Furthermore, when two (or more) rhythmic limb movements are coordinated, interesting and complex phenomena can be observed that provide insight into the neural processes underlying interlimb coordination (Swinnen, 2002). Most importantly, in multilimb movements the behavior is attracted toward a limited number of temporal relations between the limbs. For isofrequency tasks, these relations are typically characterized by the phase difference, or relative phase (Kelso, 1984), between the limbs, and for multifrequency tasks by the frequency ratio (Peper, Beek, & van Wieringen, 1995), or the generalized relative phase (Sternad, Turvey, & Saltzman, 1999). These multilimb patterns result from and thus reflect interactions between the limbs (Kelso, Southard, & Goodman, 1979; Marteniuk, MacKenzie, & Baba, 1984; Von Holst, 1939/1973), and their stability usually depends on movement frequency (Carson, Goodman, Kelso, & Elliott, 1995; Kelso, 1984; Peper et al., 1995).

For isofrequency movements, it has been shown that frequency-induced changes in stability of the relative phase $\phi$ can be explained by a model of two coupled limit cycle oscillators (Haken, Kelso, & Bunz, 1985). Like the experimental observations that motivated its derivation (Kelso, 1984), this so-called HKB model showed a spontaneous switch (or phase transition) from the antiphase pattern ($\phi = 180^\circ$) to the more stable in-phase pattern ($\phi = 0^\circ$) when the movement frequency was gradually increased (Haken et al., 1985). In addition, the HKB model explained experimental observations like critical slowing down (Scholz, Kelso, & Schönér, 1987) and critical fluctuations (Kelso, Scholz, & Schönér, 1986; Schönér, 1986) in the vicinity of a phase transition. In later years, many (stability-related) aspects of the relative phase dynamics and the kinematic characteristics of interlimb coordination have been captured successfully by the HKB model and its various extensions (Daffertshofer, Van den Berg, & Beek, 1999; Fuchs, Jirsa, Haken, & Kelso, 1996; Haken, Peper, Beek, & Daffertshofer, 1996; Kelso & Jeka, 1992; Post, Peper, Daffertshofer, & Beek, 2000; Treffner & Turvey, 1995).

However, a recent review of the available empirical evidence indicated some shortcomings of the HKB model that cannot be remedied by simple changes in the oscillator or coupling parameters (Beek et al., 2002). To overcome these limitations, a two-tiered model for interlimb coordination was presented, in which the
movements of each individual limb were modeled by means of a nonlinear ‘neural’ oscillator, bi-directionally coupled to a linear ‘effector’ oscillator (Beek et al., 2002; Peper, Beek, & Daffertshofer, 2000). By making this explicit distinction between the neural dynamics and the effector dynamics, the explanatory power of the two-tiered model was substantially enhanced relative to that of the HKB model, as a variety of empirical observations that were inconsistent with the system of coupled oscillators in the original HKB model could now also be accounted for (for details, see Beek et al., 2002). In addition, such a two-tiered model may help to reduce the phenomenological character of the HKB model. For example, the abstract nature of the HKB model implies that no distinction is made between neurophysiological, muscular, and biomechanical aspects. Hence, the HKB model is mute with regard to, for instance, the debate as to whether (the stability of) the relative phase should be defined in relation to the activation of homologous muscle groups, or in relation to perceived displacements (Carson, Riek, Smethurst, Parraga, & Byblow, 2000; Mechsner, Kerzel, Knoblich, & Prinz, 2001; Park, Collins, & Turvey, 2001; Riek, Carson, & Byblow, 1992; Swinnen, Jardin, Meulenbroek, Dounskaia, & Hofkens-Van Den Brandt, 1997; Temprado, Swinnen, Carson, Tourment, & Laurent, 2003). A two-tiered model, on the other hand, offers a more natural framework for developing an understanding of the functional form of the neural coupling and essential features of the effector dynamics, thereby providing a connection between the observed relative phase dynamics and the underlying (neuro)physiological processes.

Studies in which the mechanical characteristics of the effectors have been manipulated indicated that the significance of the effector dynamics for interlimb coordination is substantial. For example, increasing the inertia of one of the moving limbs may generate a degree of detuning that is sufficient to induce phase wrapping (Jeka & Kelso, 1995; Kelso & Jeka, 1992). In addition, loading a single rhythmically moving limb has been reported to result in adaptations in the electromyogram (EMG) (Baldissera & Cavallari, 2001; Mackey, Meichenbaum, Shemmell, Riek, & Carson, 2002). Moreover, the effects of asymmetrical loading of the limbs have been shown to depend on the coordination pattern performed (in-phase or antiphase) (Baldissera & Cavallari, 2001; Baldissera, Cavallari, Marini, & Tassone, 1991; Rosenblum & Turvey, 1988; Schmidt, Shaw, & Turvey, 1993; Schmidt & Turvey, 1995). Together these results underscore the relevance of an analysis of effector dynamics for the study of interlimb coordination.

The aim of the present paper is to analyze the effector dynamics of rhythmic wrist activity, in particular with regard to the asymmetries that may arise in interlimb coordination when two different tasks are performed simultaneously. To this end, two tasks (rhythmic isometric torque production and rhythmic movement) were compared with respect to the temporal characteristics of the effector dynamics. In
practice, the effector dynamics were defined as the transfer function between EMG and the (task-related) output with a special focus on the phase shifts involved, because these phase shifts are of particular importance to the dynamics of rhythmic interlimb coordination. Using model simulations it was shown that the differences between these tasks can be explained in terms of the dynamics of the musculoskeletal system. In addition, the model was applied to analyze the effects of inertial loading of (one of) the limb segments, a manipulation that is rather common in the literature. The results of these analyses are discussed in relation to (models of) interlimb coordination.

2. Methods and results

Two different tasks were analyzed with respect to the effector dynamics. In one task participants generated an alternating flexion–extension torque around a restrained wrist (isometric task). In the other task participants made a smooth oscillatory flexion–extension movement (dynamic task). Hence, the output variables of the two oscillatory activities were qualitatively different (torque vs. displacement). Empirical data on these tasks were obtained in an experiment by Peper and Carson (1999); for a full description of the experiment, and a discussion of other differences (e.g., sensory aspects) in the context of coordination dynamics, the reader is referred to this original article. As regards the effector dynamics, a comparison of isometric and dynamic tasks is interesting for two reasons. First, considerable differences exist between isometric and dynamic (eccentric and concentric) contractions, as is well-known from classical studies on muscle physiology (Fenn, 1923; Hill, 1938). Second, interactions between muscle dynamics and joint dynamics occur in the dynamic task, due to the relationship between muscle length and joint angle. For example, joint angle determines muscle length and thereby affects the contraction process, resulting in changes in muscle forces. In turn, the changes in muscle forces affect the net joint torque, which influences the trajectory of the joint angle. Such interactions are absent in the isometric task, because the joint angle is kept constant.

2.1. Analysis of experimental data

In the experiment of Peper and Carson (1999), six participants performed isometric and dynamic tasks unimanually as well as bimanually (i.e., simultaneous performance of the two tasks) under two pacing conditions (1.0 and 1.4Hz). The isometric task was performed at two force levels (high and comfort) in both the unimanual and bimanual conditions. EMG of two muscles, flexor carpi radialis (FCR) and extensor carpi radialis (ECR), was registered during the experiment. EMG, torques and kinematics were all recorded at a sample frequency of 1000Hz. For our current purposes, the phase shift between EMG and task-related output was estimated based on the cross-spectral density of the EMG and associated output as determined for
each muscle separately. The procedure is illustrated in Fig. 1. In preprocessing, EMG
records were bandpass filtered (10–500 Hz) using a second-order bi-directional (zero-
lag) Butterworth filter, and subsequently full-wave rectified (Merletti, Farina,
Hermens, Freriks, & Harlaar, 1999). The cross-spectral density was estimated with

Fig. 1. Estimation of phase shifts due to effector dynamics. Left column: basic harmonics obtained from
the power spectral density (solid) were superimposed on the data of a single trial (EMG of both muscles
and joint angle) of the dynamic task (dashed; here paced at 1.0 Hz). Frequency and phase of the basic
harmonics are indicated in the titles. For clarity only a part of the time series is shown. EMG data (top
and middle panel) were bandpass filtered and full-wave rectified. Right column: the power (top panel) and
phase shift (middle panel) of the cross-spectral density of EMG and joint angle for ECR (solid) and
FCR (dashed). In the bottom panel, the phase shifts for ECR (solid) are shifted by $\pi$ to correct for the
negative joint angle obtained for extension movements; the (unchanged) phase shifts for FCR are also
shown (dashed) for comparison. The phase shift between EMG and joint angle was estimated at the
frequency of the basic harmonic (equal to the pacing frequency of 1.0 Hz as indicated by the vertical dotted
dline).
Welch’s modified periodogram method, as implemented in MATLAB®. 2 In all trials the highest power in the cross-spectrum was found at the movement frequency. The phase shift due to the effector dynamics was estimated from the cross-spectral density by taking the phase difference between EMG and task-related output as obtained for the movement frequency. Because extension corresponded to negative joint angles and torques, the phase shift between ECR EMG and activity in the direction of wrist extension was determined by shifting the values for ECR by \( \pi \).

The average and standard deviations of the phase shifts across all subjects, which were calculated using circular statistics (Mardia, 1972), are presented in Table 1. To facilitate interpretation, phase shifts are presented in degrees in the remainder of this paper. A negative phase shift indicates that the joint angle or torque time series was lagging the EMG signals. A \( 3 \times 2 \times 2 \) repeated measures ANOVA was used to examine the effects of Type of Contraction (dynamic, isometric at comfort force, or isometric at high force), Frequency (1.0Hz or 1.4Hz), and Muscle (ECR or FCR) on the phase shift between EMG and the task-relevant output (torque for the isometric task; joint angle for the dynamic task). A significant effect of Type of Contraction was found \( (F(2,10) = 120.1, p < 0.001) \). The average phase shift in the dynamic task was almost twice as large as in the isometric task \( (-102^\circ \text{ vs. } -53^\circ, \text{ respectively}) \), whereas the difference between the two isometric conditions was much smaller \( (-52^\circ \text{ vs. } -55^\circ \text{ for high force and low force, respectively}) \). Post-hoc paired sample \( t \)-tests \( (p < 0.05) \) indicated that the differences between all conditions were significant. In addition, a main effect of Frequency \( (F(1,5) = 646.2, p < 0.001) \) revealed that phase shifts were larger for the higher movement frequency. A significant interaction between Type of Contraction \( \times \) Frequency \( (F(2,10) = 16.8, p < 0.01) \) indicated that phase shifts increased more with movement frequency in the dynamic task than in the isometric task (an average increase of \( 21^\circ \text{ vs. } 14^\circ, \text{ respectively}) \). Also a main effect

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2 A Hamming window was used, the length of which corresponded to an integer multiple of the cycle duration in order to minimize spectral leakage. For maximum accuracy, the window length equaled three cycles (3000 samples for 1Hz and 2143 samples for 1.4Hz) with a between-window overlap of half the window length (Chatfield, 1984; Oppenheim & Schafer, 1975). The number of points in the Fourier transform was the same as the number of samples in each window, that is, zero-padding or truncation was avoided.
of Muscle was found ($F(1,5) = 12.4, p < 0.05$), which occurred because the phase shifts of FCR were larger than those of ECR. However, this effect of Muscle was rather small (about 7°) and the absence of significant interaction effects suggested that this effect was unrelated to the effects of Frequency or Type of Contraction. In sum, the results of the experiment of Peper and Carson (1999) revealed marked differences in the phase shifts between the neural control signal (EMG) and the task-related output (displacement or torque), in particular between the dynamic and isometric task and between the two movement frequencies. Evidently, an adequate model of the effector dynamics should be able to account for these effects.

2.2. Reference signals for model simulations

For the model simulations representative time series for the EMG-based input signal and the task-related output signal (torque or position) were required. To construct an appropriate input signal, the preprocessed EMG signals (see above) were low-pass filtered (bi-directional (zero-lag) generalized 10th-order Butterworth filter; cut-off frequency 15 Hz) to obtain linear envelopes. The relatively high cut-off frequency was selected to preserve most of the shape of the EMG bursts.

The reference signals for the model simulations had to meet the following four criteria. First, the signals should be representative in terms of mean cycle trajectory and variability. Second, the number of cycles should be sufficient to avoid transient effects at the beginning of the simulations and numerical artifacts at the end. Third, the number of cycles used for comparison should be sufficient to detect error propagation (drift). Fourth, to avoid the excessive use of computational time for the optimization of the model, the number of cycles should be as small as possible. In practice, the last three criteria were satisfied by using a series of six cycles, out of which cycles 3–5 were used for comparison between simulations and experiment.

To meet the first criterion, a representative series of six cycles was obtained using the following procedure. First, all possible series of 10 successive cycles were taken from a single trial. This was done for all trials for all participants resulting in a new intermediate dataset, from which a representative series of 10 cycles was constructed by principal component analysis (PCA). For each experimental condition three such time series were created: one for the linear envelopes of the EMG of each muscle (ECR and FCR) and one for the behavioral variable of interest (joint angle for the dynamic task and torque for the isometric task). To avoid transients, an ‘equilibrium’ initial condition was obtained by selecting the sample in the first four cycles at which the derivative of the variable of interest (angle or torque) was closest to zero. The six cycles following that sample were used as reference signal.

Using PCA to obtain the representative time series of torques, joint angles, and EMG amplitudes also provided information about the variance in the original dataset that was accounted for by the constructed time series (Post, Daffertshofer, & Beek, 2000), which ranged from 52% to 95%, with a median value of 70%. These relatively low values were caused by the high variability of the EMG signals, both within and between participants. Reference input (EMG) and output (angle or torque) were determined for four conditions of the isometric task (two frequencies × two
force levels), and two conditions of the dynamic task (two frequencies). For both muscles, the highest EMG amplitude was found in the high force condition of the isometric task at a frequency of 1.4 Hz. The maximum values obtained for this condition were used to normalize all input signals to the interval [0, 1] in order to facilitate scaling.

3. Musculoskeletal model

In the wrist model developed in the present study, four muscles (flexor carpi radialis—FCR, flexor digitorum superficialis—FDS, extensor carpi radialis—ECR, and extensor digitorum—EDC) act as prime movers on a single hinge joint (the radiocarpal joint). The musculoskeletal model comprises joint dynamics and muscle dynamics, as well as their interaction. The joint dynamics is modeled as a mechanical system with a single degree of freedom, which is fully described by two state variables corresponding to joint angle and joint angular velocity. The model of muscle dynamics was based on the Hill-type model presented by Van Soest and Bobbert (1993), which consists of two subsystems in series representing the activation dynamics and the contraction dynamics of skeletal muscle. The nonlinear muscle model, which is described in the next section, has two state variables, intramuscular concentration of Ca$^{2+}$ and length of the muscle fibers. In total, the musculoskeletal model has 10 state variables (4 × 2 for the muscles and two for the joint). The behavior of the model is rather complex due to its high dimensionality, the nonlinear contraction dynamics, and the interactions between activation dynamics, contraction dynamics, and joint dynamics (see Fig. 2).

The joint dynamics of the musculoskeletal model was described by a linear damped mass–spring system:

$$J\ddot{\theta} + B\dot{\theta} + K(\theta - \theta_{eq}) = T(t)$$  \hspace{1cm} (1)

![Fig. 2. Schematic representation of the subsystems of the musculoskeletal model of the effector dynamics and their interactions. The change in muscle force $F$ in response to changes in the input vector $STIM$ is determined by the activation dynamics ($A$), the contraction dynamics ($C$), and the spring-like tendon and other elastic structures in series (SE). The sum of muscle forces multiplied with the respective moment arms $r$ yields the driving moment $T$, which is input to the damped mass–spring system $JBK$. The output of the complete system is the joint angle $\theta$. The dimension of the variables is indicated between brackets.](image-url)
where $J$ is the moment of inertia of the hand (0.0054kgm$^2$ for an average subject of 75kg according to Winter, 1990), and $T(t)$ is the resultant muscle torque. The stiffness ($K$) and damping ($B$) parameters represent the lumped mechanical effect of the deformations of the passive structures spanning the wrist. The joint angle is represented by $\theta$, with the system being in static equilibrium at resting angle $\theta_{eq}$. The neutral position of the wrist ($\theta = 0$) was defined as alignment of the longitudinal axis of hand and forearm, and flexion was defined as positive. In the present context, the most relevant property of the joint dynamics is the phase shift between the input $T$ and the output $\theta$. Linear systems theory (Doebelin, 1998) dictates that this phase shift $\Delta \Phi_{\text{joint}}$ is a function of the movement frequency $f$:

$$\Delta \Phi_{\text{joint}} = \arctan \left( \frac{-B \omega}{-J \omega^2 + K} \right)$$

(2)

where $\omega = 2\pi f$.

The interaction between muscle and skeleton results on the one hand from changes in the length of the muscle–tendon complex ($l_{MTC}$) due to changes in joint angle, and on the other hand from muscle forces that determine the driving torque of the joint dynamics given by Eq. (1). This driving torque is the sum of muscle moments $T = \sum_j r_j F_j$, where $F_j$ and $r_j$ are the force and moment arm of the $j$th muscle, respectively. The moment arm $r_j$ is equal to the derivative of $l_{MTC}$ with respect to $\theta$ (Murray, Delp, & Buchanan, 1995). The relation between $l_{MTC}$ and the joint angle $\theta$ is determined by the anatomy of the musculoskeletal system, and approximated by a third-order polynomial in our model (see Table 2). The muscle length at $\theta = 0$ is determined for a reference posture with a ‘neutral’ pronation/supination angle of 0° (for wrist movements in the horizontal plane) and an enclosed elbow angle of 120° (with 180° reflecting full extension). The parameters for the musculoskeletal geometry reported in Lemay and Crago (1996) were used as reference.

The independent input of the musculoskeletal model is the vector of neural control signals $STIM(t)$, which is related to the normalized, EMG-based, neural control signal $\xi$, the derivation of which was described in the previous section:

$$STIM_j(t) = z_j \xi_j(t)^{\theta_j}$$

(3)

where the indices $j$ indicate flexors ($j = 1$) or extensors ($j = 2$). Thus, a single $STIM$ signal was determined as input for both flexors, while another $STIM$ signal formed

<table>
<thead>
<tr>
<th>Muscle</th>
<th>$a_0$</th>
<th>$a_1$</th>
<th>$a_2$</th>
<th>$a_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>FCR</td>
<td>0.2951</td>
<td>-0.0174</td>
<td>0.36 x 10^{-3}</td>
<td>0.4 x 10^{-3}</td>
</tr>
<tr>
<td>FDS</td>
<td>0.2953</td>
<td>-0.0163</td>
<td>-0.34 x 10^{-2}</td>
<td>0.4 x 10^{-3}</td>
</tr>
<tr>
<td>ECR</td>
<td>0.2938</td>
<td>0.0133</td>
<td>-0.68 x 10^{-3}</td>
<td>-0.19 x 10^{-3}</td>
</tr>
<tr>
<td>EDC</td>
<td>0.2853</td>
<td>0.0133</td>
<td>-0.68 x 10^{-3}</td>
<td>-0.2 x 10^{-3}</td>
</tr>
</tbody>
</table>

The coefficients were based on the relations between joint angle and muscle length presented by Lemay and Crago (1996).
the input for both extensors in the model. The parameters $a_j$ correspond to the maximum values of $STIM_j$. Since $STIM$ represents the normalized average firing rate of the $\alpha$-motoneurons, the parameters $a$ are dimensionless and bounded on the interval $[0,1]$. Parameter $b_j$ transforms the shape of the control signal; for instance, $\beta > 1$ results in a relative amplification of higher values of $\zeta$.

3.1. Muscle model

The muscle model describes the activation dynamics and the contraction dynamics of skeletal muscle. The muscle is modeled as a contractile element (CE) and an elastic element (SE) in series, representing muscle fibers and tendon, respectively. Because in the examined tasks the muscle fibers did not operate at lengths far beyond the optimum, the contribution of a parallel elastic element was assumed negligible (Van Soest, 1992; Van Soest & Bobbert, 1993). The original model, developed by Van Soest (1992) and Van Soest and Bobbert (1993), was extended in two ways to simulate smooth rhythmic activity: the activation dynamics was slightly altered to include muscle relaxation, and the force–velocity relation for eccentric contractions was adapted to account for muscle behavior at low activity levels.

3.1.1. Activation dynamics

The independent input of the muscle model is $STIM$, representing the normalized average firing frequency of the $\alpha$-motoneurons. The state variable of the activation dynamics is the intramuscular concentration of Ca$^{2+}$ ($\gamma$), and the output of this system is the active state $q$. The activation process accounts for the gross dynamical effect of Ca$^{2+}$ efflux and influx across the sarcolemma. It is modeled as a first-order system according to Hatze (1981, pp. 37–42):

$$\dot{\gamma} = \frac{cSTIM - \gamma}{\tau_\gamma}, \quad \tau_\gamma = \begin{cases} \tau_{act} & cSTIM \geq \gamma \\ \tau_{rel} & cSTIM < \gamma \end{cases}$$

with gain coefficient $c = 0.1373 \times 10^{-3}$ and time constant $\tau_\gamma$. In the present model, different time constants were used for activation and relaxation. The activation time constant $\tau_{act}$ was 89ms (Hatze, 1981). The relaxation time constant $\tau_{rel}$ is related to the half-relaxation time $HRT$, used in physiological studies, by

$$\tau_{rel} = -\frac{HRT}{\ln(0.5)}$$

In the current study, we used a relaxation time constant that was twice as large as the time constant for activation (178ms), which is comparable to empirically estimated $HRT$ (Yoshitake, Shinohara, Ue, & Moritani, 2002).

The active state also depends on the length of the muscle fibers ($l_{CE}$), given the length-dependent Ca$^{2+}$ sensitivity of muscle fibers. This sensitivity is reflected in the active state of the muscle $q$, according to (Hatze, 1981)
\[ q = \frac{q_0 + (\rho \gamma)^3}{1 + (\rho \gamma)^3}, \quad \rho = G l_{CE}^3 \frac{\lambda - 1}{\lambda l_{CE,OPT} - l_{CE}} \]  

(6)

where \( q_0 = 0.005 \), \( G = 52700 \), and \( \lambda = 2.9 \) for all muscles (Hatze, 1981). A practical consequence of this relation is that the active state increases if the muscle fiber lengthens, which can be interpreted as a source of muscle stiffness (Kistemaker, Van Soest, & Bobbert, in press; Van der Burg, 2003).

### 3.1.2. Contraction dynamics

The force exerted by the muscle equals the force generated by SE, which is modeled as a quadratic spring:

\[ F_{SE} = \max \left( k_{SE} (l_{MTC} - l_{CE} - l_{SE,SLACK})^2, 0 \right) \]  

(7)

where \( l_{SE,SLACK} \) is the tendon slack length and \( k_{SE} \) the spring coefficient. The values of \( k_{SE} \) were adapted to yield \( F_{SE} = F_{MAX} \) at a tendon elongation of 0.0333 relative to \( l_{SE,SLACK} \) (Lemay & Crago, 1996). The maximum isometric forces \( F_{MAX} \) are determined by the physiological cross-section area of the muscles reported by Lemay and Crago (1996). Values of \( F_{MAX} \) and \( l_{SE,SLACK} \) are presented in Table 3. According to Eq. (7) the muscle force is a function of \( l_{MTC} \), which is determined by the joint angle as described in the previous section, and \( l_{CE} \), the state variable of the contraction dynamics, which is defined by the force–length–velocity relation of skeletal muscle fibers. The force–length relationship is approximated by a parabola:

\[ F_{ISOM} = \max \left( 1 - \frac{1}{w^2} \left( \frac{l_{CE}}{l_{CE,OPT}} - 1 \right)^2, 10^{-5} \right) \]  

(8)

where \( F_{ISOM} \) is the normalized maximum isometric force that the muscle fibers can generate at a given length. The parameter \( w \) ( = 0.56) determines the width of the force–length relationship. The optimum fiber lengths \( l_{CE,OPT} \) were derived from the morphological characteristics of the wrist muscles as described by Lemay and Crago (1996) and are presented in Table 3.

The ratio of the force generated by the muscle fibers \( F_{CE} (= F_{SE}) \) and the maximum isometric force as a function of fiber length and active state determines the

### Table 3

Muscle-specific model parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>FCR</th>
<th>FDS</th>
<th>ECR</th>
<th>EDC</th>
</tr>
</thead>
<tbody>
<tr>
<td>( F_{MAX} )</td>
<td>Maximum isometric force [N]</td>
<td>150</td>
<td>470</td>
<td>202</td>
<td>211</td>
</tr>
<tr>
<td>( l_{CE,OPT} )</td>
<td>Optimum fiber length [m]</td>
<td>0.164</td>
<td>0.145</td>
<td>0.127</td>
<td>0.118</td>
</tr>
<tr>
<td>( l_{SE,SLACK} )</td>
<td>Tendon slack length [m]</td>
<td>0.122</td>
<td>0.150</td>
<td>0.169</td>
<td>0.164</td>
</tr>
</tbody>
</table>

The maximum isometric force \( F_{MAX} \) is equal to \( \sigma_{MAX} \cdot PCSA \cdot \cos(\psi) \). The physiological cross-section area \( PCSA \), the pennation angle \( \psi \), and the optimum fiber lengths were taken from Lemay and Crago (1996). The maximum tension per unit \( PCSA (\sigma_{MAX}) \) was set to 75N cm \(^{-2}\) to obtain a good fit with the isometric moment–angle relation of the wrist (Delp et al., 1996; cf. Epstein and Herzog (1998) for a range of reported values for \( \sigma_{MAX} \)). Tendon slack lengths were calculated according to the method described by Lemay and Crago (1996).
contraction velocity of the muscle fiber, according to the inverse force–velocity relationship. For concentric contractions \((F_{CE} \leq qF_{MAX}F_{ISOM})\) the contraction velocity is given by

\[
\dot{l}_{CE} = -v_{SCALE}l_{CE,OPT}\left(\frac{F_{ISOM} + v_{SHAPE}}{\frac{F_{CE}}{qF_{MAX}} + v_{SHAPE}} - 1\right)
\]

where the parameter \(v_{SHAPE}\) determines the shape of the relationship (increasing \(v_{SHAPE}\) flattens the curve) and depends on the length of the fibers as \(v_{SHAPE} = A_{REL}\) if \(l_{CE} < l_{CE,OPT}\) and \(v_{SHAPE} = A_{REL}F_{ISOM}\) if \(l_{CE} \geq l_{CE,OPT}\). The parameter \(v_{SCALE}\) depends on the active state \(q\): \(v_{SCALE} = \min(1, 3.333q) B_{REL}\). The same parameters \(A_{REL} (0.41)\) and \(B_{REL} (5.2)\) were used for all muscles (Van Soest, 1992; Van Soest & Bobbert, 1993). The ratio \(B_{REL}/A_{REL}\) determines the maximum shortening velocity in optimum fiber lengths per second.

For eccentric contractions \((F_{CE} > qF_{MAX}F_{ISOM})\), the hyperbolic force–velocity characteristic was derived from Eq. (9) using the constraints specified by Van Soest and Bobbert (1993):

\[
\dot{l}_{CE} = \frac{p_1}{\frac{F_{CE}}{qF_{MAX}} + p_2} + p_3
\]

where

\[
p_1 = -\frac{v_{SCALE}}{\sigma} \left(1 - \frac{F_{ECC}}{F_{ISOM}}\right)^2
\]

\[
p_2 = -F_{ECC}F_{ISOM}
\]

\[
p_3 = -\frac{p_1}{\frac{F_{ISOM}}{p_1} + p_2}
\]

The parameter \(F_{ECC}\) is the maximum eccentric force that the muscle can generate as a fraction of \(F_{ISOM}\), and \(\sigma\) defines the ratio between the slopes of the eccentric and concentric curves in the point where the contraction velocity is zero. For all muscles, these parameters were equal to 1.5 and 2, respectively. From Eqs. (9) and (10) can be inferred that the lower bounds on \(q\) and \(F_{ISOM}\) are necessary to avoid division by zero.

In the simulations of rhythmic movements, the active state \(q\) approaches its minimum in the half-cycle that the muscle is stretched due to the activity of its antagonists. Under those conditions numerical problems might arise if, as a consequence of stretching, the force of the muscle \(= F_{CE}\) exceeds the asymptotic maximum of the active eccentric force, i.e., \(F_{CE} > qF_{MAX}F_{ISOM}F_{ECC}\). However, the very low active state implies that the behavior resembles that of a passive muscle: at higher forces the lengthening velocity of the muscle fiber increases linearly with the applied force (linear damping), whereas at lower forces the behavior mimics that of the (hyperbolic) active force–velocity relation (Krause, Choi, & McMahon, 1995). For numerical reasons, the change between the linear and the hyperbolic regime was made dependent on the muscle force rather than \(q\).
\[
\frac{F_{CE}}{qF_{MAX}} > -\sqrt{\frac{-p_1}{\delta_{PASS}v_{SCALE}}} - p_2
\]  
(11)
defines the linear regime, with
\[
\dot{\lambda}_{CE} = \delta_{PASS}v_{SCALE}\left(\frac{F_{CE}}{qF_{MAX}} + p_2\right) + p_3 + 2\sqrt{p_1\delta_{PASS}v_{SCALE}}
\]  
(12)
where \(\delta_{PASS} = 1000\) is the slope of the linear part of the passive force–velocity curve. The boundary condition assures that the hyperbolic and the linear part of the eccentric force–velocity relation are continuous.

3.2. Optimization in two steps

The model parameters in Eqs. (1) and (3) were fitted to the experimental reference data in two steps, whereas all other parameters were obtained from the literature. First the model was used to simulate the four conditions of the isometric task, and subsequently the model was used to simulate the two conditions of the dynamic task. In both steps, a different set of parameters was fitted, and the parameters obtained in the optimization of the isometric task remained fixed during the optimization of the dynamic task. The parameters were optimized so as to minimize the criterion function
\[
d = \sum_{l=1}^{M} \frac{1}{N_l} \sum_{k=1}^{N_l} \left\{(y_{l,k} - x_{l,k})^2 / (y_{l,k}^2 + x_{l,k}^2)\right\}
\]  
(13)
where \(y_{l,k}\) and \(x_{l,k}\) are the \(k\)th sample of the \(l\)th time series, for reference and simulation, respectively. \(M\) is the number of reference time series, i.e., four for the isometric task and two for the dynamic task, and \(N_l\) is the number of samples used for comparison of simulation and experiment.

Under the assumption that the joint angle was fixed at \(\theta_{eq}\), the parameters of the damped mass–spring model in Eq. (1) were irrelevant when the isometric task was simulated. Thus, only the four parameters \((x_1, x_2, \beta_1, \beta_2)\) in Eq. (3) were optimized in the simulations of the isometric task. The second step entailed optimization of stiffness \(K\), damping \(B\), and joint equilibrium angle \(\theta_{eq}\) in Eq. (1) using the reference signals of the dynamic task with parameter values for Eq. (3) as determined for the isometric task. A variable step-size, variable order Runge–Kutta method available in MATLAB® was applied in the simulations. This method involves an interpolation technique to obtain equidistant time-steps, which facilitated comparison with the experimental data. Since the model input \(STIM\) was based on the recorded EMG signals, it was also a time series with a sample frequency of 1000Hz. Linear interpolation between samples was used to obtain values of \(STIM\) for intermediate time-steps during the simulations.

For the isometric task, the simulated torques corresponded closely to the experimental reference curves, with \(R^2\) values between 0.96 and 0.98 (see Fig. 3a–d). The optimal parameter values were \(x_1 = 0.18, x_2 = 0.22, \beta_1 = 0.79, \) and \(\beta_2 = 0.77\). The values of \(x\) are dimensionless scaling factors that correspond to the maximum values
STIM can reach. Since STIM was normalized to the interval $[0, 1]$, the input to the muscles reached about 1/5 of its maximum value. At first sight, this might suggest that even in the ‘high force’ condition the isometric force remained well below its maximum. However, note that the isometric force of the muscle model is determined by the active state $q$, which is related nonlinearly to STIM due to saturation effects (see Eqs. (4) and (6)). Consequently, peak active state per cycle reached values between 0.6 and 0.8 in the simulated high force condition. This implies that, according to the model, participants were producing about 60–80% of their maximum isometric force, which is a relatively high force level to be reached repeatedly in a 30-s trial.

Also in the simulations of the dynamic task, the musculoskeletal model reproduced the temporal characteristics quite well, with $R^2$ values of 0.92 and 0.98 for movement frequencies of 1.0 Hz and 1.4 Hz, respectively (see Fig. 3e-f). The optimized equilibrium angle $\theta_{eq}$ was 20.9°, indicating that the wrist was slightly flexed when in mechanical equilibrium. Optimized values for joint stiffness and damping were $K = 0.67 \text{ Nm rad}^{-1}$ and $B = 0.11 \text{ Nm s rad}^{-1}$, which are of the same order of
magnitude as the range of values reported in the literature (Milner, 2002; Milner & Cloutier, 1998; Winters, Stark, & Seif-Naraghi, 1988).

3.3. Evaluation of the musculoskeletal model

The musculoskeletal model adequately reproduced the relations between EMG and task-related output in the two rhythmic tasks. In the isometric task, the good fit resulted mainly from a combination of morphological parameters obtained from the literature, the activation dynamics, and an optimized scaling of EMG-based input. According to the model, the contribution of contraction dynamics was negligible in the isometric task, since only very small changes in fiber length (maximally 4.6 mm, i.e. 3.9%) occurred during the stretch and release of the tendon that resulted in the alternating joint torque. In addition to the factors involved in the isometric task, also the contraction dynamics, the joint dynamics and the interactions between muscles and skeleton were involved in the model simulations of the dynamic task. In order to account for the experimentally observed phase shifts the passive joint stiffness and damping were optimized.

Using Eq. (2) the phase shift due to joint dynamics were calculated as a function of frequency, and based on this relation the contributions of the (passive) joint dynamics and the (active) muscle dynamics to the empirically observed phase shifts were dissociated (see Fig. 4, top panels). This distinction indicated that a large part of the total phase shift due to effector dynamics as observed for the dynamic task can be attributed to joint stiffness and damping. According to the model, the joint dynamics of the wrist generated the braking torques that were required for slowing down as the wrist moves toward peak excursion. As a consequence, the braking torques were generated before the antagonist muscles were activated. In sum, the model simulations demonstrated that phase shifts due to effector dynamics could be attributed to the dynamical characteristics of activation, contraction, and joint dynamics, and that, to some extent, the contributions of these subsystems could be dissociated.

However, an important practical disadvantage of musculoskeletal models is the large number of parameters required. Although all muscle parameters could be based on the results of various morphological and physiological studies reported in the literature (see above), the correspondence between the model and the average behavior of the six participants in the study by Peper and Carson (1999) may be hampered by inter-individual variations in the parameter values. Errors may have resulted, for example, from inaccurate estimates of optimum fiber lengths, time constants for (de)activation, tendon slack lengths, maximum isometric forces, maximum shortening velocities, etc. Hence, to achieve a very high degree of accuracy in the reproduction of experimentally obtained trajectories, additional parameter calibration may be necessary for the musculoskeletal model. In this respect, an important assumption regarding musculoskeletal models is that the optimization of the model parameters is task-independent, that is, the parameters reflect structural properties of the system that are not susceptible to rapid changes related to the task at hand. This implies that any additional calibrations, like the optimization of input parameters and joint dynamics in the present study, may be performed with reference to
data that are not directly related to the primary research questions. Thus, this practical disadvantage of the musculoskeletal model can be overcome, if the assumption of task independence is correct.

Unfortunately, such an additional calibration and validation of parameters could not be performed in the present study, because adequate reference data was lacking, like, for example, the relation between EMG and constant isometric torque at various joint angles, or between EMG and joint angle trajectories for discrete movements at various movement velocities. To examine the effect of model parameters on the optimized values of joint stiffness and damping the influence of changing the parameters $A_{REL}$ and $B_{REL}$ of the force–velocity relationship in Eqs. (9)–(12) was investigated. These parameters are directly related to the intrinsic muscle damping and different values of these parameters have been suggested in the literature (Van Zandwijk, Bobbert, Harlaar, & Hof, 1998). The model optimization was repeated with the parameter values $B_{REL} = 2.01$ and $A_{REL} = 0.21$ (the averages of the values reported by Van Zandwijk et al., 1998), resulting in a 27% decrease in

![Joint Dynamics: Original Model](image1)

![Joint Dynamics: HMD model](image2)

Fig. 4. Contribution of muscle and joint dynamics to the phase shifts in the dynamic task. Left panels: the optimized joint dynamics defines the phase shift between the resultant muscle torque and joint angle ($\Delta \Phi_{joint}$) as a function of frequency. The negative values on the vertical axis indicate that the driving torque leads the joint angle. The dotted lines specify the phase shifts at the two frequencies used in the experiment of Peper and Carson (1999). Right panels: by subtracting $\Delta \Phi_{joint}$ from $\Delta \Phi_{total}$, the total phase shift between EMG and joint angle (see Table 1; values averaged over FCR and ECR), the phase shift due to muscle dynamics ($\Delta \Phi_{muscle}$) can be estimated. Top panels: phase shifts as obtained for the original model. Bottom panels: phase shifts as obtained for the model with high muscle damping (HMD).
the maximum shortening velocity. In other words, for these parameter settings the force of the muscles declines more rapidly with increasing contraction velocity, which implies a larger intrinsic muscle damping. The results of the optimization of this high muscle damping (HMD) model were compared with those obtained for the original model.

The increase in muscle damping hardly changed the goodness-of-fit between model and experiment ($R^2$ values between 0.97 and 0.98 for the isometric task and $R^2$ values of 0.94 and 0.98 for the dynamic task, at movement frequencies of 1.0 Hz and 1.4 Hz, respectively). The input scaling parameters were almost identical to those obtained for the original model ($a_1 = 0.18$, $a_2 = 0.23$, $b_1 = 0.81$ and $b_2 = 0.79$), which indicated that the role of muscle damping was negligible in the isometric task. This was not surprising, because only slight changes in fiber lengths occur during isometric contractions. In contrast, the influence of increased muscle damping on the optimized parameters of the joint stiffness and damping was quite substantial (optimized values: $K = 0.98$ Nm rad$^{-1}$ and $B = 0.03$ Nms rad$^{-1}$). As a consequence, the HMD model differed considerably from the original model with respect to the estimated contributions of (active) muscle and (passive) joint dynamics to the experimentally observed phase shifts in the dynamic task (compare bottom and top panels in Fig. 4).

Although changes in the other parameters of the model had only minor effects on the outcome of the optimization (and are, therefore, not presented), the consequences of increased muscle damping indicated that the simulation results should be treated with some caution. Physiologically, the characteristics of a muscle are largely determined by the relative amount of slow twitch (type I) and fast twitch (type II) fibers. The muscles of the HMD model have a lower maximum shortening velocity, which implies a larger percentage of slow twitch (type I) fibers than in the original model. Unfortunately, the physiological basis for the selection of values for parameters $A_{REL}$ and $B_{REL}$ is rather slim, since no explicit relation between fiber type distribution and model parameter values has been established. However, the parameter values for the HMD model were derived from experiments on triceps surae (Van Zandwijk et al., 1998), which has a larger percentage of type I fibers than forearm muscles (Johnson, Polgar, Weightman, & Appleton, 1973). This indicates that for our model of the wrist dynamics the original set of model parameters (Van Soest, 1992; Van Soest & Bobbert, 1993) is to be preferred over the second (HMD) set.

4. Changes in the effector dynamics induced by inertial loading

The analysis of the effector dynamics of the wrist presented in the preceding sections focused on the differences between two tasks: an isometric task (rhythmic torque production) and a dynamic task (rhythmic movement). The results showed that the temporal relation between the EMG and the relevant behavioral variable (torque and joint angle, respectively) was markedly influenced by the task context. Another, more common manipulation of the effector dynamics in the study of interlimb
coordination concerns the application of (unequal) inertial loads to the coordinated limbs (Baldissera, Borroni, & Cavallari, 2000; Baldissera & Cavallari, 2001; Jeka & Kelso, 1995; Kelso & Jeka, 1992; Peper, Nooij, & Van Soest, in press; Schmidt, Beek, Trefner, & Turvey, 1991; Schmidt et al., 1993; Sternad, Collins, & Turvey, 1995; Sternad, Turvey, & Schmidt, 1992; Trefner & Turvey, 1995). Several studies have indicated that loading changes the relation between EMG and movement, which implies that the effector dynamics was affected (Baldissera et al., 2000; Baldissera & Cavallari, 2001; Baldissera et al., 1991; Mackey et al., 2002). Explicit models of the effector dynamics provide a means for examining the functional significance of these input (EMG)–output (movement) relations.

Using a linear damped mass–spring model of the effector system, Baldissera and Cavallari (2001) quantified the changes in the effector properties due to loading based on the empirically obtained phase shifts. This model was similar to the model of the joint dynamics used in the present study (see Eq. (1)), with the crucial difference that the driving torque $T$ was not a function of contraction and activation dynamics but was assumed to be directly proportional to the EMG. Based on this assumption Baldissera and Cavallari estimated the values of stiffness $K$ and damping $B$ directly from the frequency-dependent phase shift between EMG and joint movements using the relation given by Eq. (2). Their analysis revealed that attaching an additional inertial load to a limb segment induced changes in the model parameters $K$ and $B$. In particular, an increase in $K$ was found, which was interpreted as “a reactive increase in the apparent stiffness of the limb” (Baldissera & Cavallari, 2001, p. 22). Although this use of the linear damped mass–spring model may be regarded as a convenient method to parameterize data sets and thus to quantify the differences between conditions, the mechanical interpretation of the $K$ parameter in terms of limb stiffness is questionable.

The key problem is that EMG does not correspond directly to a driving torque, since the relation between EMG and the driving torque (or net muscle torque) is affected non-trivially by the muscle dynamics and the interactions between muscles and skeleton. For example, a part of the phase shift between EMG and net muscle torque should be attributed to the activation dynamics of skeletal muscle, and is thereby unrelated to the stiffness and damping of the musculoskeletal system. Thus, the stiffness and damping of the system cannot be inferred directly from the relation between EMG and movement. Moreover, the apparent changes in the dynamics of the limb induced by inertial load application, raise the question whether these changes are to be attributed to the intrinsic dynamics of the musculoskeletal system (i.e., the effector dynamics) or to an adaptation of the neural control processes underlying the rhythmic movement. This issue can only be resolved if the analyses are based on an explicit model of the musculoskeletal dynamics. To illustrate the surplus value of such a model, the effects of adding an inertial load to a limb segment are analyzed using the musculoskeletal model that was developed in the present study, and compared to the results reported by Baldissera and Cavallari (2001).

In the experiment of Baldissera and Cavallari (2001) participants had to produce cyclic flexion–extension hand movements in the vertical plane. By taking the delay
between the onset of ECR activity and the beginning of the hand extension phase, the input–output phase shift of the wrist was determined at frequencies ranging from 0.8 to 3.0 Hz. As experimental manipulation an inertial load of 0.015 kg m$^2$ was applied by adding lead discs. Because the wrist movements examined by Baldissera and Cavallari and those that formed the basis for our analyses (Peper & Carson, 1999) were performed in different planes (vertical vs. horizontal, respectively), and because Baldissera and Cavallari used a different method to calculate phase shifts, a direct comparison between their experimental results and the values obtained in the present study is not possible. However, a reasonable comparison can be made by considering the differences between the phase shifts in the loaded and the unloaded condition. To this end, the stiffness, damping and inertia parameters of the linear damped mass–spring model that was fitted to the data by Baldissera and Cavallari were used to reconstruct the frequency-dependent phase shifts of both conditions. Since Baldissera and Cavallari obtained separate parameter sets for men and women, the estimated phase shifts for both genders were averaged to match the mixed population of participants in the experiment of Peper and Carson. Subsequently, the curves fitted to the data of the unloaded condition were subtracted from the curves fitted to the data of the loaded condition to obtain the additional phase shift induced by the inertial load as a function of frequency (see Fig. 5).

Fig. 5. The additional phase shifts induced by an added inertial load. Baldissera and Cavallari (2001) measured the phase shift between EMG and joint angle at different movement frequencies, with and without an additional inertial load of 0.015 [kg m$^2$]. Parameters for a linear damped mass–spring model were fitted to the data for the loaded and unloaded condition. For both conditions the phase shift was calculated as a function of frequency with Eq. (2), the difference between these conditions is shown as the additional phase shift induced by the inertial load (dashed line). In addition, the musculoskeletal model was used to simulate the oscillatory movements in both conditions of loading, and for each simulation the phase shift between the control signal STIM and the resultant joint angle was estimated. The difference between the loaded and unloaded condition is shown as a function of movement frequency (solid line).
In order to analyze the effects of loading with the musculoskeletal model, neural control signals \textit{STIM} had to be obtained for flexors and extensors and for movement frequencies ranging from 0.8 Hz to 3.0 Hz, that is, the frequencies used by Baldissera and Cavallari. These input signals were based on the EMG data of the study of Peper and Carson, which was available only for the movement frequencies 1.0 Hz and 1.4 Hz, by means of a combination of interpolation and extrapolation (see A). With these estimated control signals the musculoskeletal model simulated cyclic movements at the desired frequencies, and from these simulations the phase shifts between joint angle trajectories and the input signals was determined for each frequency and each condition with the method described in Section 2.1. Only the inertia of the musculoskeletal model was adapted to match the experimental manipulation. Note that the other parameters and \textit{STIM} signals were identical in the simulations of the two conditions with the musculoskeletal model, whereas the fits of the linear damped mass–spring model involved different parameters for the stiffness and damping in the two conditions. To obtain the additional phase shift induced by the inertial load as a function of frequency for the musculoskeletal model, the curves of the unloaded condition were subtracted from the curves of the loaded condition (see Fig. 5). Comparison of the two curves shown in Fig. 5 indicated that the musculoskeletal reproduced the experimentally observed effects of an added inertial load on the phase shifts quite well.

The simulation results of the musculoskeletal model indicated that the changes in the apparent dynamics of the limb reported by Baldissera and Cavallari (2001) could be attributed largely to the intrinsic dynamics of muscles. For example, if the neural control signal remains unaltered, an additional inertial load results in less acceleration and hence lower movement velocities. As a consequence, the shortening velocity of the agonists is smaller than in the unloaded situation, which results in increased muscle torque due to the muscle fibers’ force–velocity relationship. Thus, at any given point in the movement cycle, the net torque generated by the muscles will be increased relative to the unloaded condition. Hence, to explain the observed effects of loading, it may not be necessary to assume an additional control process for adaptively regulating the stiffness of the effector system, be it by increasing the intrinsic stiffness (by co-contraction) or the reflexive stiffness (by adjusting feedback gains). Admittedly, the various assumptions underlying the present simulation are not readily verified. Nevertheless, the load simulation underscores the added value of a musculoskeletal model for studying the effects of loading and their proper interpretation in terms of underlying neurophysiological processes vis-à-vis the intrinsic dynamics of the musculoskeletal system.

5. Discussion

The aim of the present study was to analyze the effector dynamics of rhythmic wrist activity, and to assess its consequences for (models of) interlimb coordination. To this end, the results of model simulations were compared to empirical data ob-
tained by Peper and Carson (1999). A physiologically and anatomically motivated musculoskeletal model of the wrist was developed in which the active muscle dynamics was dissociated from the passive joint dynamics. The empirical reference data revealed quite substantial differences in the phase shifts due to effector dynamics between the isometric task and the dynamic task. The proposed model of the effector dynamics could adequately reproduce these phase shifts between the (EMG-based) neural control signal and the task-related output (torque and joint angle, respectively).

The distinction between the contributions of (passive) joint dynamics and (active) muscle dynamics to the phase shift between EMG and movement indicated that joint stiffness and damping (i.e., joint dynamics) have a substantial effect on the dynamics of the wrist. Interestingly, such a significant contribution of passive mechanisms to the overall dynamics was also suggested in relation to repetitive finger movements (Dennerlein, Mote, & Rempel, 1998). It is important to note that such insights into the effector dynamics could not have not been obtained with a more conventional linear damped mass–spring model (with lumped contributions of active and passive structures to the effector dynamics). In addition, comparison between the simulations of the musculoskeletal model at various frequencies with and without an added inertial load, indicated that the empirically observed effects of the inertial load onto the relation between EMG and movement may be attributed to the intrinsic dynamics of the musculoskeletal system. Thus, the apparent changes in stiffness and damping that may be inferred from an analysis using the linear damped mass–spring model (see Baldisserra & Cavallari, 2001) do not necessarily imply the existence of a neural control process associated with these parameter changes. Hence, we may conclude that the musculoskeletal model, which is physiologically, anatomically, and mechanically motivated, provides an adequate and relevant account of the effector dynamics of the wrist. This conclusion is consistent with the results of previous studies that have successfully applied this type of model to a wide range of tasks (e.g., vertical jumps, Van Soest, 1992; counter-movement jumps, Bobbert, Gerritsen, Litjens, & Van Soest, 1996; standing long jumps, Ridderikhoff, Batelaan, & Bobbert, 1999; ballistic arm movements, Welter & Bobbert, 2002; lifting movements, Van der Burg, 2003; bipedal stance, Van Soest, Haenen, & Rozendaal, 2003; and cycling movements, Van Soest & Casius, 2000).

5.1. Phenomenological models

A linear damped mass–spring model represents a special case of a linear second-order system in the sense that the model parameters are interpreted in mechanical terms (i.e., stiffness, damping and inertia). As argued in Section 4, this interpretation is inappropriate if a linear second-order system is used to model the input–output relation between EMG and joint angle. Nevertheless, a second-order linear system can reproduce empirically observed relations between EMG and the task-related output (Baldisserra & Cavallari, 2001; Lehman & Calhoun, 1990; Stein, Cody, & Capaday, 1988). This indicates that, in line with the approach of Beek et al. (2002; see also Jirsa & Haken, 1997; Sternad et al., 1998), such a model of effector
Dynamics may be used in the two-tiered model of rhythmic coordination mentioned in Section 1. In this context, the second-order linear systems model serves to incorporate the input–output relations of the effector dynamics into a formal model of the coordination dynamics. For this purpose, a mechanical interpretation of the model parameters is not necessary, since the second-order system is used as a descriptive (i.e., phenomenological) model, and the problems related to this interpretation can (and should) be avoided.

As a tool in the mechanical analysis of a limb segment, the model can be regarded as a linearization of the dynamics of a (nearly) passive limb segment oscillating about a working point in state space (Van Soest, Peper, & Selles, in press). In that case, a linear damped mass–spring system may provide a good approximation of the mechanics of the limb segment, provided that the analysis only concerns states in the vicinity of the working point. Formally, this implies that the model is only applicable in the mechanical analysis of movements with small amplitudes, and that with increasing amplitudes the use of a second-order linear system in the mechanical analysis of rhythmic movements becomes less appropriate.

Evidently, a model of a linear second-order system, given its relative computational and conceptual simplicity, is much easier to implement than the musculoskeletal model. In addition, the dynamics of a two-tiered model of interlimb coordination with a linear second-order model of the effector dynamics may be assessed by means of analytical methods instead of simulations (Beek et al., 2002). This is an important practical advantage, as it is not easy to see how similar analytical conclusions can be gleaned from a musculoskeletal model, given its complexity and degree of nonlinearity.

5.2. Implications for interlimb coordination

As we have seen, considerable differences in the temporal characteristics of the effector dynamics may arise as a function of task context. Such asymmetries in the effector dynamics, reflected in the different phase shifts, may pose an additional challenge for the nervous system during interlimb coordination. Fig. 6 provides a schematic illustration of the asymmetries that arose when the isometric task and the dynamic task were performed simultaneously in the experiment of Peper and Carson (1999). At a movement frequency of 1.0 Hz the EMG leads the torque by about $47^\circ$ in the isometric task, whereas the EMG leads the movement by about $92^\circ$ in the dynamic task. Hence, in order to produce an in-phase coordination pattern at the behavioral level (a relative phase of $0^\circ$ between torque and movement), the average activity of contralateral $\alpha$-motoneuron pools should be about $45^\circ$ out-of-phase.

In other words, the asymmetry between the two tasks results in an asymmetry between the timing of the control and the observable behavior (see Fig. 6). It is conceivable that this type of asymmetry affects the coordination dynamics. In this respect, it is noteworthy that Peper and Carson (1999) observed that during coordinated isometric and dynamic activity the characteristic difference in stability between the in-phase and antiphase modes of coordination was absent. In a similar vein, the dif-
Differential effects obtained for in-phase and antiphase coordination under asymmetric loading of the limbs might be related to the way in which the nervous system deals with the induced phase shifts between EMG and behavioral output (Baldissera & Cavallari, 2001; Baldissera et al., 1991).

In the literature, two general hypotheses can be found regarding the accommodation of the neuromuscular control to an asymmetry in the phase shifts due to effector dynamics. An adaptation in the timing of the neural control signals is necessary, as feedforward control fails to compensate for the difference in the phase shifts of the effector dynamics and thus would result in large errors in the relative phase at the behavioral level (see Fig. 7a). Indeed, the results of the study of Peper and Carson (1999) showed that the error in the average relative phasing at the behavioral level (about 18° and 27° for 1.0Hz and 1.4Hz, respectively, see Table 3 in Peper & Carson) was considerably smaller than the difference between the phase shifts expected on the basis of effector dynamics alone (about 45° and 52° for 1.0Hz and 1.4Hz, respectively, see Table 1 in the present paper). This reduction in the behavioral phase shifts relative to those attributable to the different effector dynamics suggests active involvement of sensory systems in establishing the required coordination pattern. On the assumption that the stability of in-phase and antiphase coordination results from attraction of the phasing between the associated neural control signals to phase relations of 0° or 180°, respectively, the observed behavior can be regarded as the result of two competing subsystems: a feedforward system that favors in-phase and antiphase coordination, and a feedback system that allows for a compensation for the asymmetry in effector dynamics. This hypothesis, which is akin to the hypothesis of Baldissera et al. (1991), is illustrated in Fig. 7c. An alternative hypothesis asserts that adequate behavioral phasing of the limbs may be attained on the basis of perceptual information only (Mechsner et al., 2001). According to this latter hypothesis, deviations from the required relative phase observed at the behavioral level

![Fig. 6. Schematic illustration of the phase shifts due to effector dynamics in the experiment of Peper and Carson (1999). Circles and arrows represent signal sources and time-delays (i.e. phase shifts), respectively. T is the isometric torque, θ is the joint angle, and ξ is the neural control signal (~EMG amplitude). The inclination angles of the thin lines between the two signals at the behavioral or the control level indicate the relative phase between the two signals at the level in question. If both hands perform the isometric task (left diagram) or the dynamic task (middle diagram) simultaneously, the relative phases at the control and the behavioral level are identical. In contrast, if the isometric task and the dynamic task are performed simultaneously the relative phases at the two levels differ.](image-url)
should be attributed only to the finite accuracy of the perceptual system (Zaal, Bingham, & Schmidt, 2000), as is illustrated in Fig. 7b.

The present study shows that the relation between effector asymmetry and the task-related error can be studied systematically by inducing variations in the temporal relationship between muscle activation and the resulting behavioral output for the limbs involved (e.g., by application of appropriate loads). The hypothesis would receive support if, at a particular movement frequency, the phase shift observed in terms of behavior is independent of the degree of asymmetry between the effectors. In contrast, a consistent relationship between the degree of effector asymmetry and the observed error would constitute evidence in favor of the competition hypothesis. The empirical results of several studies indicated that the error in the relative phase was dependent on the difference in eigenfrequency between two hand-held pendulums (Schmidt & Turvey, 1995). Since the difference in eigenfrequency was due to unequal loading of the limbs, it is likely that they were associated with an asymmetry in the phase shifts between the control signals and the rhythmic movement (Baldissera & Cavallari, 2001; Baldissera et al., 1991). Thus, these results suggested that the error in the relative phase was dependent on the degree of asymmetry between the effectors, which contradicts the ‘perception only’ hypothesis. Although in the case of the coordination of hand-held pendulums the degree of asymmetry can be adequately determined in terms of an eigenfrequency difference (Kugler & Turvey, 1987), it should be noted that such a quantification of the degree
of asymmetry cannot always be established. Therefore, characterization of the asymmetry in terms of the phase shifts due to effector dynamics (for instance, those based on an analysis of the cross-spectral density of the EMG and associated task-related output, as carried out in the present study) may be preferred.

To deal with the effects of effector dynamics on the coordination dynamics, it is essential that these dynamics are explicitly incorporated in the associated models of interlimb coordination. Two-tiered models, like the one proposed by Beek et al. (2002), provide an adequate framework for such model development. Depending on the research questions, the effector dynamics may be incorporated in a more phenomenological manner (viz. as a linear second-order system) or, resorting to a more detailed level of analysis, a physiologically motivated model of the musculoskeletal dynamics may be used. The latter type of model allows for additional insights regarding the neuromuscular control processes, and may, thus, help to explain the effects of manipulations and the associated neuromuscular compensations (Baldissera et al., 2000; Baldissera & Cavallari, 2001; Mackey et al., 2002) in relation to the underlying physiological processes. The work in the present study may provide a basis upon which to select the most appropriate type of model, given an appreciation of the particular research questions, which are at hand.

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Appendix A. Estimation of control signals

Input signals corresponding to six cycles of movement at 1.0Hz and 1.4Hz were obtained from the data of the experiment by Peper and Carson (1999). Using cubic spline interpolation these 1000Hz signals were cast into series of 6001 samples, labeled $X_{1.0}$ and $X_{1.4}$ respectively. For each movement frequency $f$ an input signal of 6001 samples could be obtained using linear scaling:

$$X_f = X_{1.0} + (f - 1) \frac{(X_{1.4} - X_{1.0})}{0.4} \quad (A.1)$$

At the highest frequencies (2.8Hz and 3.0Hz) this scaling resulted in values of $X_f$ below 0, which was not allowed since the input signals to the model were restricted to the interval (0, 1). Hence, limits were imposed on $X_f$:

$$X_f = \max(0.005, \min(X_f, 0.995)) \quad (A.2)$$

Subsequently, cubic spline interpolation was used again to create an input signal $\xi$ with equidistant steps of 1ms by adjusting the number of samples in the series $X_f$. Finally, the signal $\xi$ was low-pass filtered with a cut-off frequency of 15Hz to remove
the discontinuities due to the clipping in Eq. (A.2), in order to facilitate the numerical simulations.

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